



Modern Human Anatomy Program

UNIVERSITY OF COLORADO **ANSCHUTZ MEDICAL CAMPUS**

COURSE PACKET

ANAT 6310 Neuroanatomy Fall 2026

Prepared by
Maureen Estévez Stabio, PhD
Modern Human Anatomy Program
University of Colorado Anschutz Medical Campus
maureen.stabio@cuanschutz.edu

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TABLE OF CONTENTS

Syllabus.....	3
Schedule	10
Unit I: Cellular & Physiological Foundations	15
Lectures 1-8.....	16
Lab 1	40
Unit II: Anatomy & Imaging of the Brain.....	42
Lectures 9-15.....	43
Labs 2-3	55
Unit III: Spinal Cord, Brainstem & Cranial Nerves	63
Lectures 16-24.....	64
Labs 4-6	83
Unit IV: Special Senses & Brainstem Integration.....	101
Lectures 25-32.....	102
Labs 7-9	120
Unit V: Motor, Limbic & Integrative Systems.....	137
Lectures 33-40.....	138
Labs 10-11	155
Neuro Glossary.....	169

ANAT 6310: NEUROANATOMY SYLLABUS 2026

Course Directors

Maureen Estévez Stabio, PhD, Associate Professor
Dept. of Cell & Developmental Biology (CDB)
University of Colorado School of Medicine
Office Tel: 303-724-7461 *Office location:* Bldg 500, Rm. N5209B
Office hours: By appointment maureen.stabio@cuanschutz.edu

John Caldwell, PhD, Professor
Dept. of Cell & Developmental Biology (CDB)
University of Colorado School of Medicine
Office Tel: 303-724-3190 *Office location:* RC-1 South, Room 11116
Office hours: By appointment john.caldwell@cuanschutz.edu

Additional Faculty & Guest Speakers

Erika Alor, MS, Cell & Developmental Biology	erika.alor@cuanschutz.edu
Kerry Barba, MS, Cell & Developmental Biology	kerry.barba@cuanschutz.edu
Tom Finger, PhD, Cell & Developmental Biology	tom.finger@cuanschutz.edu
Cory Gritton, PhD, Cell & Developmental Biology	cory.gritton@cuanschutz.edu
Ethan Hughes, PhD, Cell & Developmental Biology	ethan.hughes@cuanschutz.edu
Christy Niemeyer, PhD, <i>Neurology</i>	christy.niemeyer@cuanschutz.edu
Brad Pawlikowski, PhD <i>Pediatrics</i>	bradley.pawlikowski@cuanschutz.edu
John Thompson, PhD, <i>Neurosurgery</i>	john.thompson@cuanschutz.edu
Courtney Wilson, PhD, <i>Otolaryngology</i>	courtney.wilson@cuanschutz.edu

Teaching Assistants

Two upper division MHA students will assist you in this course as TAs. Please contact TAs directly for assistance outside of scheduled class or lab time.

Muazzam Ramziddin (3 credits)	Muazzam.Ramziddin@cuanschutz.edu
Sloan Fridrich (1 credit)	Sloan.Fridrich@cuanschutz.edu

Credits

ANAT 6310 (Neuroanatomy) is a 4-credit course, with lecture and lab components, that fulfills a core requirement of the Master of Science in Modern Human Anatomy program.

Prerequisites & Enrollment Restrictions

Students must be enrolled in the Master of Science in Modern Human Anatomy program or have special permission from the course director.

Learning Management System

Course materials and announcements will be posted on Canvas. It is your responsibility to monitor Canvas daily.

Required Free Software

Visible Brain Atlas www.toltech.net/downloads/visible-brain-atlas. For software support email support@toltech.net Watch the instructional video that describes how to use this software <https://vimeo.com/1111771974/393a3760bf>

Required Online Free Textbook

Introduction to Neuroscience by Valerie Hedges, <https://openbooks.lib.msu.edu/introneuroscience1/>
This is a free open access online textbook developed by Michigan State University. The reading assignments from this book are designed for those who have no prior experience in neuroscience. These readings will help provide a foundation for the content we will learn in this course, but are optional for those who already have a background in neuroscience.

Optional Illustrative Atlas

THIEME Atlas of Head, Neck and Neuroanatomy. 2nd Edition © 2016 by Thieme Medical Publishers, Inc. ISBN: 978-1-62623-120-7. A copy of this textbook is available for use in the MHA suite.

Free (or nearly free) Online Resources

- *University of British Columbia Functional Neuroanatomy* <http://www.neuroanatomy.ca/>
- *The Neurosurgical Atlas* <https://www.neurosurgicalatlas.com/neuroanatomy>
- *Brain Facts* © 2018 Society for Neuroscience, <https://www.brainfacts.org/the-brain-facts-book>
- *Neuroscience Online* <http://nba.uth.tmc.edu/neuroscience/index.htm>
- *University of Washington Digital Brain Atlas* <http://www9.biostr.washington.edu/cgi-bin/DA/PageMaster?atlas:Neuroanatomy+ffpathIndex:Splash^Page+2>
- *3D Brain for iTunes App*, <http://itunes.apple.com/us/app/3d-brain/id331399332?mt=8>
- *Brain Tutor HD App*, <https://itunes.apple.com/us/app/brain-tutor-hd/id392586830?ls=1&mt=8>

Course Format

ANAT 6310 is a synchronous in-person on-campus course from September 1 to December 17, 2026:

- **Lectures:** Lectures will typically occur on Tuesdays and Thursdays from 9am – 11am.
- **Labs:** Labs will typically be held Wednesdays from 1-3pm (with some exceptions; please see the schedule). Labs vary in format including:
 - **Wet Labs** – located in the gross anatomy laboratories on the 5th floor of Ed 1 or on the first floor of RC1 North (MPLs). XR exploration will be held in the plastination library Ed1-5101.
 - **Dry Labs** – These will typically be held in a classroom and include case studies or clinical demonstrations.
 - **Computer Lab** – 3D MRI lab will be held in Ed2N – 2201 AB/CD
- **Neuroscience in the News Presentations:** see details below
- **Practice Quizzes:** These are ungraded quizzes available on Canvas to help you prepare for exams.
- **On-Campus Exams:** Examinations will be held on campus in Ed2N-2201 Computer Labs (D,E) and administered via Canvas.

Course Description & Learning Objectives

The purpose of this course is to provide learners with a comprehensive overview of the field of neuroscience, with a focus on neuroanatomy. We will explore how the nervous system functions in health and disease. We will learn through lectures, laboratory, digital imaging tools, and clinical case studies. The course is organized into five units with learning objectives listed below and in your course packet.

Unit I - Cellular & Physiological Foundations

- Describe the organizational plan of the nervous system and the anatomical terms and planes used to study it.
- Explain the structure and function of neurons and glia, including mechanisms of neuronal injury.
- Describe how neurons generate and propagate electrical signals and communicate at synapses.
- Explain the physiological basis of neuroplasticity, including long-term potentiation, as the foundation for learning and memory.

Unit II – Anatomy & Imaging of the Brain

- Describe the vascular supply, coverings, and fluid systems (meninges, CSF, ventricles) of the brain.
- Identify the major external and internal structures of the brain using both three-dimensional (gross tissue/models) and two-dimensional (cross-sectional) images.
- Compare neuroimaging modalities, including CT, MRI, fMRI, and DTI/white matter tractography, and their clinical applications.
- Describe the major functions of the central and autonomic nervous systems.

Unit III - Spinal Cord, Brainstem, & Cranial Nerves

- Describe the anatomy of the spinal cord and brainstem in both 3D and cross-sectional views.
- Trace major ascending and descending spinal cord pathways and identify the structures they pass through at each level of the CNS.
- Identify the cranial nerves, their functions, and the location of their nuclei within the brainstem.
- Apply neuroanatomical knowledge to the cranial nerve exam.

Unit IV – Special Senses & Brainstem Integration

- Describe the neurobiology and central pathways of the special senses (vision, olfaction, gustation, vestibular, and auditory systems).
- Trace the pathways of the cranial nerves within the brainstem, including key reflex circuits (e.g., MLF, corticobulbar, trigeminal).
- Relate disruptions in sensory processing to clinical disorders of sensation or perception.

Unit V - Motor, Limbic & Integrative Systems

- Compare and contrast the anatomy and function of the major motor systems (motor cortex, cerebellum, basal ganglia).
- Describe the structure and function of the limbic system, including the biological basis of memory, emotion, and reward.
- Explain the neural basis of sleep and arousal, including EEG correlates and the reticular formation.
- Describe the eight categories of neuropathology (VITAMIN-C) with representative examples of each.

Grading Policy

At the end of the course, a final letter grade will be assigned according to the MHA program scale. As per program policy, a minimum grade of B- is required for successful completion of the course.

A	93-100%	B-	80-82%	D+	67-69%
A-	90-92%	C+	77-79%	D	63-66
B+	87-89%	C	73-76%	D-	60-62
B	83-86%	C-	70-72%	F	59% and below

Assessment	Final Grade Contribution (%)
Exam 1 (covers Lectures 1-8 & Lab 1)	18
Exam 2 (covers Lectures 9-15 & Labs 2-3)	18
Exam 3 (covers Lectures 16-24 & Labs 4-6)	18
Exam 4 (covers Lectures 25-32 & Labs 7-9)	18
Exam 5 (covers Lectures 33-40 & Labs 10-11)	18
Cumulative FINAL Exam	Pass/Fail
Weekly Lab Prep: videos, modules, & quizzes to prepare for labs or lectures	5
Presentation: "Neuroscience in the News"	5
TOTAL	100%

Exams (90% of grade):

Exams will consist of multiple choice and will incorporate material from lectures and labs. There is not a separate practical exam for the labs. Lab material will be incorporated into the lecture exams in the form of diagrams, photographs, and pictures. A proctored, optional Exam Review Session will be offered after each exam.

Practice Quizzes (not graded):

One key to success in graduate school is to self-evaluate your own learning. To help reach this goal, there will be weekly non-graded practice quizzes that test material from the previous week. Good scores on practice quizzes indicate high probability of success for the exam. Poor scores on practice quizzes indicate low probability of exam success. If a student consistently performs poorly on practice quizzes, an appointment with the instructor or TAs should be arranged to discuss study strategies.

Lab Prep Assignments (5% of grade):

Lab Prep Assignments (via Canvas) are designed to prepare learners for lab, and are due prior to lab.

Extra Credit Module

The Neuroanatomy Pre-course module is optional but worth 2% extra credit on the 3rd exam.

<https://cusom.h5p.com/content/1292336593565472108>

“Neuroscience in the News” (NIN) Presentation (5% of grade):

Researchers continue to advance the field of neuroscience to further our understanding of how the nervous system functions and what happens when parts of this system fail. As these breakthroughs occur, science writers take primary science literature and compose news articles that enable the general public to understand these discoveries and their significance to society. To help you draw parallels between inside and outside the classroom, you will present to the class a news article (from the local news source or magazine) that reports a story involving some aspect of the nervous system related to health or disease. You will present as teams determined at the beginning of the course. You can choose a topic covered in class or a topic related to the nervous system that will not be covered in class but is of interest to you. You must post on Canvas (under “Discussions”) the title, link, and short description of your article one week BEFORE your designated presentation. The NIN presentation should be 15 minutes with an additional 5 minutes for questions and include the following:

1. What is the title of the article or news report? Where and when was it published? If you were to describe this article in a few words, what keywords or phrases would you use?
2. What is the major finding in the news story? Give a brief synopsis of the report and/or discovery.
3. What is the basic science underlying the finding? What part of the brain or nervous system is involved? What is the pathway or mechanism? You may need to *teach* the class a new anatomical relationship and/or a neural pathway that was *not* covered in class. If the topic was already covered in class, then you should acknowledge when the topic was covered and *review* the information for the class. In addition, you must look up the **original research article** (NOT a review article) on pubmed.gov, read the article, and then use this article to describe the scientific aspects of the study in further detail than what was provided in the news report.
4. Identify and highlight one controversy or discussion question for the class associated with the science in your article. Lead a discussion based on your questions.
5. The presentation overall should be clear, prepared, and well-organized.

“Neuroscience in the News” Grading Rubric (100 points total)

(Student = team)

Part 1 (5 points) Title/Intro of News Article

4 points: News Article Intro: Student mentions title (1 point), news source (1 point), the publication date (1 point) and pastes the URL (1 point) of the news article. NO REVIEW PAPERS.

1 point: Student gives 2-3 keywords or phrases that describe the topic.

Part 2 (15 points) Synopsis of News Article

10 points: Student gives thorough and clear synopsis of the main points of the news story as it was presented to the public.

5 points: Student relates the topic back to information that was taught in class (reminding students when that material was taught and reviewing it). The student acknowledges if the material was not yet taught in class and teaches it new to the class.

Part 3 (60 points) The Original Research and the Science Behind It

4 points: Original Article Intro: Student has gone to www.pubmed.gov and found the original article. Student mentions title (1 point), journal name (1 point), the publication date (1 point) and pastes the URL (1 point) of the original research article.

2 points: Student lists the scientists (1 point) and institutions (1 point) where the research was performed (note: this is not the author of the news article; this is where & by whom the research was done).

10 points: Student correctly identifies the *part of the brain or nervous system* involved in the article.

10 points: Student correctly describes the *mechanism involved*. This may include a description of a biochemical pathway, a reaction, or a physiolo

gical circuit. You will likely have to look up additional information for this section! Do your research! Teach the class!

10 points: Student presents relevant tables and/or figures from the original article that enhances our understanding of the research. The tables and/or figures are described clearly and succinctly. Please ensure you tell your audience what is being plotted on the y and x-axis before you talk about the results of the figure.

4 points: References (including the figure #) are listed below any tables or figures that come straight from the original article.

5 points: Student critiques the original research article for strengths and weaknesses. Student is able to identify potential flaws or excellence in the experimental design.

5 points: Student uploads the news article and the original research article to an AI large language model and asks AI to “perform a comparative analysis of the news article vs. the original research article”. Then, determine if AI missed any important differences in terms of how the science was represented to the public, based on your own reading of the two articles.

10 points: Student demonstrates mastery of the article and the neuroscience material.

Part 4: (10 points) Discussion

5 points: Student identifies at least ONE discussion question related to the research.

5 points: Student stimulates class participation and discussion on the research.

Part 5: (10 points) Overall preparedness

2 points: Student submits the title and link of both the news article and the original research article to Canvas at least one week before the scheduled presentation.

2 points: Student’s PowerPoint presentation is well-organized and well-designed (order of slides is logical, text is not too small, figures are legible, etc.).

2 points: Student starts and finishes on time (and stays within the 15-minute time limit).

2 points: Student presents without reading verbatim from PowerPoint slides.

2 points: Student has good stage presence, speaks clearly, and engages the class professionally.

Student Responsibilities & Expectations:

- Attend all in-person sessions and examination days. In the case of an emergency or illness, please contact the instructor as soon as possible by email.
- Arrive on time. In exchange, the instructor will finish on time.
- Turn off/silence cell phones before class. Use phones only during the break.
- Stay curious and ask questions. If you do not understand something, it is likely that someone else in the class doesn't understand either. Help your classmates and ask!
- Pay attention. This class proceeds at a rapid pace. Study regularly. It is expected that you will do 1-2 hours of studying outside of class for every hour of lecture. Keep up with the material on a daily basis, take practice quizzes and attend review sessions.

Attendance & Make-ups

- Student success is intimately connected to collaboration between instructor and student. Participation in each class is crucial so that we can all grow together as we bring our insights and perspectives. As much as possible, please inform me in advance if you are not able to attend a particular session. Attendance at exams and NIN presentations is required.
- Make-ups for missing an exam will **NOT** be allowed except in the case of illness, funeral of family member, or a personal, catastrophic, or religious event (as defined by the University policies). Written documentation is required.
- A missed exam for any reason other than those listed above will be assigned a zero. A late "LAB PREP" assignment or "Neuroscience in the News" assignment for any reason other than the above listed will be automatically reduced by 10%, with an additional 10% deduction for each additional 24 hours late.

Honor Code

Students are expected to act professionally in accordance with the Graduate School's Student Academic Honor & Conduct Code and the MHA Honor Code, as well as policies outlined in the MHA Student Handbook. Academic dishonesty, including cheating or sharing exam details will not be tolerated. The removal of exam material, including scratch paper or any other form of notation, from the testing room is considered a violation of the academic honor code. In your laboratory sessions, you will have the opportunity to work with brains recovered from human donors. Please treat all donor tissue with gentleness and respect. Please adhere to all rules and guidelines in the lab.

Religious Accommodation Policy

Students who anticipate the necessity of being absent from class due to the observance of a major religious holiday must provide advance notice to the Course Director by email as soon as possible.

Disability Accommodation Policy

Students with documented learning and/or physical disabilities should inform the Course Director as soon as possible to discuss and arrange for accommodations. All reasonable efforts will be made to accommodate students with regard to note taking, reading assignments, and test taking by working with the ODAI office.

Equal Opportunity Policy

It is the goal of the University to maintain a work and study environment free of discrimination on the basis of age, race, sex, gender, marital status, religion, national origin, veteran status, or disability. It is a goal of this course to maintain an environment of respect for all.

Recording Policy

You are permitted to use tablets or laptop computers to take notes. In addition, you are permitted to video or audio record any or all lectures for the purpose of self-study. However, you are not permitted to use notes, tapes,

or other recorded data for the purposes of sale or web distribution. Photos of donated human tissue may never be posted on websites, AI platforms, or social media of any kind unless approved by the course director for direct educational or research use.

Resolution of Conflicts Policy

Good faith efforts will be made by students, faculty, and program and university administration to settle all appeals, complaints, and grievances on an informal basis. Such efforts include conferences between the persons directly involved and others who may help solve the problems. Formal conflict resolution policies are detailed in the policies and procedures of the Graduate School.

Course Enrollment Outcomes

There are five possible outcomes for each ANAT course in which a student enrolls each semester:

1. The student will receive a letter grade in the course.
2. The student may drop* the course on or before the add/drop deadline defined on the Graduate School's Academic Calendar.
3. The student may withdraw* from the course before the MHA withdrawal deadline.
4. The student may receive a grade of Incomplete* in the course.
5. The student may seek a Medical Leave of Absence* through the Office of Student Affairs.

***Please see the MHA Handbook for additional information.** Note that a drop, withdrawal, incomplete, or medical leave of absence may impact a student's ability to continue in the MHA Pathway to Dentistry Program, Certificate in Anatomical Education, Anatomical Imaging and Modeling Track, and/or may alter a student's estimated time to graduation. Students are encouraged to meet with their faculty academic advisor before altering their enrollment.

Add/Drop Date

Students may add/drop courses in UCDAccess until **September 11, 2026**. Students are not charged tuition for a dropped course and the course is not noted on the student's transcript.

Withdrawal Policy

Discontinuing enrollment in this course after the add/drop date will result in a withdrawal, a "W" grade will be posted on the student's transcript for that course, and the student will pay tuition. A grade of "W" does not impact a student's grade point average. **Withdrawals from an ANAT course must be done on or before Friday, November 13, 2026.**

Incomplete Policy

Incomplete (I) grades are not granted for low academic performance. To be eligible for an "I" grade, you must:

1. Successfully complete a majority of the course (3 out of 5 units).
2. Have a special circumstance(s) beyond your control that prevents you from attending class and/or completing coursework. Documentation is required.
3. Make arrangements to complete the missing coursework with the original instructor.
4. If the missing coursework is not completed within 1 year from the end of the semester in which the original course was scheduled, the "I" grade will convert to an "F" grade on your official transcript.
5. Refer to MHA Handbook for additional instructions.

Remediation Policy

Students who do not achieve an overall 80% (B-) grade required to fulfill the degree requirements of the MS Modern Human Anatomy program will be allowed one opportunity to remediate the course under the following conditions:

- The student's overall score at the end of the course must be between 77%-79.99% (C+) to qualify for remediation.
- The student must have a record of 80% attendance for lectures and 90% attendance for labs in courses that have labs (excepting formally excused absences).

Remediation will typically consist of a comprehensive written exam, and the student must earn at least 80% on that assessment to pass the course with a B-. However, the course directors reserve the right to revise the nature and format of the remediation to address individual cases as appropriate. If the student chooses not to take the remediation exam or does not score at least 80% on it, the student will receive the original grade earned at the end of the course.

Course Evaluations

Course evaluations will be administered at the end of the semester and are an important component of course improvement and your professional development as a graduate student. While your responses remain anonymous, we maintain a completion record (separate from the evaluation content) for administrative purposes. Feedback should be specific, constructive, and actionable. Completion of all MHA course evaluations is required for MHA student travel award eligibility.

Student Support

We care about you and your success. The CU Anschutz Medical Campus offers multiple resources and services to support you and your needs. The Office of Student Affairs—Student Support can help you navigate the academic and non-academic resources across campus. Please visit their website www.cuanschutz.edu/student

UNIT I - CELLULAR & PHYSIOLOGICAL FOUNDATIONS					
DATE	TIME	#	TOPIC	PRESENTER	LOCATION
WEEK 1					
9/1/2026	9:00 AM		Course Intro & NIN Project Info	Stabio	Ed2S-1307
	10:00 AM		NIN Expectations for Critical Review of Research	Caldwell	Ed2S-1307
9/3/2026	9:00 AM	1	Organization of the Nervous System	Finger	Ed2S-1307
	10:00 AM	2	Neurons	Caldwell	Ed2S-1307
WEEK 2					
9/8/2026	9:00 AM	3	Resting Membrane Potential	Stabio	Ed2S-1307
	10:00 AM	4	Action Potential	Stabio	Ed2S-1307
9/10/2026	9:00 AM	5	Synapses & Neuroplasticity	Stabio	Ed2S-1307
	10:00 AM	6	Glia	Hughes	Ed2S-1307
WEEK 3					
9/15/2026	9:00 AM	7	NT	Caldwell	Ed2S-1307
	10:00 AM	8	Receptors	Caldwell	Ed2S-1307
9/16/2026	1-3pm	LAB 1	Learning Lab: How the brain likes to learn	Stabio, Fridrich Pawlikowski, Ramziddin	Ed2S-2305
9/17/2026	9:00 AM		Exam 1 (8 lectures + lab?) + all prework!!	Caldwell	Ed2N-2201 DE
	10:00 AM		Exam 1 Review	Caldwell	Ed2N-2201 DE

UNIT II - ANATOMY & IMAGING OF THE BRAIN					
WEEK 4					
9/22/2026	9:00 AM	9	Blood Supply	Pawlikowski	Ed2S-1307
	10:00 AM	10	Meninges, CSF, Ventricles	Pawlikowski	Ed2S-1307
9/23/2026	1-4 PM	Lab 2	WET LAB: Brain Slicing Lab Lab prep due before lab (Maybe the body donation module)	Teaching Team (TAs + Faculty) Fridrich, Ramziddin	Ed 1: 5th floor anatomy labs
9/24/2026	9:00 AM	11	The Central Nervous System	Stabio	Ed2S-1307
	10:00 AM	12	The Autonomic Nervous System	Stabio	Ed2S-1307
WEEK 5					
9/29/2026	9:00 AM	13	Neuroimaging 1 - CT & MRI	Stabio	Ed2S-1307
	10:00 AM	14	Neuroimaging 2 - fMRI, dTI and white matter tracts	Stabio	Ed2S-1307
9/30/2026	1-3pm	Lab 3	DRY LAB: Build-a-Brain 3D MRI Lab	Stabio	Ed2S-2305
10/1/2026	9:00 AM	15	Cross-Sectional Neuroanatomy Review	Stabio	Ed2S-1307
	10:00 AM	NIN	Neuroscience in the News Presentations 1-2	Caldwell	Ed2S-1307
WEEK 6					
10/6/2026	9:00 AM		Exam 2 (7 lectures + 2 labs) + all prework!!	Stabio	Ed2N-2201 DE
	10:00 AM		Exam 2 Review	Stabio	Ed2N-2201 DE

UNIT III - SPINAL CORD, BRAINSTEM, & CRANIAL NERVES					
WEEK 6 (continued)					
10/8/2026	9:00 AM	16	Brainstem Anatomy	Stabio	Ed2S-1307
	10:00 AM	17	Brainstem Cross-Sections	Stabio	Ed2S-1307
WEEK 7					
10/13/2026	9:00 AM	18	Spinal Cord Anatomy, Cross-sections & reflexes	Stabio	Ed2S-1307
	10:00 AM	NIN	Neuroscience in the News Presentations 3-4	Caldwell	Ed2S-1307
10/14/2026	1-3 PM	Lab 4	WET LAB: Spinal Cord & Brainstem LAB PREP DUE BEFORE LAB.	Teaching Team (TAs + Faculty) Fridrich, Ramziddin	Ed 1: 5th floor anatomy labs & Plastination room
10/15/2026	9:00 AM	19	Somatosensation: Fine touch, proprioception & vibration	Finger	Ed2S-1307
	10:00 AM	20	Somatosensation: Pain & Temperature	Finger	Ed2S-1307
WEEK 8					
10/20/2026	9:00 AM	21	Corticospinal Pathway	Stabio	Ed2S-1307
	10:00 AM	22	Cross-Sectional Neuroanatomy Review	Stabio	Ed2S-1307
10/21/2026	1-3 PM	Lab 5	DRY LAB: Lesion Localization of the Spinal Cord	Stabio; Ramziddin	Ed2S-2305
10/22/2026	9:00 AM	23	Cranial Nerves	Fridrich	Ed2S-1307
	10:00 AM	24	Cranial Nerve Nuclei & Reflexes	Stabio	Ed2S-1307
WEEK 9					
10/27/2026	9:00 AM	NIN	Neuroscience in the News #5-6	Caldwell	Ed2S-1307
	10:00 AM	NIN	Neuroscience in the News #7-8	Caldwell	Ed2S-1307
10/28/2026	1-3 PM	Lab 6	DRY LAB: Cranial Nerve Lab (Intro)	Stabio, Fridrich, Ramziddin	Ed2S-2305
10/29/2026	9:00 AM		Exam 3 (9 lectures, 3 labs) + all prework!!	Stabio	Ed2N-2201 DE
	10:00 AM		Exam 3 Review	Stabio	Ed2N-2201 DE

UNIT IV - SPECIAL SENSES & BRAINSTEM INTEGRATION					
WEEK 10					
11/3/2026	9:00 AM	25	Overview of Special Senses	Stabio	Ed2S-1307
	10:00 AM	26	Visual System	Stabio	Ed2S-1307
11/4/2026	1:00 PM	NIN	Neuroscience in the News #9-10	Caldwell	Ed2S-2305
	2:00 PM	NIN	Neuroscience in the News #11-12	Caldwell	
11/5/2026	9:00 AM	27	Olfactory System	Finger	Ed2S-1307
	10:00 AM	28	Gustatory System	Finger	Ed2S-1307
WEEK 11					
11/10/2026	9:00 AM	29	Vestibular System	Caldwell	Ed2S-1307
	10:00 AM	30	Auditory System	Finger	Ed2S-1307
11/11/2026	1-3 PM	Lab 7	Special Senses Lab with Ultrasound LAB PREP DUE BEFORE LAB.	Teaching Team (TAs + Faculty) Fridrich, Ramziddin	Ed2S-2305
11/12/2026	9:00 AM	31	Brainstem Pathways & Reflexes	Stabio	Ed2S-1307
	10:00 AM	32	Brainstem Pathways & Reflexes	Stabio	Ed2S-1307
WEEK 12					
11/17/2026	9 - 11am	Lab 8	BRAINSTEM CASES LAB	Stabio, Ramziddin	Ed2S-1307
11/18/2026	1-3 PM	Lab 9	DISORDERS OF PERCEPTION	Stabio	Ed2S-2305
11/19/2026	9:00 AM		Exam 4 (8 lectures, 4 labs) + all prework!!	Caldwell	Ed2N-2201 DE
	10:00 AM		Exam Review	Caldwell	Ed2N-2201 DE

UNIT V - MOTOR, LIMBIC & INTEGRATIVE SYSTEMS					
WEEK 13					
11/24/2026	remote video	33	Overview of Motor Systems Video	Caldwell	
11/26/2026			THANKSGIVING HOLIDAY - NO SCHOOL		
WEEK 14					
12/1/2026	9:00 AM	34	Basal Ganglia	Caldwell	Ed2S-1307
	10:00 AM	35	Cerebellum	Niemeyer	Ed2S-1307
12/2/2026	1-3 PM	Lab 10	DRY LAB: Motor Systems (add backyard brains) LAB PREP DUE BEFORE LAB.	Niemeyer & Caldwell	Ed2S-2305
12/3/2026	9:00 AM	36	Limbic System	Stabio	Ed2S-1307
	10:00 AM	37	Meso-Systems	Alor	Ed2S-1307
WEEK 15					
12/8/2026	9:00 AM	38	EEG & Sleep	Caldwell	Ed2S-1307
	10:00 AM	39	Neuropathology Review	Pawlikowski	Ed2S-1307
12/9/2026	1-3 PM	Lab 11	WET LAB: Neuropathology Lab LAB PREP DUE BEFORE LAB.	Teaching Team (TAs + Faculty); Fridrich, Ramziddin	RC1 North MPL LABS 1406, 1407, 1408
12/10/2026	9:00 AM	40	Emerging Neurotechnologies	Stabio	Ed2S-1307
	10:00 AM		Final Course Review	Stabio	Ed2S-1307
WEEK 16					
12/17/2026	9:00 AM		Exam 5 (8 lectures + 2 labs)	Caldwell	Ed2N-2201 DE
	11:00 AM		Cummulative exam - pass/fail only	Caldwell	Ed2N-2201 DE

UNIT I:

CELLULAR & PHYSIOLOGICAL FOUNDATIONS

Lecture 1: Nervous System Organization

Tom Finger, PhD – Department of Cell & Developmental Biology

Optional Foundational Reading

Anatomical Terminology (Ch. 21) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/anatomical-terminology/>

Development (Ch. 22) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/brain-structure-differentiation/>

Learning Objectives

1. Describe the developmental origins of the nervous system and correlate each embryonic division with its adult structure.
2. Use terminology for anatomical directions and planes of section as applied to the neuraxis, including the flexure that reorients the rostral and caudal axes.
3. Describe the structural divisions of the CNS and PNS and their distinguishing features, including gray versus white matter and the paired terms nucleus/ganglion and tract/nerve.
4. Describe the functional divisions of the PNS, including sensory (afferent) and motor (efferent).
5. Describe the cellular/histological divisions of the CNS and what are “Brodman Areas” (note you only need to know Areas 3, 1, 2; 4; 17; and 41 in this course)
6. Differentiate three types of maps used in functional neuroanatomy: dermatomes, myotomes, and the cortical homunculus.

Terms to Know

DIVISIONS:

- Developmental Divisions of the Nervous System
 - Early development & relationship to adult divisions
 - Neural tube and neural crest - The neural tube gives rise to the brain and spinal cord (CNS) while the neural crest generates all of the ganglia of the peripheral nervous system. Neural crest cells are multipotent and generate not only the peripheral ganglia, but also give rise to all the pigment cells in the epithelium, many of the skeletal elements of the cranium, teeth, and parts of the eye, among other features.
 - Alar plate and basal plate – alar plate gives rise to sensory neurons; basal plate gives rise to motor neurons.
 - Prosencephalon (Forebrain)
 - Telencephalon
 - Diencephalon
 - Brainstem
 - Mesencephalon
 - Rhombencephalon
 - Metencephalon
 - Myelencephalon
- Structural Divisions of the Nervous system
 - Central Nervous System (CNS) = cerebrum, brainstem, cerebellum, and spinal cord
 - Peripheral Nervous System (PNS) = cranial nerves and spinal nerves
- Functional Divisions of the PNS
 - Somatic
 - Sensory (afferent)
 - Motor (efferent) – Voluntary

- Visceral
 - Sensory (afferent)
 - Motor (efferent) – involuntary or “Autonomic Nervous System”
 - Sympathetic – “fight or flight”
 - Parasympathetic – “rest and digest”
- Histological divisions
 - Grey Matter vs. White Matter
 - Brodmann Numbers

DIRECTIONS:

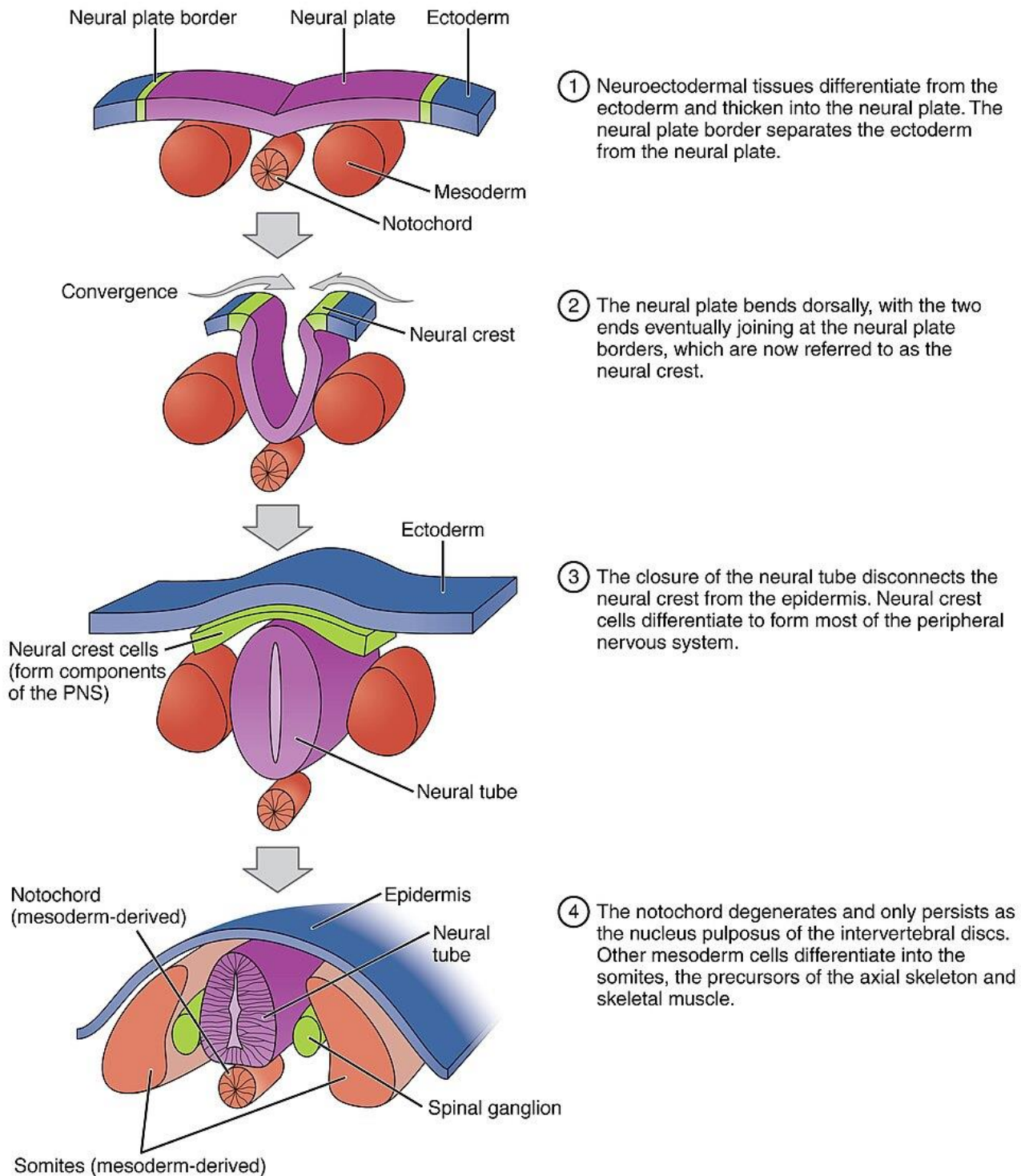
- Anatomical Terms
 - Dorsal vs Ventral
 - Anterior vs Posterior
 - Rostral vs Caudal
 - Superior vs Inferior
 - Lateral vs Medial
 - Ipsilateral vs Contralateral
- Planes of Section
 - Sagittal
 - Coronal
 - Horizontal (axial)

MAPS:

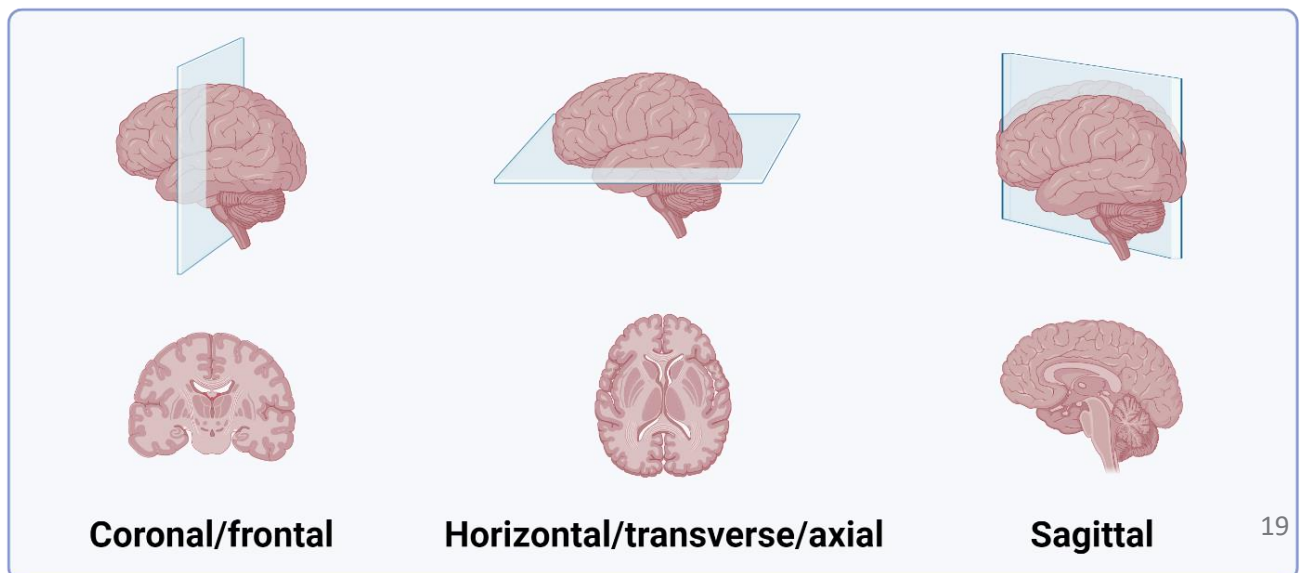
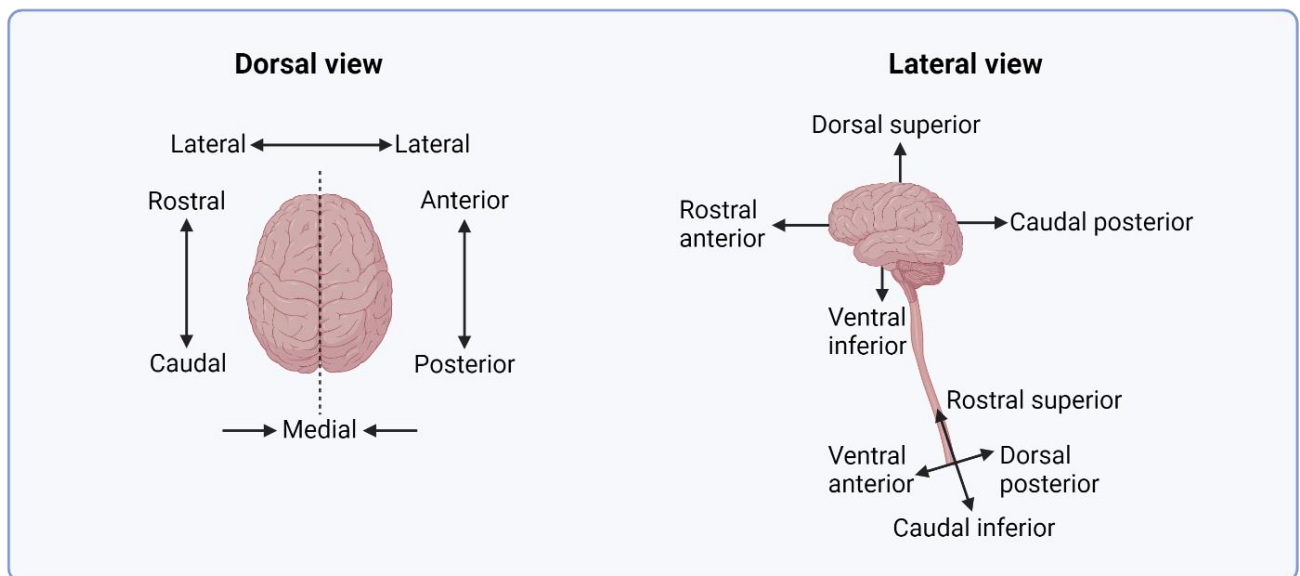
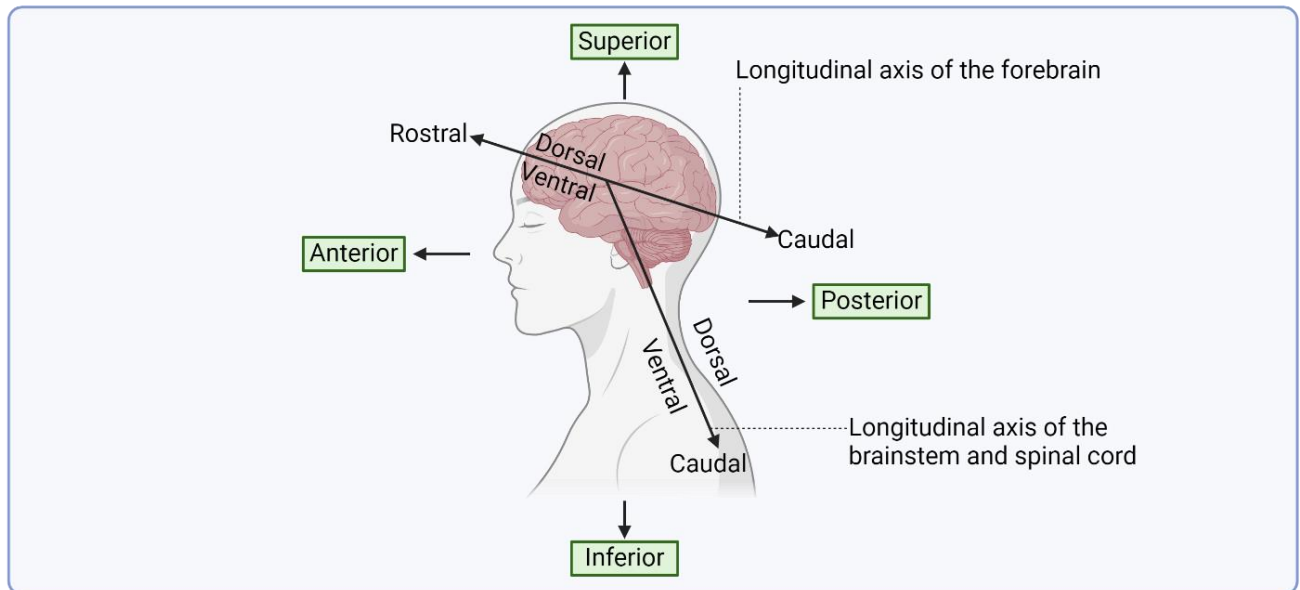
- Dermatomes (somatosensory maps)
- Myotomes (motor maps)
- Homunculus (cortical maps)

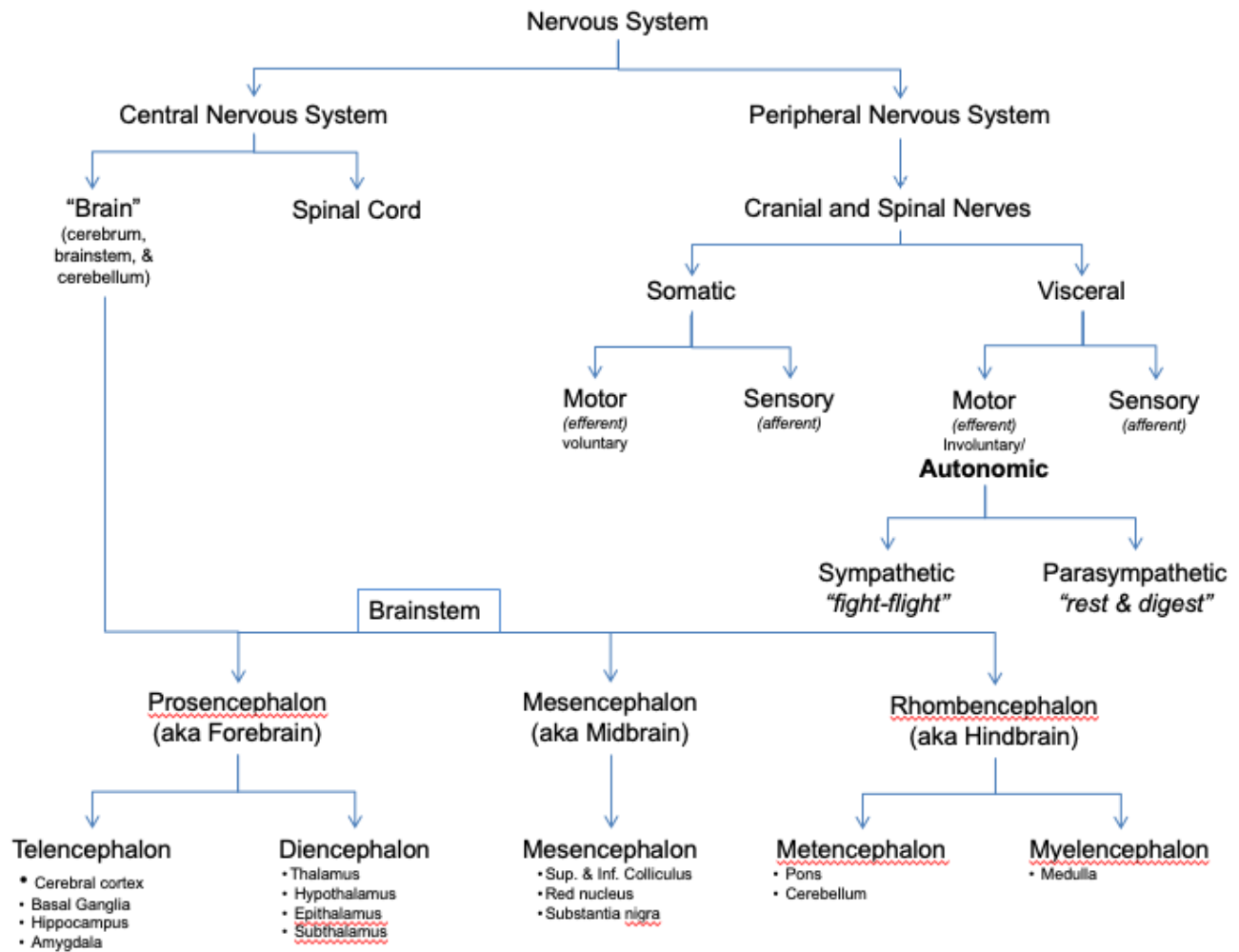
INFORMATION FLOW:

1. **Separation of Modalities:** Different modalities are carried in different channels (tracts)
2. **Parallel Processing:** Sensory information is distributed into different processing streams, e.g., pitch & location for sound, color & movement for vision, etc.
3. **Hierarchical Processing:** Information is relayed through a series of processing areas (nuclei), becoming more complex as one ascends in the system.
4. **Mapping of Functions:** Sensory inputs are mapped within each brain area, e.g. somatosensory information from the foot is carried and processed in different parts of a nucleus or cortex.

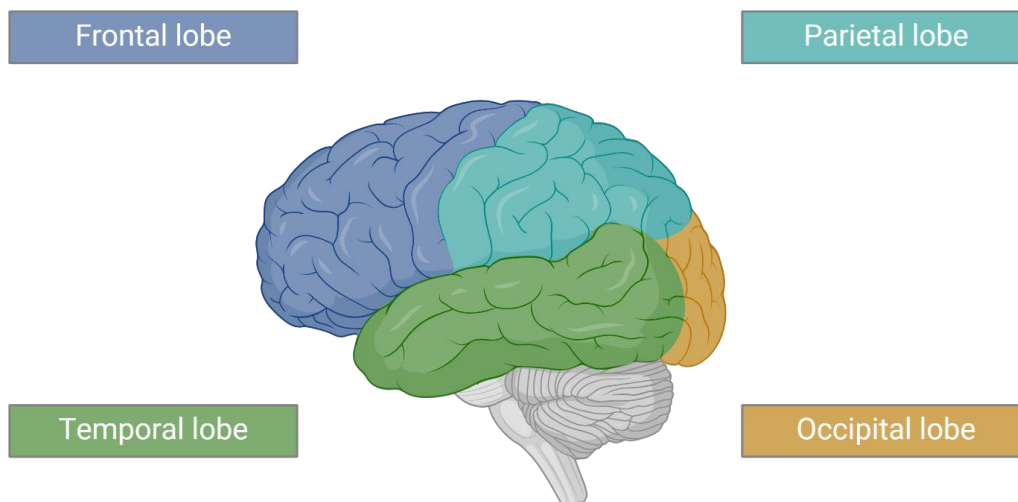


Neuroanatomical Directions and Planes





Lobes of the Brain



Lecture 2: Neurons

John Caldwell, PhD – Department of Cell & Developmental Biology

Optional Foundational Reading

The Neuron (Ch. 2) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/the-neuron/>

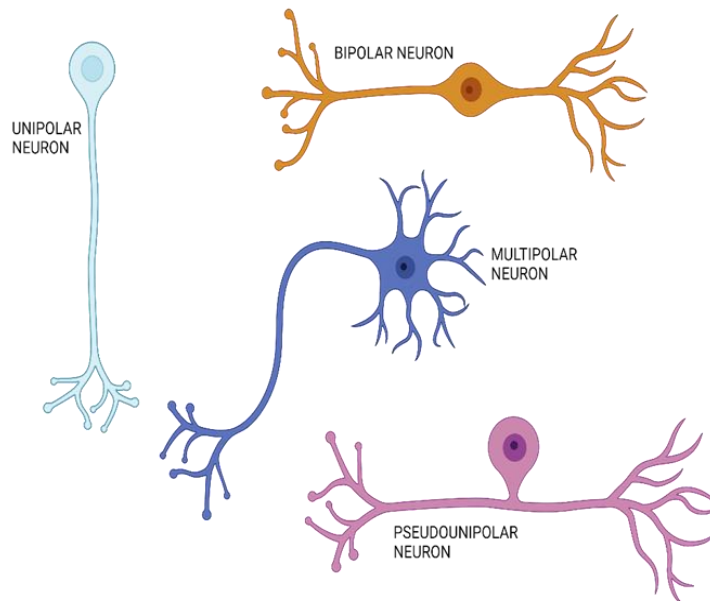
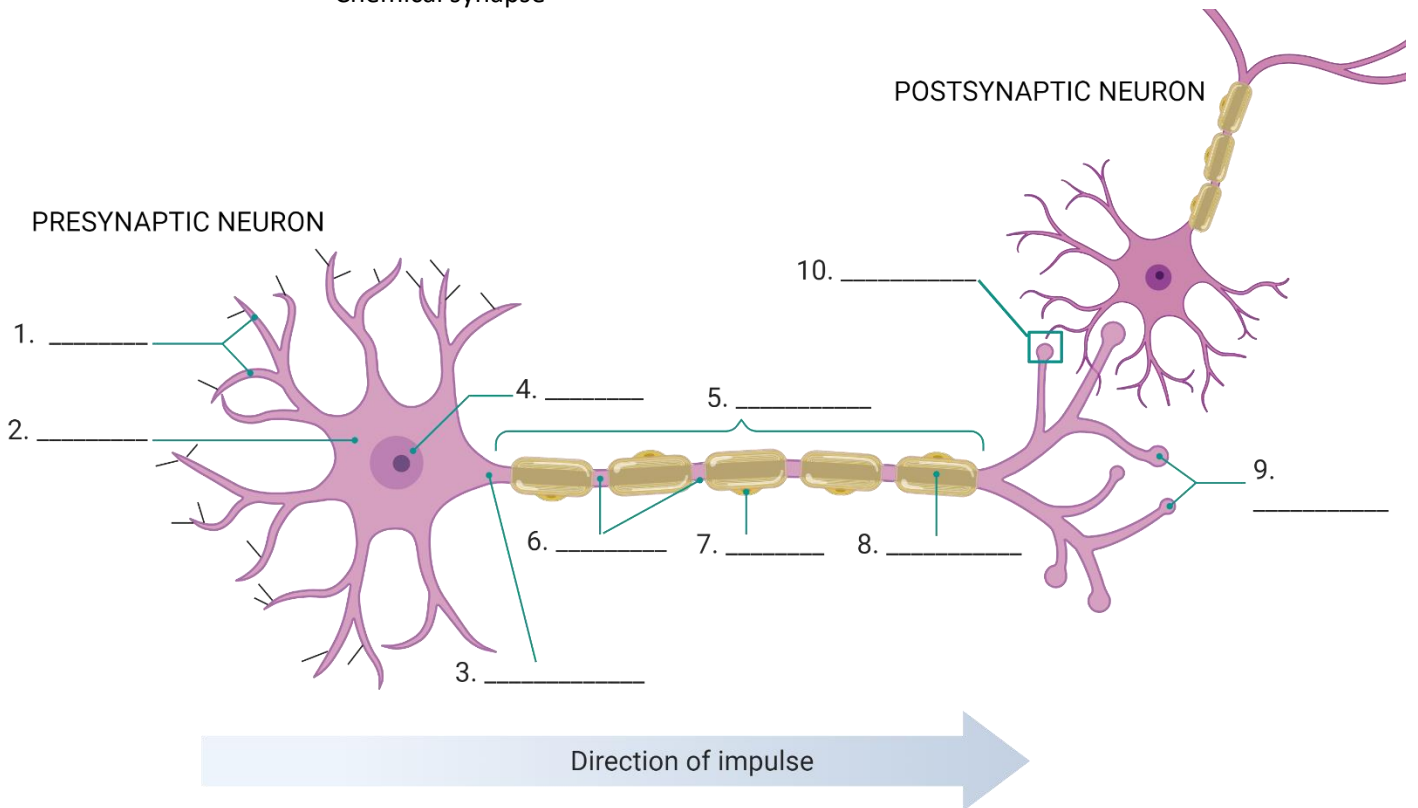
Learning Objectives

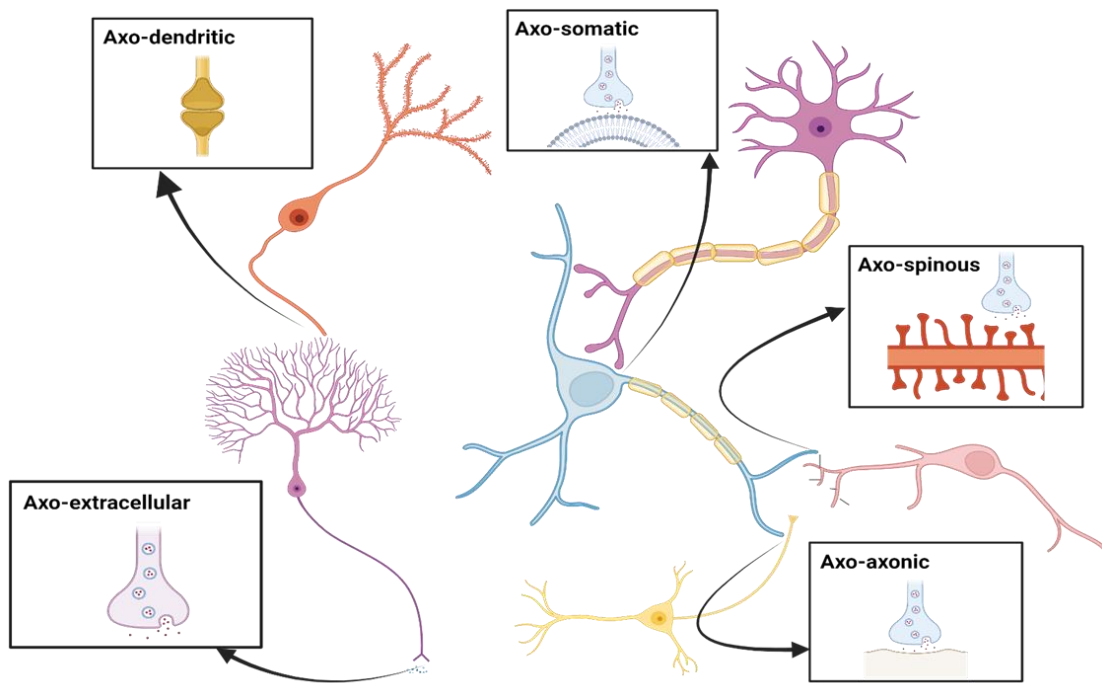
1. Draw a typical neuron and label all of its parts.
2. List the structural & functional classifications of neurons and recognize their distinguishing features from a diagram.
3. List the structural and functional classification of synapses and recognize their distinguishing features from a diagram.
4. Describe the six cortical layers, including which are the major input (4) and output (5) layers.
5. Describe the horizontal and vertical organization of the cortex (layers and columns) and their anatomical and functional input and output.
6. Describe how a neuron responds to injury (necrosis and chromatolysis) and how an axon responds to injury via Wallerian degeneration.
7. Describe and contrast the ability of the CNS versus PNS axons to regenerate after injury.

Terms to Know

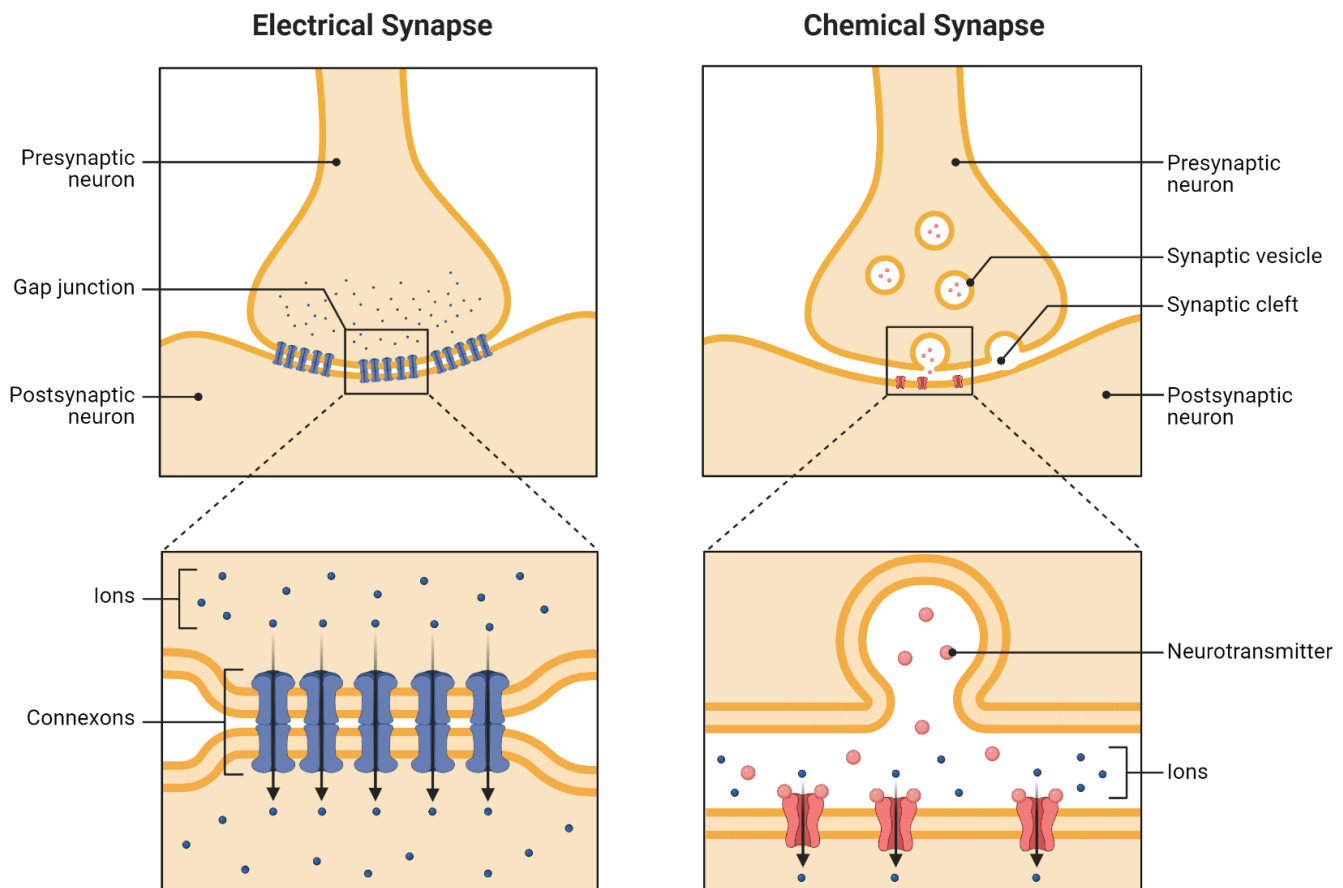
- Neuron
 - Soma (cell body)
 - Dendrites
 - Dendritic spine
 - Axon
 - Axon hillock
 - Terminal button (synaptic terminal)
 - Synapse
 - Neurotransmitter
 - Pre-synaptic neuron
 - Post-synaptic neuron
 - Myelin sheath
 - Nodes of Ranvier
- Classifications of Neurons
 - Structural classifications of Neurons
 - Unipolar
 - Bipolar
 - Multipolar
 - Pseudounipolar
 - Functional classification of neurons
 - Afferent
 - Efferent
 - Interneuron
- Classifications of synapses
 - Structural classification of synapses
 - Axo-dendritic

- Axo-spinous
- Axo-somatic
- Axo-axonic
- Functional classification of synapses
 - Electrical synapse (gap junctions)
 - Chemical synapse





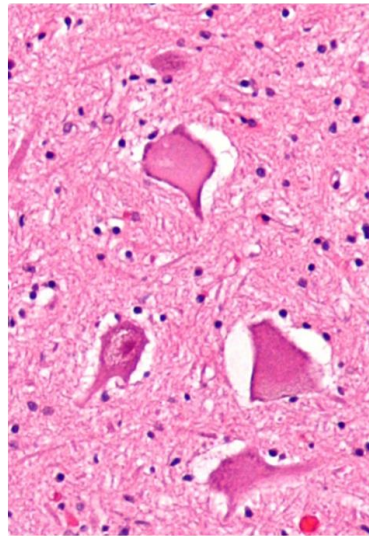
Electrical Synapses vs. Chemical Synapses



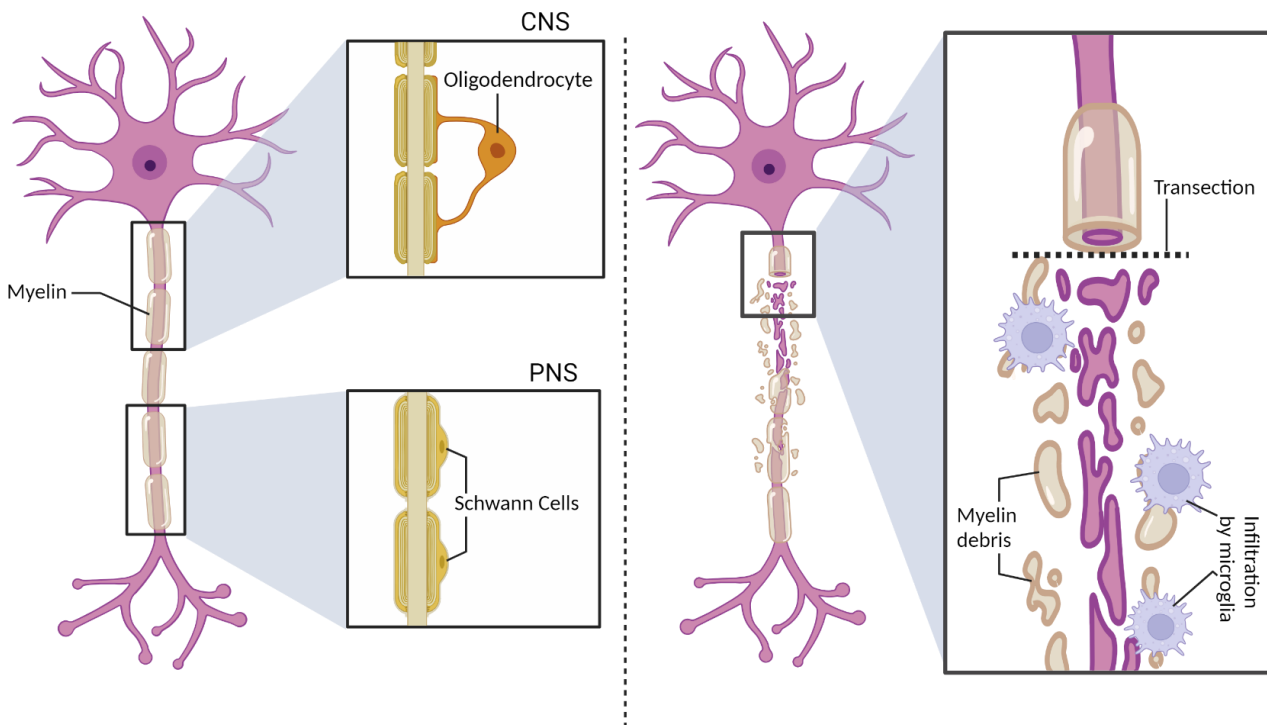
WHAT HAPPENS WHEN A NEURON GETS INJURED?

CHROMATOLYSIS

- Round cellular swelling
- Nucleus displacement to periphery
- Nissl substance dispersion in cytoplasm



WALLERIAN DEGENERATION



Lecture 3: Resting Membrane Potential

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Ion Movement (Ch. 5) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/ion-movement/>

Membrane Potential (Ch. 6) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/membrane-potential/>

Resting Membrane Potential (Ch. 7) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/the-membrane-at-rest/>

Learning Objectives

1. Describe the membrane properties of a typical neuron.
2. Compare and contrast the ionic environment inside and outside a typical mammalian neuron.
3. Define the equilibrium potential (Nernst Equation) and resting membrane potential (Goldman Equation) of a typical neuron and describe how they are established.
4. Compare and contrast driving force and conductance.
5. Describe the role of the Na⁺-K⁺ pump in a typical neuron (how, if at all, does it contribute to the RMP?).

Terms to Know

- Molecular properties of the lipid bilayer membrane
 - Passive/leaky ion channels (passive transport)
 - Voltage-gated channels
 - Voltage-gated sodium channel
 - Voltage-gated potassium channel
 - Pumps (active transport)
 - Sodium-potassium pump
 - Extracellular fluid vs intracellular fluid
 - Concentration gradient vs electrical gradient
- Electrical properties of membrane
 - Ohm's Law $V=IR$, where V = voltage; I = current; R = resistance
 - Driving force (DF)
 - Conductance (g)
 - Capacitance (C)

- Equilibrium Potential (E_{ion})

- Nernst Equation

- R is the gas constant = 8.314 J/mol*K
- T is the absolute temperature in Kelvin
- Z is the charge on the ion (watch the sign on this!)
- F is the Faraday constant (the amount of electricity required to chemically alter one gram equivalent weight of reacting material) = 96,500 coulombs

$$E_{ion} = \frac{RT}{zF} \ln \frac{[ion]_{out}}{[ion]_{in}}$$

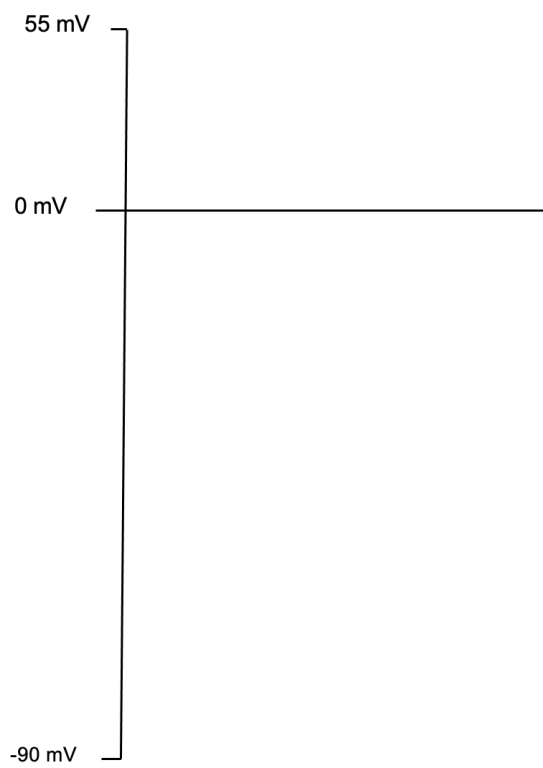
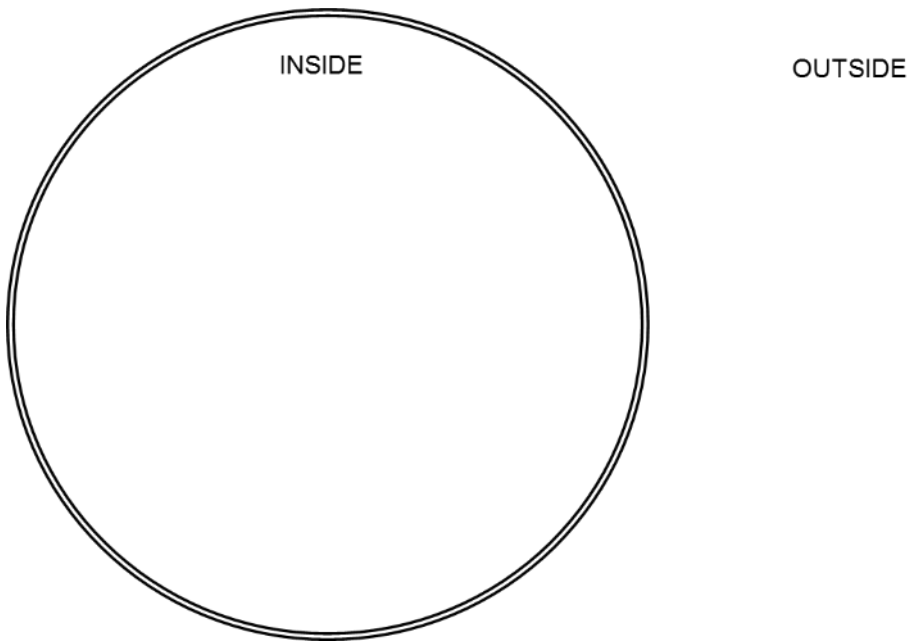
- simpler Nernst equation for 37°C (converted to log)

$$E_{ion} = \frac{61}{z} \log \frac{[ion]_{out}}{[ion]_{in}}$$

- Resting membrane potential (V_m)

- Goldman Equation = considers all ions in the system and their conductance (g)

$$V_m = \frac{(g_{Na})(E_{Na}) + (g_K)(E_K) + (g_{Cl})(E_{Cl})}{g_{Na} + g_K + g_{Cl}}$$



Lecture 4: Action Potential

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Electrical Activity (Ch. 8) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/postsynaptic-potentials/>

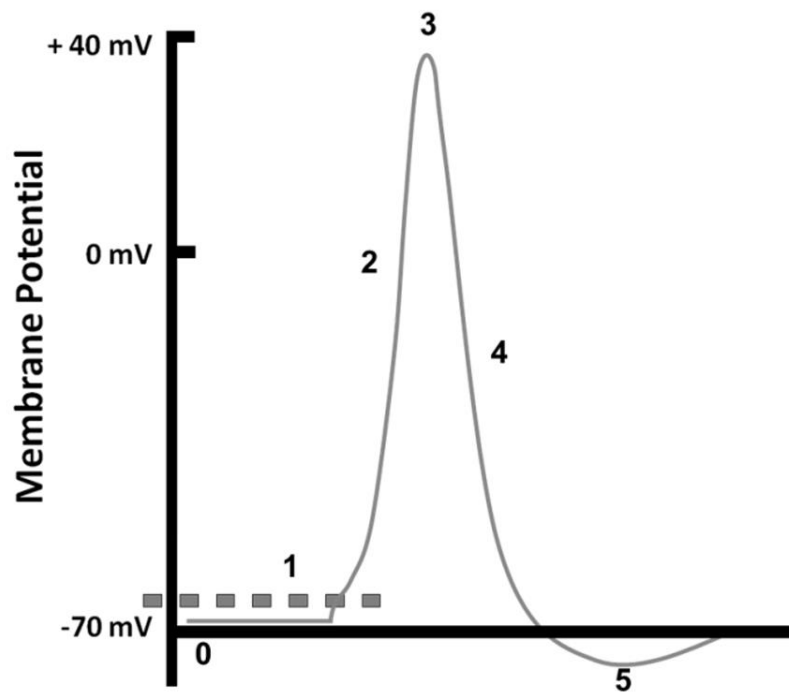
Action Potentials (Ch. 9) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/action-potentials/>

Learning Objectives

1. Describe the action potential (AP), draw an AP voltage response, and label its various parts.
2. Describe the movement of ions, as well as the status of voltage-gated channels, at different stages of the action potential.
3. Define the conduction principles of neurons, the “all or none” law of axonal conduction, and the “rate law” of neural coding.
4. Distinguish between relative and absolute refractory period in terms of the gate-state of voltage-gated sodium channels.
5. Describe the kinetics of the sodium-potassium pump after an action potential. Does it speed up or slow down?
6. Define “saltatory conduction” and describe the role of myelin in this type of conduction.

Terms to Know

- Steps of action potential
 - Threshold
 - Depolarization
 - Reversal
 - Repolarization
 - Hyperpolarization
- Conduction principles
 - All-or-none law
 - Rate law
 - Refractory period
 - Absolute refractory period
 - Relative refractory period
- Saltatory conduction
 - Capacitance
 - Myelin
 - Nodes of Ranvier



- 1.
- 2.
- 3.
- 4.
- 5.

Stage	Voltage-Gated Na ⁺ channel	Activation gate	In-activation gate	Voltage-Gated K ⁺ channel	Activation gate
Rest					
Depolarization					
Repolarization					
Hyper-polarization					

The Rate Law	The All – or – <u>None</u> Law

Unmyelinated axon = _____ conduction



Myelinated axon = _____ conduction



Lecture 5: Synapses & Neuroplasticity

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Synapse Structure (Ch. 11) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/synapse-structure/>

Synapse Steps (Ch. 12) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/neurotransmitter-release/>

Postsynaptic Potentials (Ch. 8) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/postsynaptic-potentials/>

LTP & LTD (Ch. 68) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/molecular-mechanisms-of-memory-hippocampus/>

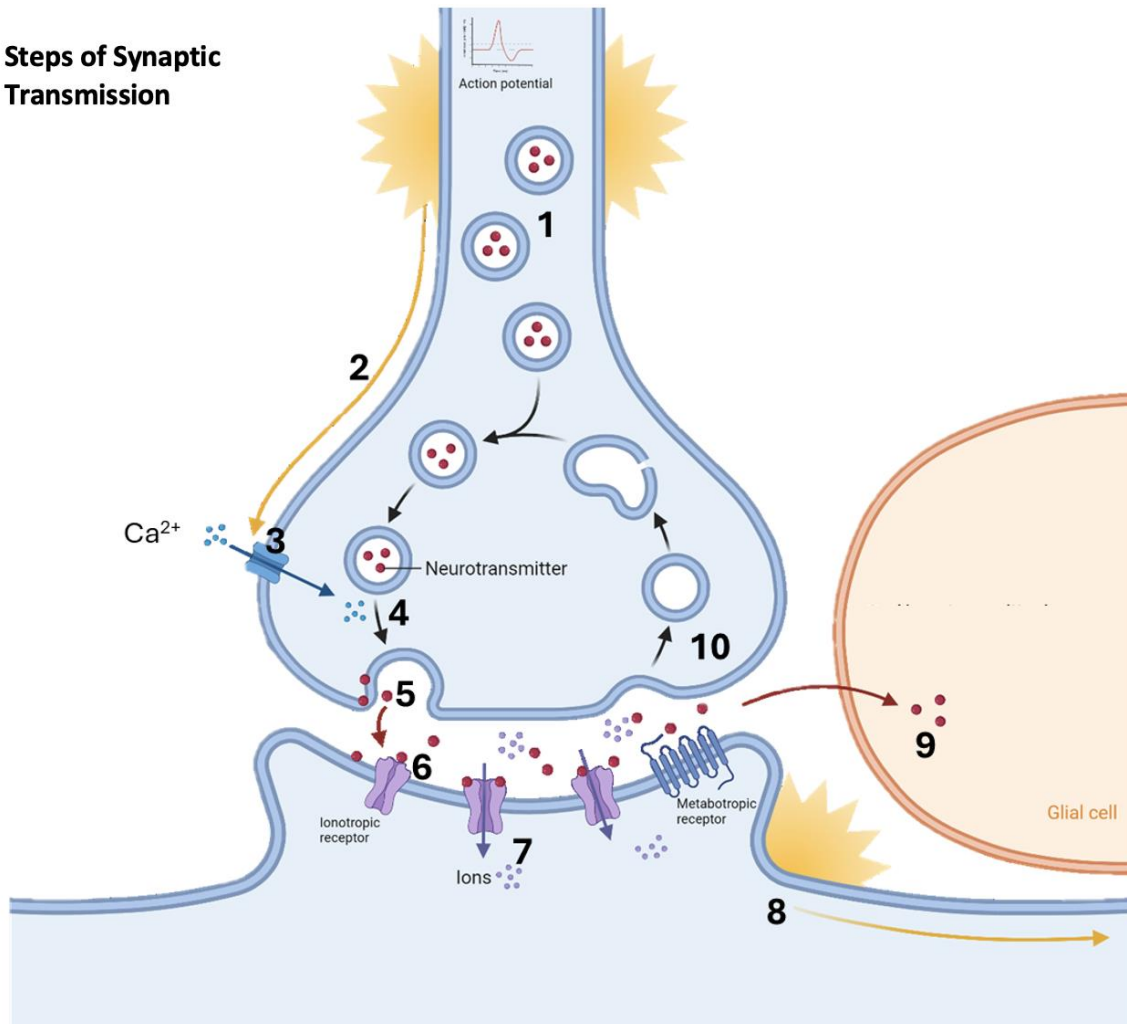
Learning Objectives

1. Recall the differences between an electrical synapse and a chemical synapse.
2. Describe the structural components of a typical chemical synapse
3. List the sequence of events during synaptic transmission, including the specific role of calcium.
4. Explain three ways that neurotransmitters are removed from the synaptic cleft.
5. Describe the mechanisms of botulinum toxin at the NMJ and tetanus toxin at inhibitory synapses in the spinal cord. What step of the synapse do they inhibit?
6. Compare and contrast EPSP and IPSP.
7. Describe neural integration (spatial vs temporal summation) and the role of the axon hillock.
8. Explain neuroplasticity, including synaptic plasticity (changes in NT & receptors) and structural plasticity (changes in dendrites and spines).
9. Explain long-term potentiation (LTP) and long-term depression (LTD) including the ion channels involved.

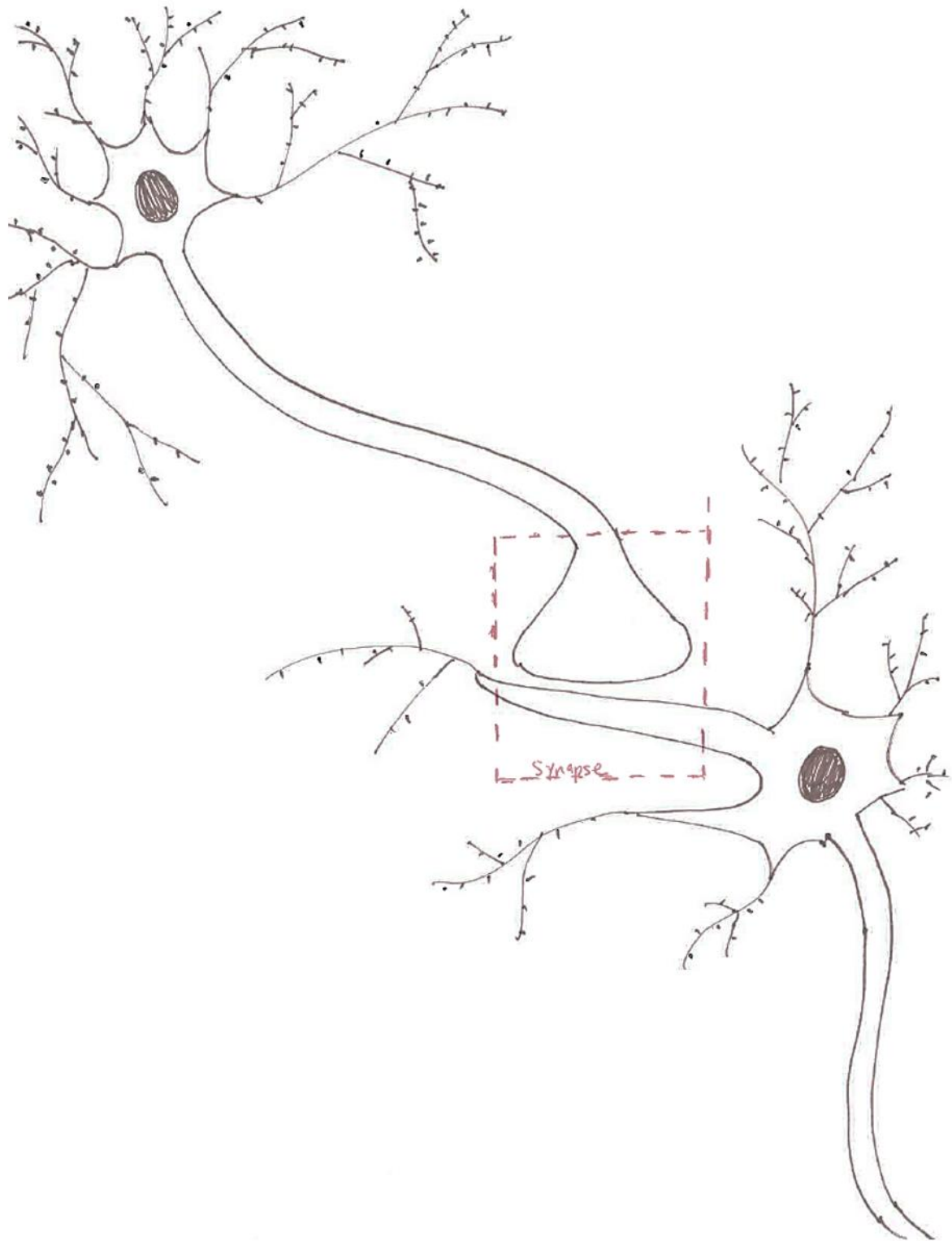
Terms to Know

- The Synapse Structure
 - Presynaptic vs. postsynaptic membrane
 - Synaptic cleft
 - Synaptic vesicles
 - Neurotransmitter
- Steps of an Electrochemical Synapse
 - Synthesis and packaging of neurotransmitters
 - Trigger
 - Depolarization of the synaptic terminal
 - Influx of Ca^{2+}
 - Vesicles fuse with the presynaptic membrane
 - Release of neurotransmitter
 - Postsynaptic binding of neurotransmitter onto the receptor
 - Opening or closing of channels
 - Postsynaptic current
 - Removal of neurotransmitter
- “Cleaning up the Synapse”
 - Reuptake
 - Enzymatic deactivation
 - Diffusion
- Postsynaptic Potentials
 - EPSP vs IPSP
 - Neural Integration
 - Spatial summation
 - Temporal summation

Steps of Synaptic Transmission



1	
2	
3	
4	
5	
6	
7	
8	
9	
10	



Lecture 6: Glia

Ethan Hughes, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Glia (Ch. 3) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/cells-of-the-nervous-system-glia/>

Learning Objectives

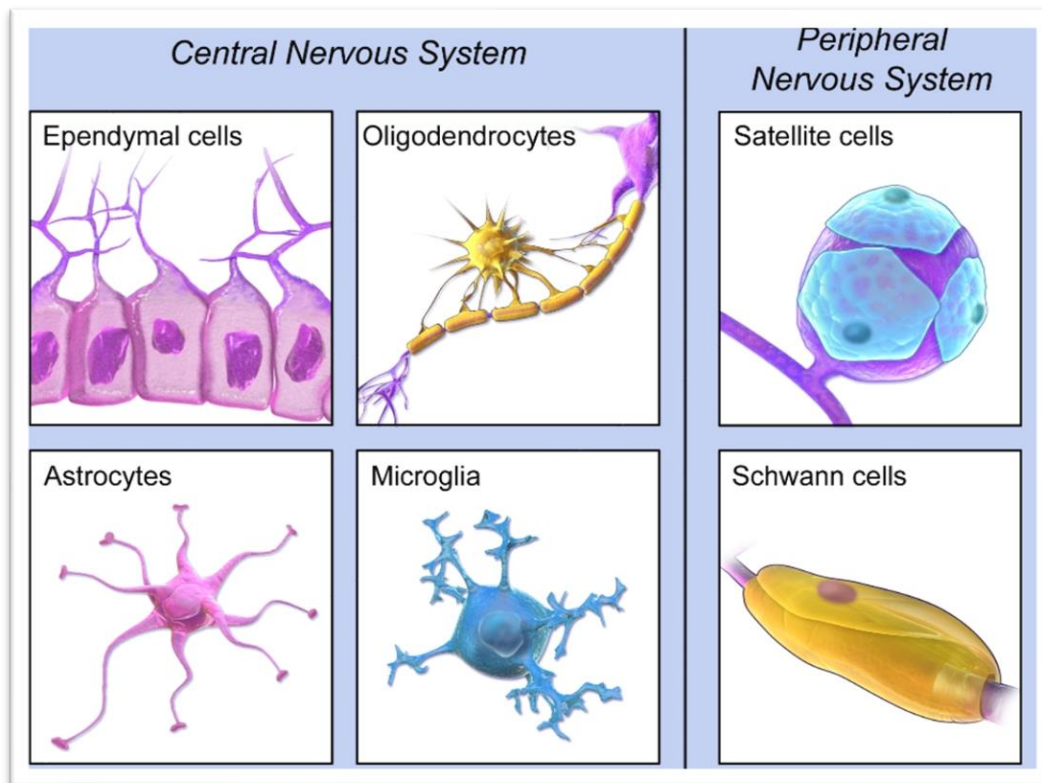
1. Name and describe the function of glial cells, including their common location in the nervous system.
2. Compare and contrast the function of oligodendrocytes and Schwann cells, particularly their role in the formation of myelin.
3. Describe the blood-brain barrier (BBB), how it is formed, and which substances can and cannot cross it.
4. Describe the immune function of microglia, astrocytes, and oligodendrocyte precursor cells in response to acute injury and chronic inflammation.
5. Describe the origins of tumors (glial vs neuronal).
6. Describe the autoimmune basis for multiple sclerosis and Guillain-Barré Syndrome.
7. Describe the role of glial cells in the “Gut-Brain axis”.

Terms to Know

- Glia of the CNS
 - Oligodendrocytes
 - Astrocytes
 - Microglia
 - Ependymal cells
- Glia of the PNS
 - Schwann cells
 - Satellite cells
- Blood-Brain Barrier
 - Components
 - Capillary endothelial cells (with tight junctions)
 - Astrocytic endfoot processes
 - Function: Prevents paracellular diffusion of hydrophilic substances from CNS capillaries (e.g., glucose passes through the insulin-independent transporter GLUT1)
- Neuroinflammatory response
 - Role of glial cells in response to injury
- Tumors (Note: A tumor derived from neurons is called medulloblastoma and is relatively rare. The majority of tumors derive not from neurons but rather from meninges (Meningioma) or glial cells (glioma)).

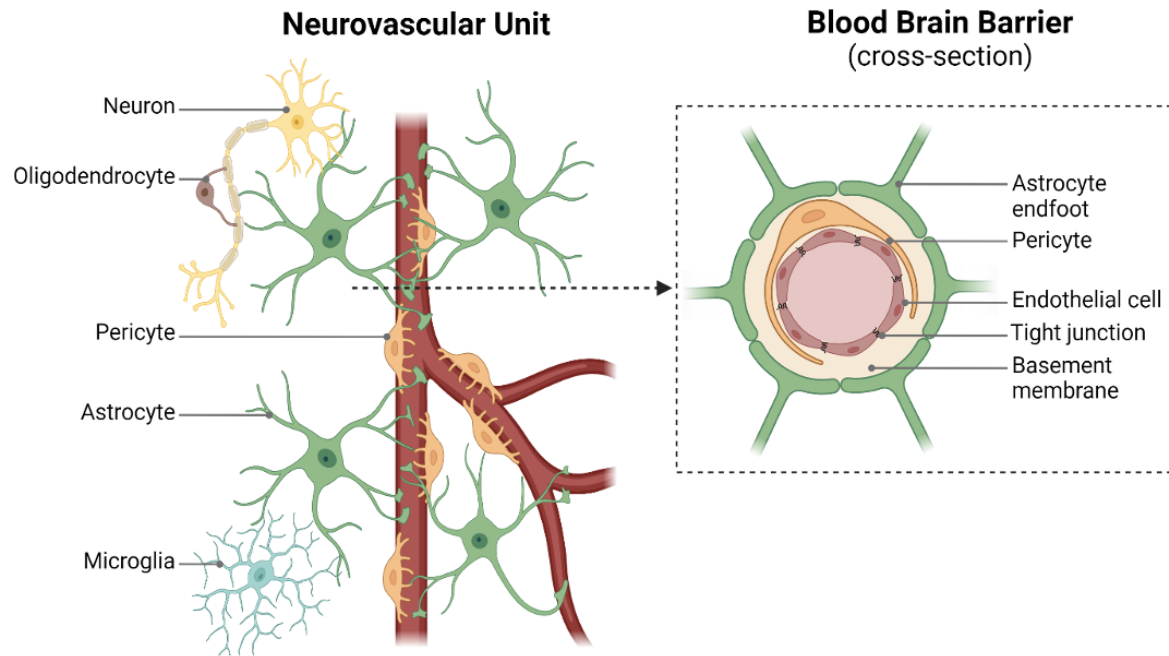
Glia Study Table

Glial Type	CNS or PNS?	Migration?	Function
Ependymal Cell	CNS	no	
Radial Glia	CNS	no	
Astrocytes	CNS	no	
Microglia	CNS	yes	
Oligodendrocyte	CNS	no	
Oligodendrocyte Precursor Cell	CNS	yes	
Schwann Cell	PNS	no	
Satellite Cell	PNS	no	
Enteric Glial Cells	PNS	no	



The Neurovascular Unit & The Blood Brain Barrier

Cellular actors of the brain blood interface



Lecture 7-8: Neurotransmitters & Receptors

John Caldwell, PhD – Department of Cell & Developmental Biology

Optional Foundational Reading

Many chapters on this topic for your reference (see chapters 13-20)

<https://openbooks.lib.msu.edu/introneuroscience1/chapter/neurotransmitter-action-ionotropic-receptors/>

Learning Objectives

1. Define ligand and receptor in comparison to a lock and key analogy.
2. Compare and contrast ionotropic and metabotropic receptors.
3. Define agonist and antagonist, and give examples of each for each neurotransmitter system.
4. Describe the four basic sites of drug action at a synapse.
5. List the main types of neurotransmitters involved in communication between neurons, including abbreviation, common functions, and receptor types.

Terms to Know

- Lock & Key Analogy
 - Lock = postsynaptic receptor
 - Ionotropic receptor
 - Metabotropic receptor
 - Key = ligand
 - Endogenous ligand
 - Exogenous ligand
 - Agonist vs antagonist
 - Competitive vs noncompetitive binding
- Four basic sites of drug action at a synapse
 - Effect on the production of neurotransmitter
 - Effect on storage and release of neurotransmitter
 - Effect on postsynaptic receptor
 - Effect on reuptake of neurotransmitter or enzymatic deactivation
- Major Neurotransmitters
 - Acetylcholine (ACh)
 - Receptor types
 - Nicotinic
 - Muscarinic
 - Drugs
 - Nicotine
 - Botulinum toxin
 - Black widow spider venom
 - Atropine
 - Dopamine (DA)
 - In family of monoamines
 - Receptor types
 - D1
 - D2
 - Drugs

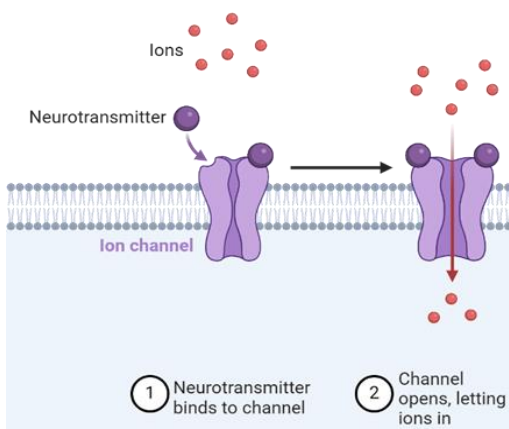


- L-Dopa
 - Cocaine
- Noradrenaline (a.k.a. Norepinephrine; NE)
 - In family of monoamines
 - Receptor types
 - Alpha
 - Beta
 - Drugs
 - MDMA
 - MAOI
- Serotonin (5-HT)
 - In family of monoamines
 - Receptor types: 5-HT1 through 5-HT7
 - Drugs
 - Fluoxetine (Prozac)
 - LSD
- Glutamate
 - Receptor types
 - NMDA
 - Kainate
 - AMPA
 - mGluR
- Gamma-aminobutyric Acid (GABA)
 - Receptor types
 - GABA_a
 - GABA_b
 - Drugs
 - Alcohol
- Glycine
 - Drugs
 - Strychnine
- Opioids
- Nitric Oxide

Two Distinct Types of Neurotransmitter Receptors

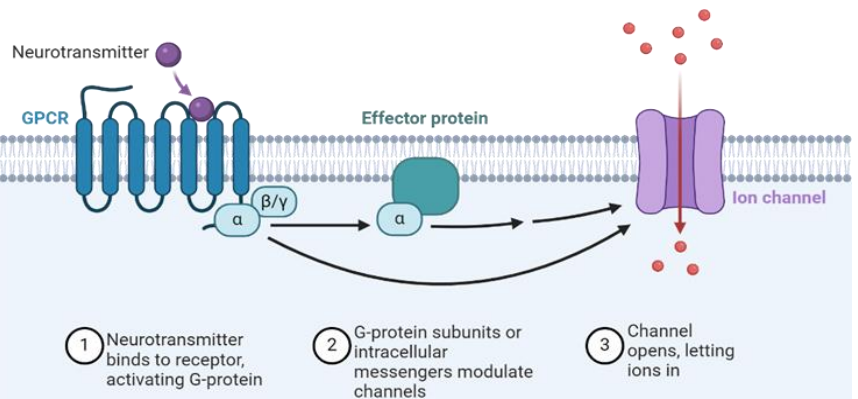
Ionotropic

Ligand-gated ion channels



Metabotropic

G-protein-coupled receptors



	AGONIST	ANTAGONIST
Effect on production of neurotransmitter		
Effect on storage and release of neurotransmitter		
Effect on receptors		
Effect on reuptake or deactivation of neurotransmitter		

NAME	Abbreviation	Receptor Types (with examples of functions)	Common Drugs (and sites of action)
Acetylcholine			
Dopamine			
Norepinephrine			
Serotonin			
Glutamate			
GABA			
Glycine			
Opioids			
Nitric Oxide			

LAB 1: Learning Lab

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Brad Pawlikowski, PhD – Department of Pediatrics

Optional Foundational Reading

Memory Systems (Ch. 66) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/memory-systems/>

LTP & LTD (Ch. 68) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/molecular-mechanisms-of-memory-hippocampus/>

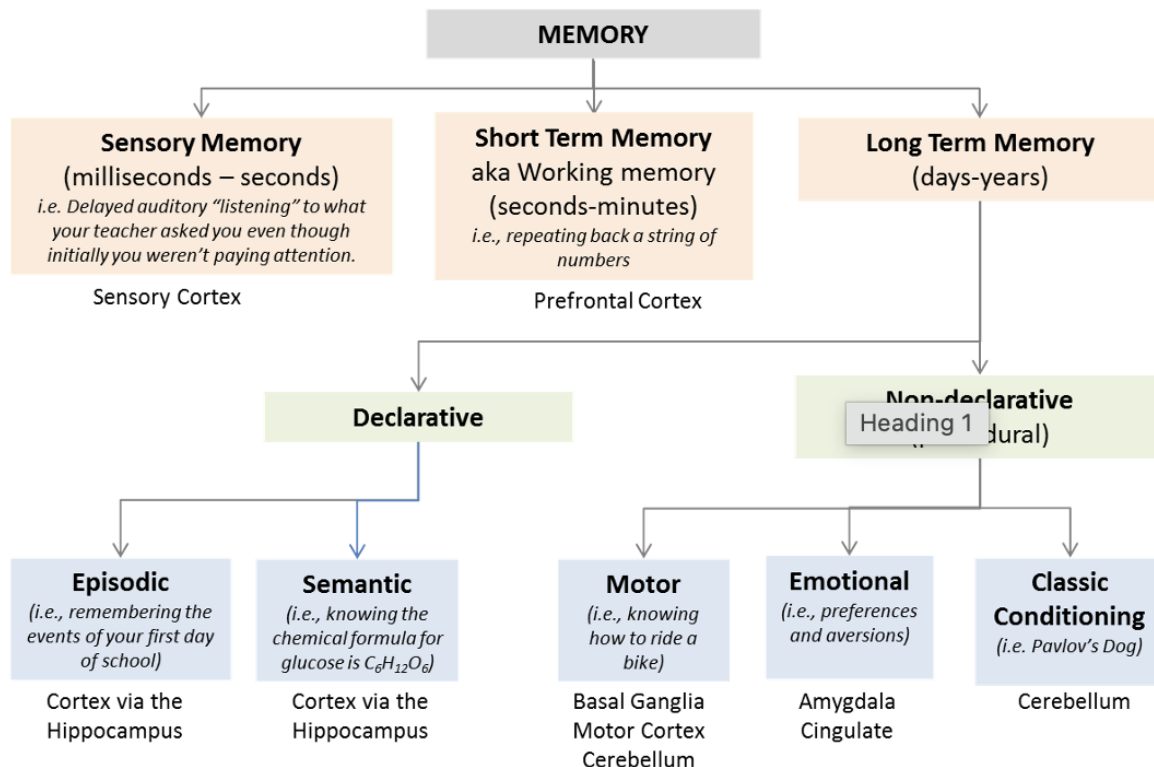
Texas Tech Health Sciences Center Success Types in Medical Education <https://www.ttuhscc.edu/medicine/medical-education/success-types/>

Learning Objectives

1. Differentiate declarative vs. non-declarative memory.
2. List the temporal categories of memory and the associated time frames for each.
3. Critically evaluate neuroeducation myths, and discriminate myth from fact.
4. Describe strategies that enhance LTP such as chunking, spaced repetition, and active recall.
5. Discuss the role of attention, sleep, and exercise in learning.

Terms to Know

- Memory categories
 - Qualitative categories
 - Declarative
 - Non-declarative (procedural)
 - Temporal categories
 - Sensory (immediate)
 - Short-term (working)
 - Long-term



How well do you know myth versus fact regarding learning and the brain? Indicate if each statement about the brain and learning is TRUE or FALSE.

Statement	T or F?
1. Although the entire brain is active over the course of the day, you only use 10% of your brain at a given time.	
2. The brain is fully developed by the age of 18, which informed the establishment of the legal age of "adulthood" in the United States.	
3. Individuals with higher IQ scores tend to have measurably larger brains and a higher total neuron count, making brain volume a reliable predictor of intelligence.	
4. Blue light is harmful to the brain, so blue-light-filtering glasses should be worn throughout the day to enhance brain health, sleep, and mood.	
5. There are critical periods of brain development in childhood after which certain things are more difficult to learn, such as language and visual processing.	
6. Research on hemispheric specialization shows that students are either left-brain dominant (analytical, logical) or right-brain dominant (creative, intuitive), and teaching is most effective when matched to the dominant hemisphere of the student.	
7. The brain cannot feel pain because it does not have pain receptors.	
8. Daily brain-training apps like Lumosity, Wordle, or crossword puzzles improve overall cognitive function, and these gains transfer to everyday tasks like memory and driving.	
9. Vigorous exercise can improve overall cognitive function.	
10. Circadian rhythms ("body-clock") shift during adolescence, causing students to be tired during the first lessons of the school day.	
11. Repeated rehearsal of mental processes physically changes the connections and shape of neurons in the brain, a capacity that persists into old age.	
12. The brain runs on sugar: glucose is the primary fuel source for neurons, and the brain consumes roughly 20% of the body's glucose and oxygen.	
13. The brain is selectively wired for multi-tasking by parallel circuits in the pre-frontal cortex that can focus on two or more attention-rich stimuli at the same time without a loss in performance.	
14. Students learn better and perform better in school when they receive information in their preferred learning style (e.g., auditory, visual, kinesthetic).	
15. Staying awake and studying all night before an exam improves next-day recall because the material stays in working memory without the decay that occurs during sleep.	

Statements adapted from: *The Dana Foundation Truth-or-Myth Flashcards* and Dekker S, Lee NC, Howard-Jones P and Jolles J (2012) *Neuromyths in education: Prevalence and predictors of misconceptions among teachers*. *Front. Psychology* 3:429. doi: 10.3389/fpsyg.2012.00429

UNIT II:

ANATOMY & IMAGING OF THE BRAIN

Lecture 9: Neurofluids – Blood & Stroke

Brad Pawlikowski, PhD – Department of Pediatrics

Optional Foundational Reading

Additional Structures of the Nervous System (Ch. 24) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/support-structures-of-the-nervous-system/>

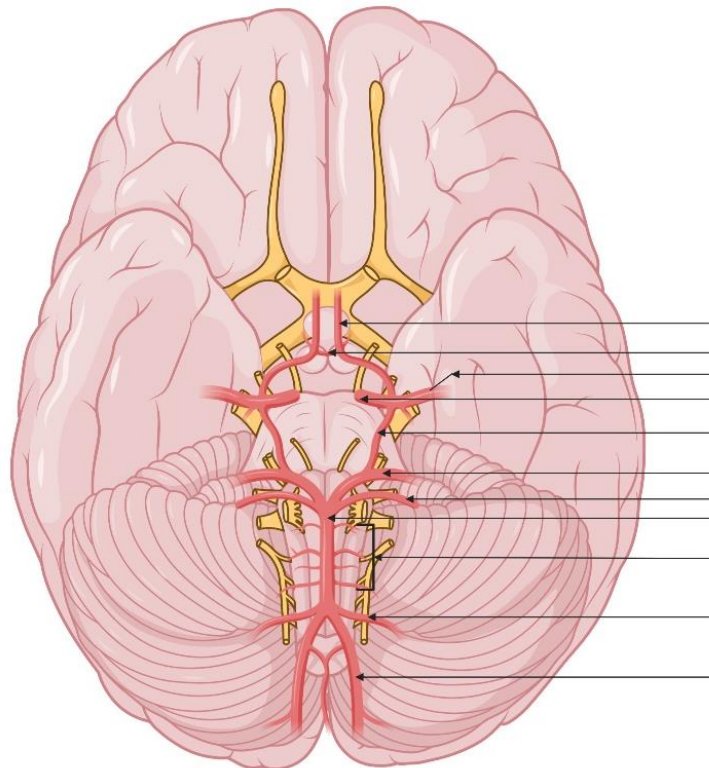
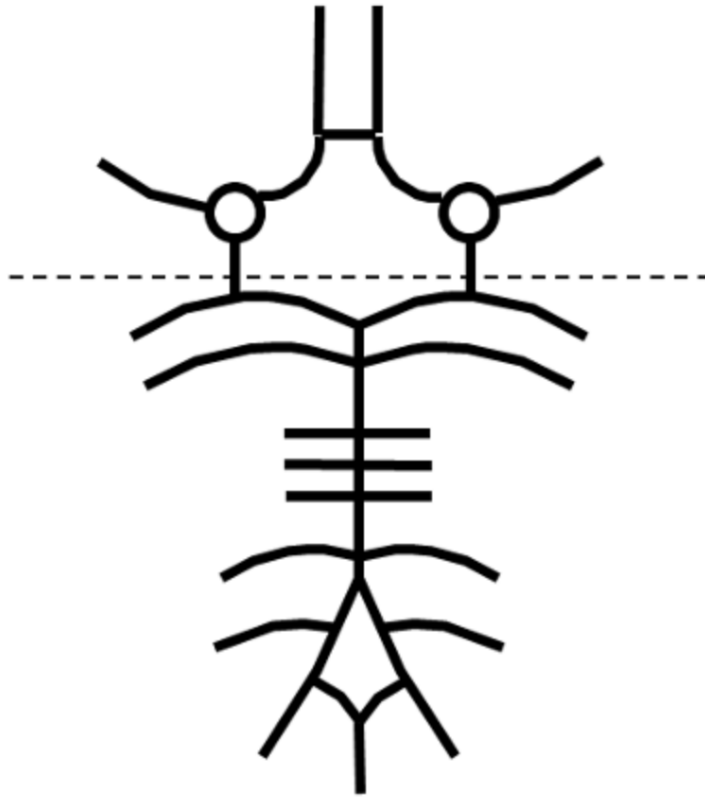
Learning Objectives

1. Draw the Cerebral Arterial Circle (aka “Circle of Willis”) and label all of its parts.
2. Differentiate meningeal arteries from cerebral arteries in terms of their anatomical location.
3. Identify the territories of the brain supplied by cerebral arteries; define watershed zones.
4. Compare and contrast hemorrhagic versus obstructive (ischemic) stroke.
5. Review cerebral topography and use the homunculus to determine the location of a stroke.
6. Compare and contrast Broca’s and Wernicke’s aphasia.
7. Define visuospatial neglect and describe how it is most often caused.

Terms to Know

- Blood vessels
 - Two origin arteries (paired)
 - Vertebral artery (source of the posterior circulation)
 - Internal carotid artery (source of the anterior circulation)
 - Three cerebral arteries (paired)
 - Anterior cerebral artery
 - Middle cerebral artery
 - Posterior cerebral artery
 - Three cerebellar arteries (paired)
 - Superior cerebellar artery
 - Anterior inferior cerebellar artery (AICA)
 - Posterior inferior cerebellar artery (PICA)
 - Two communicating arteries
 - Anterior communicating artery (unpaired)
 - Posterior communicating artery (paired)
- Stroke
 - Cerebrovascular accident (CVA)
 - Hemorrhagic stroke
 - Obstructive stroke
 - Thrombus
 - Embolus
 - Cerebral topography
 - Blood supply of primary motor homunculus
 - Blood supply of primary somatosensory homunculus
 - Blood supply of primary visual cortex
 - Aphasia (Wernicke’s vs Broca’s)
 - Visuospatial neglect

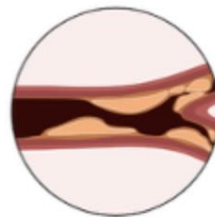
Circle of Willis (Be able to draw and label this diagram)



Brain Arterial Supply

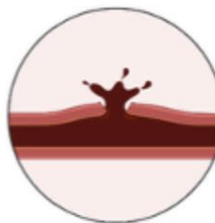


Common Types of Stroke



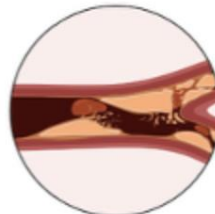
Atherosclerosis stroke

Atherosclerotic plaque build up and artery blockage



Hemorrhagic stroke

Rupture of the vessel and hemorrhage



Ischemic stroke

Atherosclerotic plaque rupture and thrombus formation

Lecture 10: Neurofluids – Meninges, Ventricles & CSF

Brad Pawlikowski, PhD – Department of Pediatrics

Optional Foundational Reading

Additional Structures of the Nervous System (Chapter 24) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/support-structures-of-the-nervous-system/>

Learning Objectives

1. Describe the function of the meninges and list their layers and potential spaces from superficial to deep.
2. Label the components of the ventricular system on a hemisected brain, 3D images and 2D cross-sections.
3. Trace a drop of CSF from the lateral ventricles to the venous sinuses.
4. Describe the functions, components and formation of CSF.
5. Describe the function of the choroid plexus (compare & contrast its epithelium to that of the blood-brain barrier).
6. Describe “the Glymphatic system”, including the role of CSF circulation in this system.
7. Define hydrocephalus, meningitis, and encephalitis.

Terms to Know

- Meninges
 - Dura
 - Dural venous sinuses (you’ll learn the individual names of the sinuses in Gross Anatomy; also note this term is synonymous with “cerebral sinuses” or simply “dural sinuses” or “venous sinuses”)
 - Bridging veins – drain cerebral veins into dural venous sinuses
 - Arachnoid
 - Subarachnoid space
 - Arachnoid granulations
 - Pia
 - Meningitis
- Ventricles
 - Lateral Ventricles – left and right
 - Interventricular foramina of Monro
 - Third Ventricle
 - Cerebral Aqueduct
 - Fourth Ventricle
 - Foramina of Luschka (2 lateral)
 - Foramen of Magendie (1 medial)
- Cerebrospinal Fluid (CSF)
 - Choroid plexus
 - “Glymphatic system”
 - Encephalitis
 - Hydrocephalus

Lecture 11: The Central Nervous System

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Brain Anatomy (Ch. 23) (external & internal structures) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/external-brain-anatomy/>

Learning Objectives

1. Recall the five developmental divisions of the CNS & identify each division in the adult brain.
2. Identify the distinguishing features and major functions of the telencephalon on diagrams and gross specimens.
3. Recall the concept of cerebral topography and explain the somatosensory and motor homunculi.
4. Recall the terms for grey matter (cortex, nucleus, ganglion) and white matter (tract, nerve) and determine if the named structure is part of the CNS or the PNS.
5. Differentiate the neuroanatomical term “pathway” vs. “tract.”
6. Describe the major functions of the cingulate gyrus, insula, amygdala, and hippocampus.
7. List the four divisions (thalami) of the diencephalon. Identify each on images of gross brain or a hemisected brain and describe their general functions.
8. List the name and general function of the 5 relay nuclei of the thalamus.
9. Describe the role of the pineal gland (within the epithalamus).
10. Describe the location of the subthalamus and its general role.
11. Describe the functions of the hypothalamus.

Terms to Know

The Telencephalon

- Cerebrum
 - Grey matter (cerebral cortex or cerebral nuclei) vs. white matter
- Sulci/gyri
 - Lateral sulcus (Sylvian fissure)
 - Central sulcus
 - Pre-central sulcus vs Post-central sulcus
 - Pre-central gyrus vs. Post-central gyrus
- Lobes
 - Frontal
 - Temporal
 - Parietal
 - Occipital
 - “Hidden lobes”: Insula and Cingulate
- Commissures
 - Corpus Callosum
 - Anterior Commissure
 - Posterior Commissure
- Specialized Cortical Centers
 - Primary somatosensory cortex
 - Primary motor cortex

- Primary visual cortex
- Primary auditory cortex
- Pre-frontal cortex
- Speech centers – Wernicke’s area and Broca’s area
- Limbic System
 - Cingulate gyrus
 - Amygdala
 - Hippocampus
 - Parahippocampal gyrus
- Basal ganglia
 - Caudate
 - Putamen
 - Globus pallidus
 - Subthalamic nucleus
 - Substantia nigra

The Diencephalon

- Thalamus
 - Relay nuclei (More about these pathways in Unit 3)
 - Anterior (limbic)
 - VA/VL (motor)
 - VPL/VPM (somatosensory)
 - LGN (visual)
 - MGN (auditory)
- Hypothalamus (more about this in the next lecture)
 - Feeding (appetite, food intake, thirst)
 - Fight or flight (autonomic control)
 - Mating (sexual drive)
 - Control of body temperature, sweating, water balance & circadian clock
- Epithalamus (pineal gland and habenula)
 - Pineal gland – releases melatonin in darkness
 - Habenula – influences cognitive and motor behavior based on the motivational values associated with the behaviors.
- Subthalamus
 - Important role in basal ganglia circuitry (more about this in Unit 5)

Type	Nucleus	General Function	Afferent	Efferent
Relay nuclei	Anterior Nucleus	Limbic	Mammillary body (Papez circuit)	Cingulate Gyrus (Papez Circuit)
	VA/VL	Motor	Cerebellum Red nucleus GPi	Motor & Supplementary Motor cortex Prefrontal cortex
	VPL/VPM	Somato-sensory	DCML pathway Spinothalamic pathway Trigeminothalamic pathway	Somatosensory cortex
	LGN	Visual	Optic tract	Primary visual cortex
	MGN	Auditory	Inferior brachium (auditory pathway)	Primary auditory cortex
Association	Dorsomedial	Executive function	Amygdala Temporal lobe (olfactory cortex)	Prefrontal cortex Limbic system
	Pulvinar	Visual and sensory association	Lots of sensory cortices	Lots of sensory cortices
Other	Centromedian	Motor	Lots of motor systems (particularly vestibulomotor systems)	Lots of motor systems (particularly basal ganglia)

Note that LGN and MGN are sometimes called LGB and MGB. N = Nuclei, B = Body, these names are equivalent. Also, "dorsomedial" can also be called "medial dorsal" nucleus.

Lecture 12: The Autonomic Nervous System

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Peripheral Nervous System (Ch. 27) (includes sympathetic & parasympathetic divisions)

<https://openbooks.lib.msu.edu/introneuroscience1/chapter/autonomic-nervous-system/>

Learning Objectives

1. Describe the function of the autonomic nervous system (ANS).
2. Compare and contrast the functions of the sympathetic and parasympathetic nervous systems.
3. Expand the SLUDGE acronym and explain how it describes the signs of parasympathetic (cholinergic) overactivation.
4. Describe the role of the hypothalamus in regulating the autonomic nervous system and its neuroendocrine control of the pituitary gland.
5. Describe the Hypothalamic-Pituitary-Adrenal (HPA) axis and its role in regulating cortisol release in response to stress.
6. Identify and discuss the functions of the autonomic control centers in the brainstem.
7. Describe the role of the reticular formation in arousal, autonomic control, and modulation of respiration and cardiovascular function.
8. Explain how the prefrontal cortex ("wizard brain") exerts top-down modulation over the hypothalamus and autonomic output ("lizard brain"), including the role of the uncinate fasciculus in connecting the prefrontal cortex to the amygdala and anterior temporal lobe.

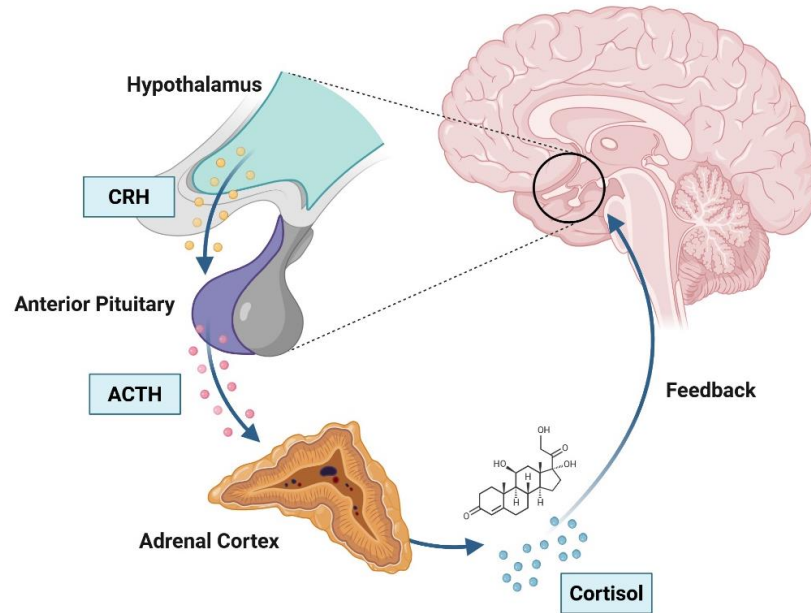
Terms to Know

- Autonomic Nervous System
 - Sympathetic vs. parasympathetic
 - Overactivation of parasympathetic = SLUDGE
 - Overactivation of sympathetic = fight-or-flight excess (mydriasis, tachycardia, hypertension, sweating)
- Hypothalamic control center of the ANS
 - HPA axis
 - Hypothalamus
 - Pituitary
 - Adrenal
- Brainstem Autonomic Centers
 - Reticular Formation
 - Respiratory Centers
 - Cardiovascular Centers
- Top-down modulation of the ANS
 - Prefrontal cortex → Uncinate fasciculus → Amygdala → Hypothalamus → ANS output

Hypothalamic Nuclei

(You do not need to memorize the names of all these different nuclei unless we specifically discuss them in class)
<https://epomedicine.com/medical-students/nuclei-hypothalamus-mnemonic/>

HPA Axis



Lecture 13-14: Neuroimaging 101

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

- View the MRI Atlas on Canvas
- Imaging the Living Brain (Ch. 25) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/imaging-the-living-brain/>
- Mori S, Oishi K, Faria AV. White matter atlases based on diffusion tensor imaging. Curr Opin Neurol. 2009. <https://pubmed.ncbi.nlm.nih.gov/19571751/>
- fMRI <https://www.open.edu/openlearn/body-mind/health/health-sciences/how-fmri-works>
- Elliott ML, Knodt AR, Ireland D, Morris ML, Poulton R, Ramrakha S, Sison ML, Moffitt TE, Caspi A, Hariri AR. What Is the Test-Retest Reliability of Common Task-Functional MRI Measures? New Empirical Evidence and a Meta-Analysis. Psychol Sci. 2020 Jul;31(7):792-806. <https://journals.sagepub.com/doi/10.1177/0956797620916786>

Learning Objectives

1. List the various types of brain imaging techniques.
2. Describe the spatial orientation (L vs. R) when viewing an MRI or CT.
3. List the advantages and disadvantages of MRI and CT as well as their clinical indications.
4. Compare and contrast T1 vs T2 vs FLAIR sequences in terms of their contrast.
5. Demonstrate brain anatomy (lobes & ventricles) on CT and MRI.
6. Compare and contrast an epidural, subdural and subarachnoid hemorrhage on a CT scan.
7. Describe how brain activity is assessed in functional magnetic resonance imaging (fMRI) and discuss recent evidence that questions the reliability of fMRI.
8. Describe the properties of neural tissue that make diffusion tensor imaging (DTI) possible.
9. Explain the basic principle of how a DTI image is generated from a DWI image.
10. Review the types of white matter tracts (commissural, projection, and association) & give one example of each type.

Terms to Know

- Computed Tomography (CT)
 - Method: uses ionizing radiation
 - View: axial (as though patient is lying on their back; you are at the foot of the bed and looking up at their brain from their feet).
 - Interpretation: hypodense = dark structures, hyperdense = white structures. The denser the object, the brighter (whiter) it will be. Thus, bone and blood are white, grey matter is light grey, white matter is a darker grey, CSF is black.
 - ALARA (As Low As Reasonably Achievable): principle applied to clinical use of radiation
 - Good for: Calcifications and trauma (viewing bone and blood)
 - Epidural hemorrhage: above (superficial to) the dura; the blood makes a lens shape or “biconvex” shape.
 - Subdural hemorrhage: under (deep to) the dura; the blood is crescent-shaped (and can cross sutures).
 - Subarachnoid hemorrhage: within the subarachnoid space; blood fills sulci.
 - Intraventricular hemorrhage: bleeding into the ventricular system
 - Advantages: fast, readily available, relatively low cost
 - Disadvantages: radiation, lower resolution

- Magnetic Resonance Imaging (MRI)
 - Method: uses a magnetic field and radio frequencies to produce cross-sectional images based on the resonance of the body's hydrogen atoms.
 - Views: coronal, axial, sagittal
 - Interpretation:
 - T1-weighted: white matter is white, grey matter is grey, water is black
 - T1-hypersignal (blood is bright)
 - T2-weighted: white matter is dark grey, grey matter is light grey, and water is white.
 - FLAIR: looks just like a T2, except the fluid is nulled and therefore is black.
 - Good for: viewing grey vs white matter, neuroanatomy, tumors, soft tissue
 - Advantages: high resolution, multiplanar, safe (no radiation), ability to get functional information (with fMRI)
 - Disadvantages: expensive, not as readily available, sensitive to patient movement, cannot have any metal near the machine since it's a huge magnet
 - Other advanced MRI sequences:
 - Magnetic resonance angiography (MRA) = "time of flight" sequence can provide anatomy of vasculature without needing to use contrast agents
 - Functional Magnetic Resonance Imaging (fMRI) = depends on the BOLD signal (blood-oxygen-level dependent) as areas of the brain with high activity require increased demand for oxygen and glucose.
 - Diffusion Tensor Imaging (DTI) derived from Diffusion Weighted Imaging (DWI). Tracts appear in three different planes
 - **Commissural Tracts (red)** = left/right; example: corpus callosum
 - **Projection Tracts (blue)** = superior/inferior; example: corticospinal tract
 - **Association Tract (green)** = from one lobe to another (often anterior/posterior); example: cingulum and uncinate fasciculus

MOD-ALITY	INDICATION	ADVANTAGES	DISADVANTAGES
CT	Trauma-OR Calcium/bone blood	Availability Fast Lower cost	Radiation Lower resolution for parenchyma
MRI	Vascular, Inf/inflam, Trauma, Congenital, Demyelination, Neoplasias	Safe High-resolution	Sedation/anesthesia High cost Availability
MRA	Detailed vasculature	Safe	Occasionally a contrast agent (gadolinium) required
fMRI	Functional activity of brain regions during a task	Safe Can tell you about function!	Two scans required (control and during task). Sensitive to movement. May not be successful (blood flow should correlate to activity). Difficult to analyze/ interpret.
DTI	Anatomy of white matter tracts	Safe	Not routinely obtained May be difficult to analyze/interpret

Lecture 15: Cross-sectional Neuroanatomy

Review

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

View the MRI and myelin-stained atlases on Canvas

Brain Anatomy (Ch. 23) (review the mid-sagittal & coronal section views)

<https://openbooks.lib.msu.edu/introneuroscience1/chapter/external-brain-anatomy/>

Learning Objectives

1. Identify the major internal brain structures on T1-weighted MRI, myelin-stained cross-sections and unstained brain sections.
2. Compare spatial relationships of major internal brain structures in 3D versus 2D views.
3. Describe why the brain structures look the way they do (shape, size, position) in 2D slices.

Terms to Know

(Be prepared to identify these structures on MRI as well as on myelin-stained and unstained brain sections)

- Ventricular system
 - Lateral Ventricle(s)
 - Interventricular foramina (of Monro)
 - Third Ventricle
 - Cerebral Aqueduct
 - Fourth ventricle
 - Septum pellucidum
- Grey Matter Nuclei
 - Thalamus
 - Hypothalamus (including Mammillary Bodies)
 - Basal Ganglia
 - Caudate
 - Putamen
 - Globus Pallidus
 - Nucleus accumbens
 - Hippocampus
 - Amygdala
- White Matter Structures
 - Corpus Callosum
 - Anterior Commissure
 - Fornix
 - Internal Capsule
- Other Structures:
 - Midbrain
 - Pons
 - Cerebellum
 - Medulla
 - Cingulate gyrus
 - Insula

LAB 2: Whole Brain Identification & Slicing

MHA Teaching Team

Learning Objectives

1. Identify gross brain structures on whole brains, hemisected brains, and brain slices.
2. Slice the brain in coronal, horizontal and sagittal sections and identify internal brain structures.

Learning Tools & Instructions

Each lab group will have one bucket. Each bucket contains one whole brain and one hemisected brain. Organize into groups of 4 students around each bucket. Please use gloves and keep the brain tissue in the bucket. Complete Steps 1-7. A copy of these instructions will be available in the lab at each station.

(USE THE WHOLE BRAIN TO COMPLETE STEPS 1- 4)

1. **ANATOMICAL DIRECTIONS:** First, orient the brain and practice naming anatomical directions (superior vs. inferior, anterior vs. posterior, ventral vs. dorsal, rostral vs. caudal). Identify left vs. right hemisphere. Which cerebral hemisphere “talks to” the left side of the body? Which “talks to” the right side of the body?
2. **MENINGES:** Next, appreciate the meninges on the whole brain. In many cases, the **dura mater** has already been removed and is floating in the bucket. Tug on and try to rip the dura mater. Can you appreciate why it was named, “tough mother”? Now identify the **arachnoid mater**. Can you find any **arachnoid granulations**? Take scissors and cut open the transverse sinus on the dura mater. Often you will find arachnoid granulations via this method. The **pia mater** is the thinnest and most intimate meningeal layer. It is adhered like “shrink wrap” to the brain and gives the brain surface a shiny/glistening appearance. You can’t really identify the pia apart from the cortex. If you tried to peel it you would destroy the brain tissue. In summary, before you go on, be sure you have identified the following:
 - Dura mater
 - Arachnoid mater
 - Subarachnoid space: what is in this space? _____, _____ & _____
 - Arachnoid granulations (may not be visible on all brains)
 - Pia mater
3. **BLOOD SUPPLY:** Next, point out the blood vessels traveling in the **subarachnoid space**. Before you remove the arachnoid mater, identify the 3 major cerebral arteries: **Anterior Cerebral Artery**, **Middle Cerebral Artery** and **Posterior Cerebral Artery**. Review the territories of the cerebrum that are supplied by each cerebral artery. Next, turn the brain and view the ventral surface. Can you identify the **Circle of Willis**? Some of these vessels may have been destroyed when the brain was removed from the skull. Review the vessels that make up the Circle of Willis and identify as many of the following as possible on your brain:
 - Two origin arteries (paired)
 - Vertebral artery (source of the posterior circulation)
 - Internal Carotid artery (source of the anterior circulation)
 - Three cerebral arteries (paired)
 - Anterior Cerebral artery
 - Middle Cerebral artery
 - Posterior Cerebral artery
 - Three cerebellar arteries (paired)
 - Superior Cerebellar artery
 - Anterior Inferior Cerebellar artery (AICA)

- Posterior Inferior Cerebellar artery (PICA)
- Two communicating arteries
 - Anterior Communicating artery (unpaired)
 - Posterior Communicating artery (paired)
- Basilar artery (unpaired)
- Pontine arteries (paired)

4. **WHOLE BRAIN ID:** Now gently dissect away the meninges to clean the surface of the cerebrum. You will invariably also remove blood vessels. If your brain has a well-preserved Circle of Willis, try to keep that intact as you dissect. First identify the **medial longitudinal fissure**, the **lateral sulcus (Sylvian fissure)** and the **central sulcus**. These sulci serve as landmarks for delineating the lobes of the brain. Identify the **frontal**, **parietal**, **temporal** and **occipital** lobes. Stick your hand or a probe in the Sylvian fissure and poke the **insula**. From the central sulcus, identify the **pre-central gyrus** and **post-central gyrus**. What primary cortices are housed in each of these gyri? Also, review the homunculus. What parts of the body are “mapped” onto what parts of cortex? What parts of the body are given the most surface area of cerebral cortex?

Identify the following structures on the whole brain:

- Lateral sulcus (aka Sylvian Fissure)
- Medial longitudinal fissure
- Central sulcus
- Frontal lobe
- Parietal lobe
- Temporal lobe
- Occipital lobe
- Insula
- Pre-frontal cortex
- Pre-central gyrus – Primary motor cortex (Brodmann Area___?)
- Post-central gyrus – Primary somatosensory cortex (Brodmann Area___?)
- Pre-central sulcus
- Post-central sulcus
- Calcarine sulcus
- Primary auditory cortex (Brodmann Area___?)
- Fusiform gyrus
- Parahippocampal gyrus
- Broca’s area
- Wernicke’s area
- Uncus

(USE THE HEMISECTED BRAIN TO COMPLETE STEPS 5- 6)

5. **HALF BRAIN ID:** In your brain bucket, find a mid-sagittally cut half brain. Identify the following on the hemisected brain:
- Cingulate gyrus
 - Cingulate sulcus
 - Corpus Callosum (rostrum, genu, body and splenium)
 - Septum pellucidum
 - Thalamus
 - Interthalamic adhesion (aka Massa Intermedia)
 - Hypothalamus
 - Optic chiasm
 - Mammillary bodies

- Fornix
- Pineal gland
- Pituitary (this has most likely been ripped off...point to where it would normally be located)
- Primary Visual Cortex (Brodmann area 17)
- Parieto-occipital sulcus
- 3 divisions of brainstem (midbrain, pons, medulla)
- Cerebellum

6. Next, pick up the cast ventricular model and review the components of the ventricular system on this physical model. What is contained in the ventricles and what is its function? Next, review the components of the ventricular system that you can see on the half brain. Now comes the interesting part! Insert the cast ventricular model into the hemisected brain. Now trace a drop of CSF from its formation in the lateral ventricles to the superior sagittal sinus (note that you will not be tested on all the names of the dural sinuses in this course; however, you will be tested on the names of the sinuses in your gross anatomy course).

Identify the following parts of the ventricular system on the model and the hemisected brain:

- Lateral ventricles
- Interventricular foramina of Monro
- Third ventricle
- Cerebral Aqueduct
- Fourth Ventricle

(FIND THE BRAIN DESIGNATED FOR SLICING TO COMPLETE STEP 7. DO NOT SLICE THE BEAUTIFUL BRAIN.)

7. **INSTRUCTIONS FOR BRAIN SLICING:** Each group will slice one brain. Do not start cutting until you have received instructions from the lab instructor. Please be careful and watch your fingers! You may choose to slice the brain coronally or horizontally. Discuss what kind of cut you will make with the table adjacent to yours so that if your group does coronal slices, the other group does horizontal. Then, swap tables so that you have the opportunity to identify the structures listed below on both coronal and horizontal brain slices. You will view these cross-sections again on MR images in a subsequent lab.

- Lateral Ventricle(s)
- Third Ventricle
- Interventricular Foramina of Monro
- Septum pellucidum
- Thalamus
- Hypothalamus
- Mammillary bodies
- Basal Ganglia
 - Caudate
 - Putamen
 - Globus Pallidus
- Corpus Callosum
- Hippocampus
- Amygdala
- Fornix
- Internal Capsule
- Cingulate gyrus
- Insula
- Midbrain
- Pons
- Cerebellum
- Medulla

LAB 3: Interactive 3D Brain Lab

Maureen Stabio, PhD – Department of Cell & Developmental Biology

Learning Objectives

1. Compare spatial relationships of major internal brain structures on 2D vs. 3D views.
2. Identify the ventricles of the brain on a physical 3D model.
3. Demonstrate the flow of CSF through the ventricles on a physical 3D model.
4. Identify on MR images, CT scans, and sections through the brain: lateral ventricle, interventricular foramen, third ventricle, cerebral aqueduct, and fourth ventricle.
5. Identify the major structures of the brain on coronal, axial, and sagittal preparations: thalamus, hypothalamus, components of the basal ganglia, hippocampus, and amygdala.
6. Identify the major white matter structures of the brain on coronal, axial and sagittal preparations: corpus callosum, fornix, internal capsule (in particular, note the internal capsule in relationship to the caudate nucleus, putamen/globus pallidus, and thalamus).

Learning Tools

This lab has two parts:

- **Part A: Physical Modeling of the Brain (1 hour)**
 - Work in pairs. Each pair will receive one cast ventricular model and one bag of colored “model magic”, which is a safe, non-toxic, hypoallergenic foam-based modeling compound. Double-sided tape will also be available.
 - Build the structures around the ventricular model using the color key and the instructions below. As you build the structures, keep them to size! If you build the structures too large, they will not fit properly onto the model.
- **Part B: Digital Modeling of the Brain (1 hour)**
 - Please ensure that you have the Visible Brain Atlas installed www.toltech.net/downloads/visible-brain-atlas. This resource is free.
 - For software support email support@toltech.net
 - Prior to lab, watch the instructional video that describes how to use this software <https://vimeo.com/1111771974/393a3760bf>
 - See instructions below in Part B for more details

MODELING COLOR KEY:

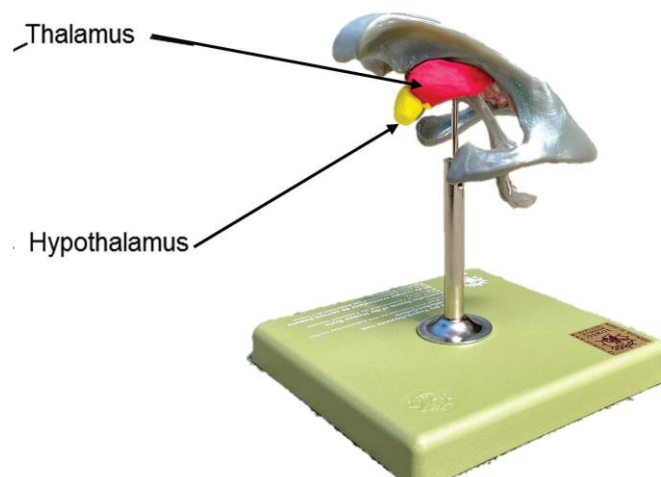
PINK -----Thalamus
YELLOW -----Hypothalamus
GREEN -----Caudate, Putamen
NEON GREEN ----Globus Pallidus
BLUE -----Hippocampus
WHITE -----Fornix, Internal capsule
RED -----Amygdala

Instructions for Part A – Physical Brain Modeling

STEP 1: First, review all parts of the ventricles using the picture below and your ventricle model.

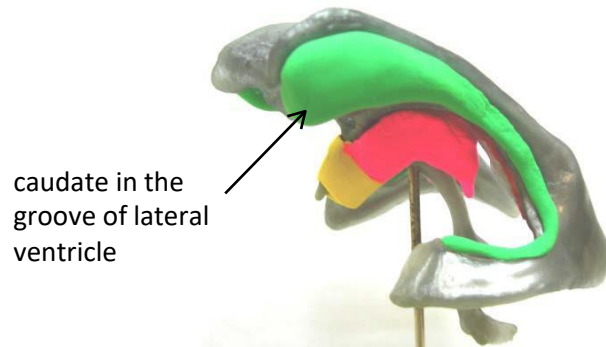
- Lateral Ventricle:
 - Anterior horn
 - Body
 - Atrium
 - Posterior horn
 - Inferior horn
 - Interventricular foramen
- Third Ventricle
- Cerebral Aqueduct
- Fourth Ventricle
- Foramen of Luschka (2)
- Foramen of Magendie (1)

STEP 2: Next, build a thalamus in **pink** and a hypothalamus in **yellow** and add these two structures onto your ventricle model. You may use double-sided tape to help stick the modeling compound to the ventricle model.
Building clues: The left and right thalami sandwich the 3rd ventricle like two hamburger buns. When you build the

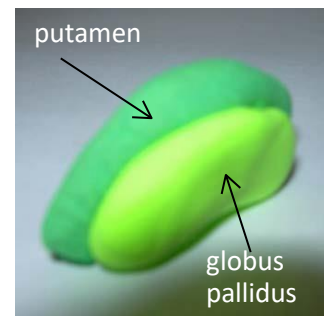
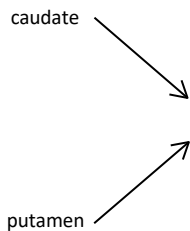


thalamus, be sure to include the LGN (the “tail” that hooks around laterally from the posterolateral aspect of the thalamus). The hole located in 3rd ventricle on your ventricle model represents the location of the interthalamic adhesion. Using this as a landmark, add the thalamus and hypothalamus. Do not build these structures too large or they will not fit properly in the model.

STEP 3: Next, build two caudate nuclei in **dark green**. Then attach the caudate nuclei to the lateral curvature of the lateral ventricles. *Building clues:* The caudate nucleus is a C-shaped structure that curves adjacent to the lateral ventricle. It consists of a head, body and tail. The caudate nucleus is part of the basal ganglia. Remember to build the caudate to the appropriate size so that it fits properly in the lateral groove of the lateral ventricles.



STEP 4: Build a putamen in **dark green** and a globus pallidus in **neon green**, but do not attach them to the model just yet. Set these two structures aside until STEP 5. *Building clues:* The putamen and globus pallidus are also part of the basal ganglia. Together these two nuclei are called the lenticular nucleus. The globus pallidus is located medial to the putamen.



Images adapted from *University of Washington Digital Anatomist Program* copyright © 1994 University of Washington

STEP 5: Now add the internal capsule in **white** to the lateral surface of the caudate and thalamus. Then, attach the putamen (with globus pallidus adhered medially) to the head of the caudate. The connection between the head of the caudate and the putamen is called the nucleus accumbens.

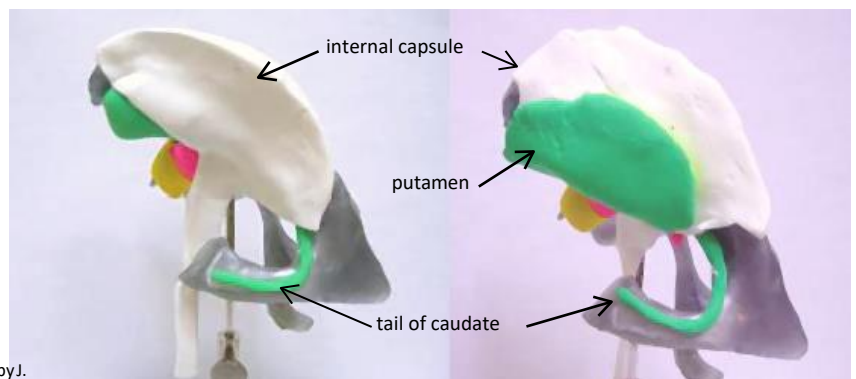
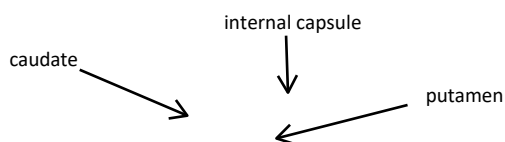


Image adapted from *The Human Brain in Photographs and Diagrams*, by J. Nolte and J. Angevine copyright © 2000, Mosby Inc.

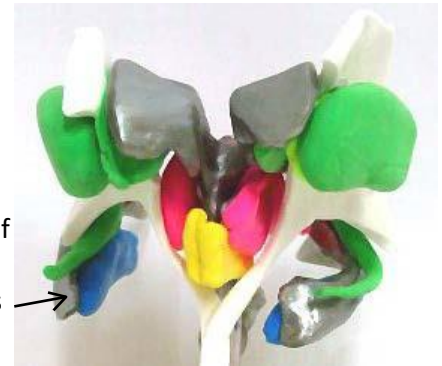
STEP 6: Build a hippocampus in **blue**. Add it to the medial edge of the inferior horn of the lateral ventricle.

Posterolateral View

Anterior View

← Hippocampus
in medial edge of
inferior horn of
lateral ventricles →

Images adapted from *University of Washington Digital Anatomist Program* copyright © 1994 University of Washington



STEP 7: Add the fornix in **white** and the amygdala in **red**. *Building clues:* The fibers of the fornix arise from the hippocampus as a fringe of axons called the fimbria. These fibers join at the midline to form the body of the fornix. Therefore, the body of the fornix forms the medial wall of the lateral ventricles. Then the fornix splits into two vertical columns that will descend through the hypothalamus and terminate in the mammillary bodies. The amygdala forms the anterior roof of the inferior horn of the lateral ventricle and is located anterior to the hippocampus.

Posterior

Anterior

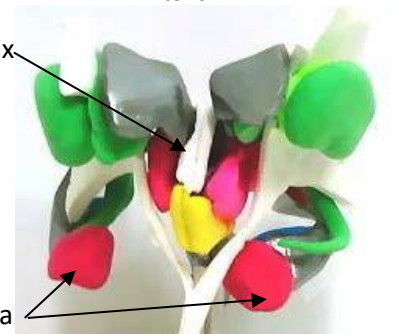


Image adapted from *University of Washington Digital Anatomist Program*

STEP 8: This modeling activity was designed to help you transition from 3-D structures to 2-D slices. As you view your finished product, study the spatial relationships of the various structures, and visualize how these structures will appear in 2-D coronal slices. Take your model into the computer lab and proceed to the second part of this lab (Part B).

Instructions for Part B – Digital Brain Modeling

STEP 1: Open the VH Dissector and navigate to Stabio's 3D Brain – 3 button mouse is recommended.

STEP 2: Navigate to University of Colorado School of Medicine Lessons. Click on “Welcome to Dr. Stabio's 3D Brain” for a brief introduction.

STEP 3: Click on “Interactive 3D Brain Lab.” Read through the learning module. Click on the hyperlinks to navigate to a view.

STEP 4: After you have completed the module, work in pairs or teams to complete the practice quiz on Canvas.

Need help? For tutorial on how to use this software refer to <https://vimeo.com/354399905/3e2125e1f0> and refer to your Week 1: Pework 3 assignment. For software support email support@toltech.net

Structures to identify:

- Ventricular System
 - Lateral Ventricles
 - Interventricular foramen (of Monro)
 - Third Ventricle
 - Cerebral aqueduct
 - Fourth ventricle
- Basal Ganglia
 - Caudate
 - Putamen
 - Globus Pallidus
- Midline structures
 - Thalamus
 - Hypothalamus
- Temporal Lobe Structures:
 - Hippocampus
 - Amygdala
- White Matter Structures
 - Corpus Callosum
 - Fornix
 - Internal Capsule
- Brainstem
 - Midbrain
 - Pons
 - Medulla
- Cerebellum
- Lobes
 - Frontal Lobe
 - Temporal Lobe
 - Parietal Lobe
 - Occipital Lobe
 - Cingulate Gyrus
 - Insula

Acknowledgements

- *VHD Lesson prepared by Laura Weinkle and Samantha Wilkerson*
- *Segmentation and Design: Zachary Drapkin, MD, Emily Mastej, MS, Michael Corigliano, PhD, Jake Shearer, PhD.*
- *Touch of Life Technologies*

References

- Estevez ME, Lindgren KA, Bergethon PR. 2010. A novel 3-dimensional tool for teaching human neuroanatomy. *Anat Sci Educ.* 3: 309-317.
- Drapkin ZA, Lindgren KA, Lopez MJ, Stabio ME. 2015. Development and assessment of a new 3D neuroanatomy teaching tool for MRI training. *Anat Sci Educ.* 8(6):502-9.
- The Human Brain in Photographs and Diagrams, by J. Nolte and J. Angevine copyright © 2000, Mosby Inc.

UNIT III:

SPINAL CORD, BRAINSTEM & CRANIAL NERVES

-

Lecture 16-17: Brainstem Anatomy & Cross-sections

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Learning Objectives

1. List the main functions of the brainstem.
2. List the 3 major divisions of the brainstem (midbrain, pons and medulla) and the 3 longitudinal divisions (tectum, tegmentum, basis).
3. Identify the external features of the midbrain, pons, and medulla on models, diagrams and cross-sections.
4. Identify the internal features of the midbrain, pons, and medulla on cross-sections.
5. List major neurotransmitter “production centers” in the brainstem for serotonin, dopamine, norepinephrine, and enkephalin.

Terms to Know

- Midbrain
 - Gross external features:
 - Cerebral peduncles
 - Interpeduncular fossa
 - Tectum of the midbrain
 - Internal features:
 - Superior colliculus (the actual nucleus inside)
 - Inferior colliculus (the actual nucleus inside)
 - Decussation of the superior cerebellar peduncle
 - Cerebral aqueduct
 - Periaqueductal grey (enkephalin)
 - Substantia nigra (dopamine)
 - Ventral tegmental area (dopamine)
 - Red nucleus
- Pons
 - Gross external features:
 - Pontine protuberance
 - Middle cerebellar peduncle
 - Internal features:
 - Pontine nuclei
 - Locus coeruleus
 - Raphe nucleus (serotonin)
 - Fourth ventricle
- Medulla oblongata:
 - Gross external features:
 - Olive
 - Pyramids
 - Inferior cerebellar peduncle
 - Internal features:
 - Inferior olivary nucleus
 - Nucleus gracilis
 - Nucleus cuneatus
 - Decussation of corticospinal tract (aka: pyramidal decussation)

Caudal Medulla	Rostral Medulla	Pons	Midbrain	
				Distinguishing features
				Cross sectional shape
				Cranial <i>nerve</i> emerges from?
				CN <i>nuclei</i> location
				Other Nuclei
				ventricular system?

Lecture 18: Spinal Cord Anatomy: Cross-Sections & Reflexes

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Spinal Cord Structure (Ch. 26): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/spinal-cord-structure/>

Neuroscience Online Reflexes (Ch. 2) [Spinal Reflexes and Descending Motor Pathways \(Section 3, Chapter 2\) Neuroscience Online: An Electronic Textbook for the Neurosciences | Department of Neurobiology and Anatomy - The University of Texas Medical School at Houston](#)

Learning Objectives

1. Describe the gross structure of the spinal cord and its anatomical relationship with the vertebral column.
2. List the name and number of the spinal nerves and describe their anatomical relationship with the vertebral column.
3. Identify structural divisions of the spinal cord: gray matter (dorsal horn, lateral horn, intermediate gray, and ventral horn) vs. white matter (dorsal funiculus, lateral funiculus & ventral funiculus).
4. Identify the major functional divisions of the spinal cord on representative sections at different levels: motor (lateral vs anterior corticospinal tracts), fine touch (fasciculus gracilis, fasciculus cuneatus) and pain/temp (Lissauer's tract, substantia gelatinosa, nucleus proprius, and spinothalamic tract).
5. Identify the location of neuronal cell bodies for preganglionic sympathetics (aka lateral horn).
6. Distinguish the different levels of the spinal cord from cross-sectional images using the "5 rules" guide.
7. Sketch the direction and type of information flow in spinal rami, spinal roots and the spinal cord.
8. Draw the afferents and efferents of the myotatic stretch reflex & describe its function.
9. Draw the afferents and efferents of the flexor-withdrawal reflex & describe its function.
10. Compare and contrast these two reflexes in terms of their function and number of synapses.

Terms to Know

- Major structures of the spinal cord (what function underlies these structural specializations?)
 - Cervical enlargement
 - Lumbar enlargement
 - Cauda equina
 - Conus medullaris
 - Filum terminale
 - Spinal tap/lumbar puncture
 - Spinal nerves
 - Cervical (8 pairs of nerves; how many vertebrae? _____)
 - Thoracic (12 pairs of nerves; how many vertebrae? _____)
 - Lumbar (5 pairs of nerves; how many vertebrae? _____)
 - Sacral (5 pairs of nerves; how many vertebrae? _____)
 - Coccygeal (1 pair; how many vertebrae? _____)
- Structural components of spinal cord cross-section
 - Grey matter/white matter
 - Dorsal horn/ventral horn
 - Dorsal root/ventral root
 - Dorsal rami/ventral rami
 - Review: remember "SAME" = "Sensory Afferent / Motor Efferent"
 - Dorsal root ganglia

- Dorsal/lateral/ventral funiculus
- Spinal cord cross-section – Functional/histological divisions
 - (see diagram on subsequent page)
- Five rules to distinguish the level of spinal cord cross-section:
 - **Look at the grey/white ratio**
 - Rule: Rostral levels have more white matter than caudal levels. Why?
 - **Look at the lateral horn.**
 - Rule: if you see a lateral horn, you're in the thoracic segment because the intermediolateral cell column is T1-L2 (preganglionic sympathetic neurons).
 - **Look at the ventral horn.**
 - Rule: if the ventral horn is enlarged laterally and looks like a boot, then you're probably in an enlargement. Why is the ventral horn enlarged in these cases?
 - Cervical enlargement is C3-T2
 - Lumbar enlargement is L1-S3
 - **Look at the dorsal columns**
 - Rule: if you see only fasciculus gracilis, you are caudal to T6; if you see both fasciculus gracilis and fasciculus cuneatus, you are at T6 or rostral to it.
 - **Look for cross-sections of the cauda equina around the spinal cord**
 - Rule: if you see lots of cauda equina, you are in the sacral levels.

Label structures related to ascending sensory systems on the left

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____

7. Clarke's Column (cerebellar

pathway)

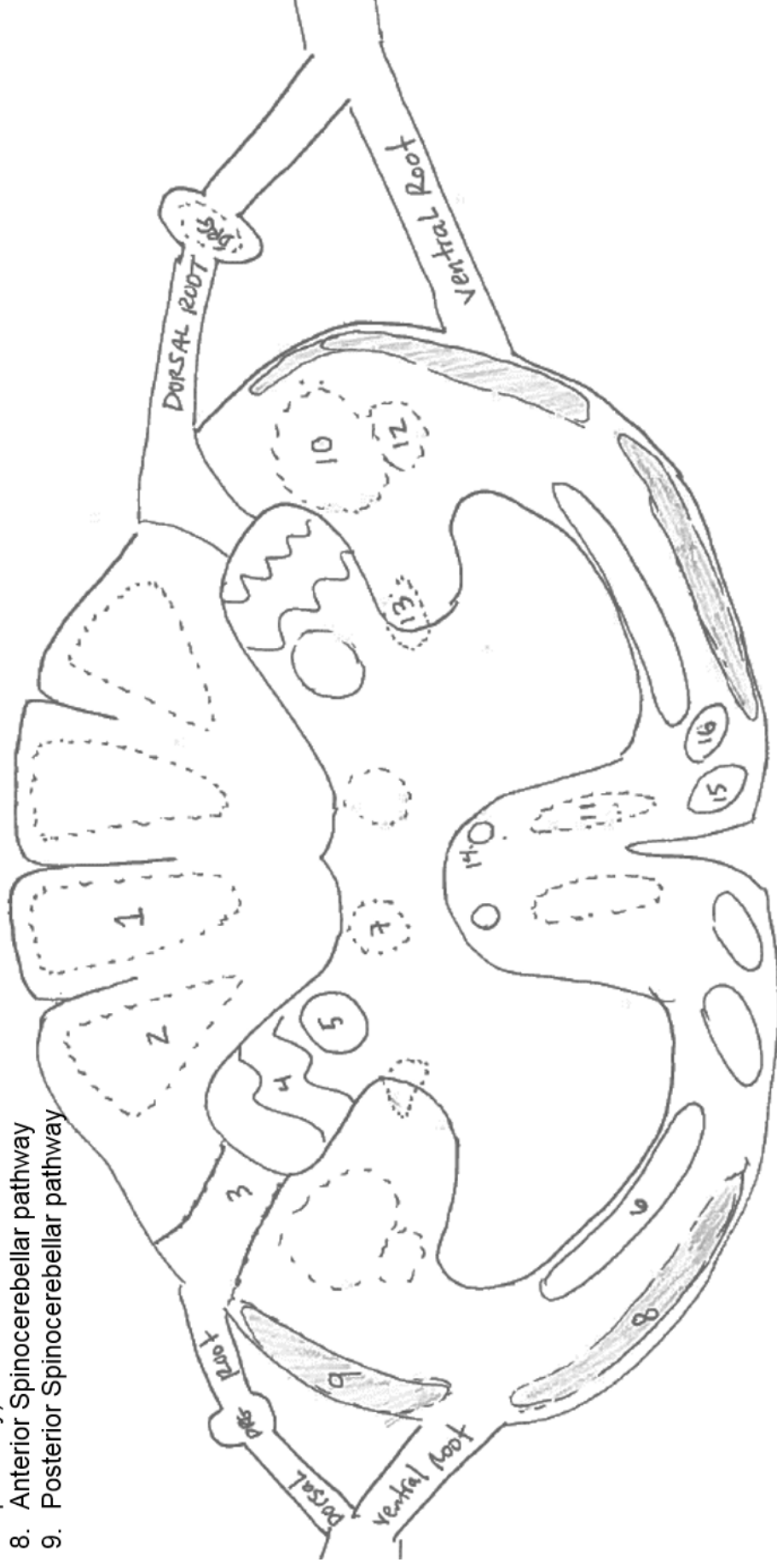
8. Anterior Spinocerebellar pathway

9. Posterior Spinocerebellar pathway

Please memorize #1-6, 10, 11 13 for your exam. The other items are for reference only.

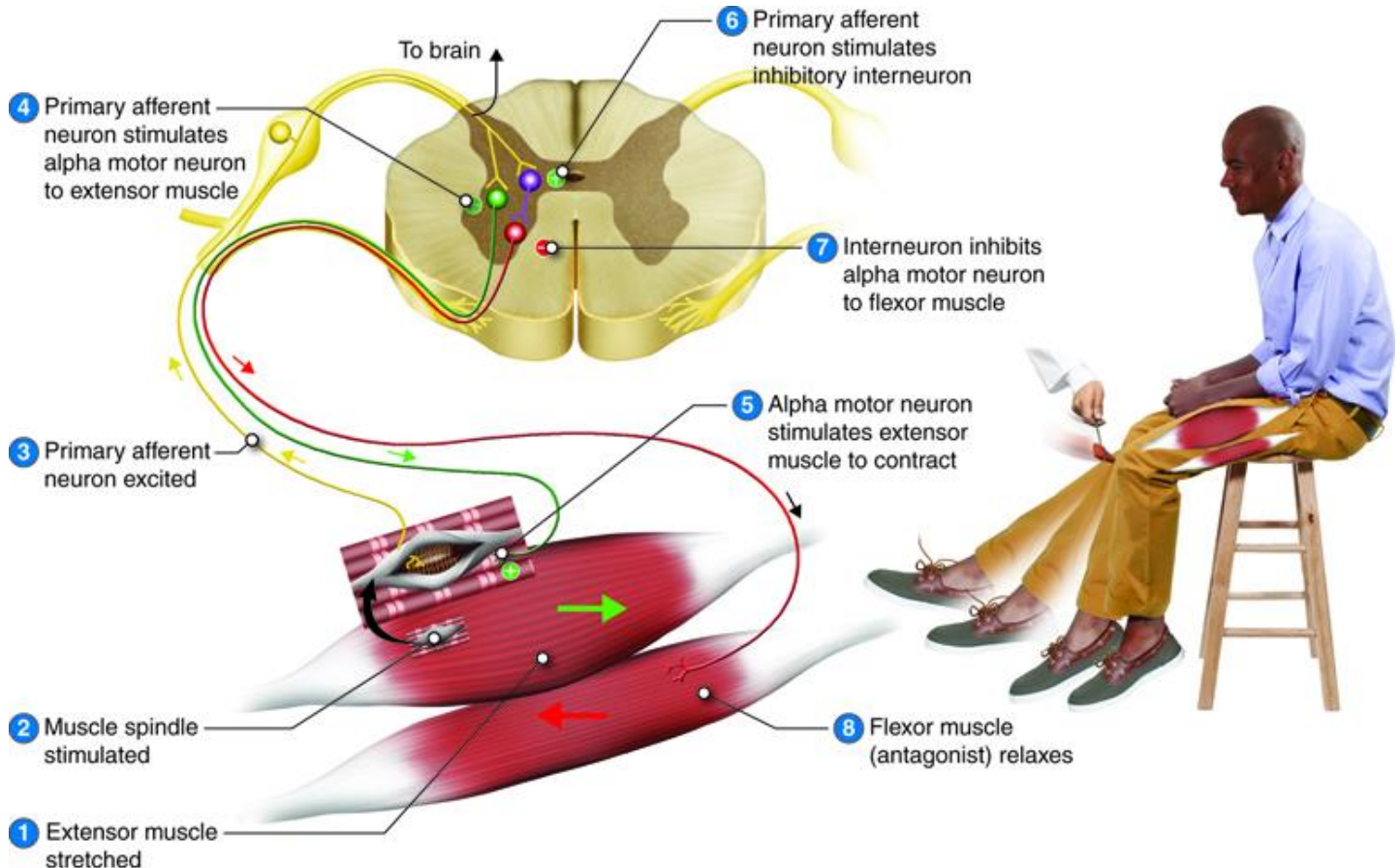
Label structures related to descending motor systems on the right

10. _____
11. _____
12. Rubrospinal Tract
13. _____
14. Medial Vestibulospinal Tract
15. Tectospinal tract
16. Reticulospinal Tract



- **Myotatic Stretch Reflex**

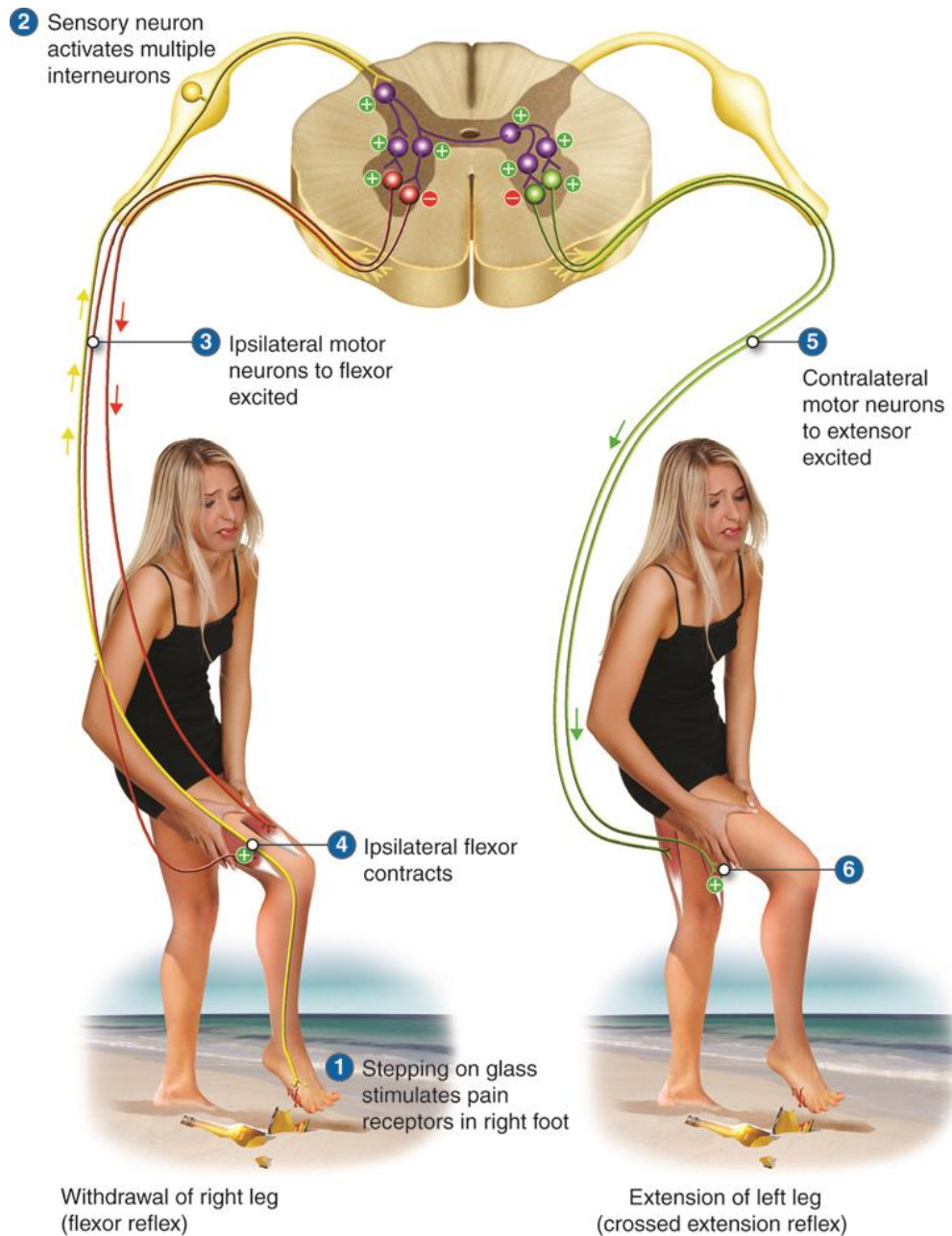
- Monosynaptic and excitatory onto the homonymous muscle and its synergistic muscles.
- Disynaptic and inhibitory onto the antagonist muscle.
- When a muscle is stretched (1), muscle spindles (2) send information to the spinal cord through a first-order afferent neuron (pseudounipolar). This information will enter through the dorsal root and head towards the DCML pathway; on its way, it sends a collateral (branch) into the dorsal horn of the spinal cord. (3) When it reaches the ventral horn, it will synapse on an alpha motor neuron of the same muscle, (4) which exits the spinal cord through the ventral root and synapses on the homonymous skeletal muscle. (5) At the same time, stimulation of an inhibitory interneuron (6) prevents contraction of the antagonistic muscle (7 and 8).



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- Flexion-crossed extension reflex

- Afferent neuron = 1st order afferent neuron carrying pain information through the dorsal root to the spinothalamic pathway sends axon collaterals (branches) into the spinal cord to synapse onto alpha motor neurons on the same side; these neurons also cross through the anterior commissure of the spinal cord to synapse onto alpha motor neurons on the opposite side.
- Efferent neuron = alpha motor neurons in the ventral horn on both sides send axons through the ventral root to synapse onto skeletal muscle. This reflex causes contraction of the flexor and relaxation of the extensor on the stimulated leg (where the pain came from). The reflex also causes contraction of the extensor and relaxation of the flexor on the opposite side (to stabilize the standing leg). All circuits are polysynaptic.



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Lecture 19: Somatosensation: Fine Touch, Proprioception & Vibration (DCML)

Tom Finger, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

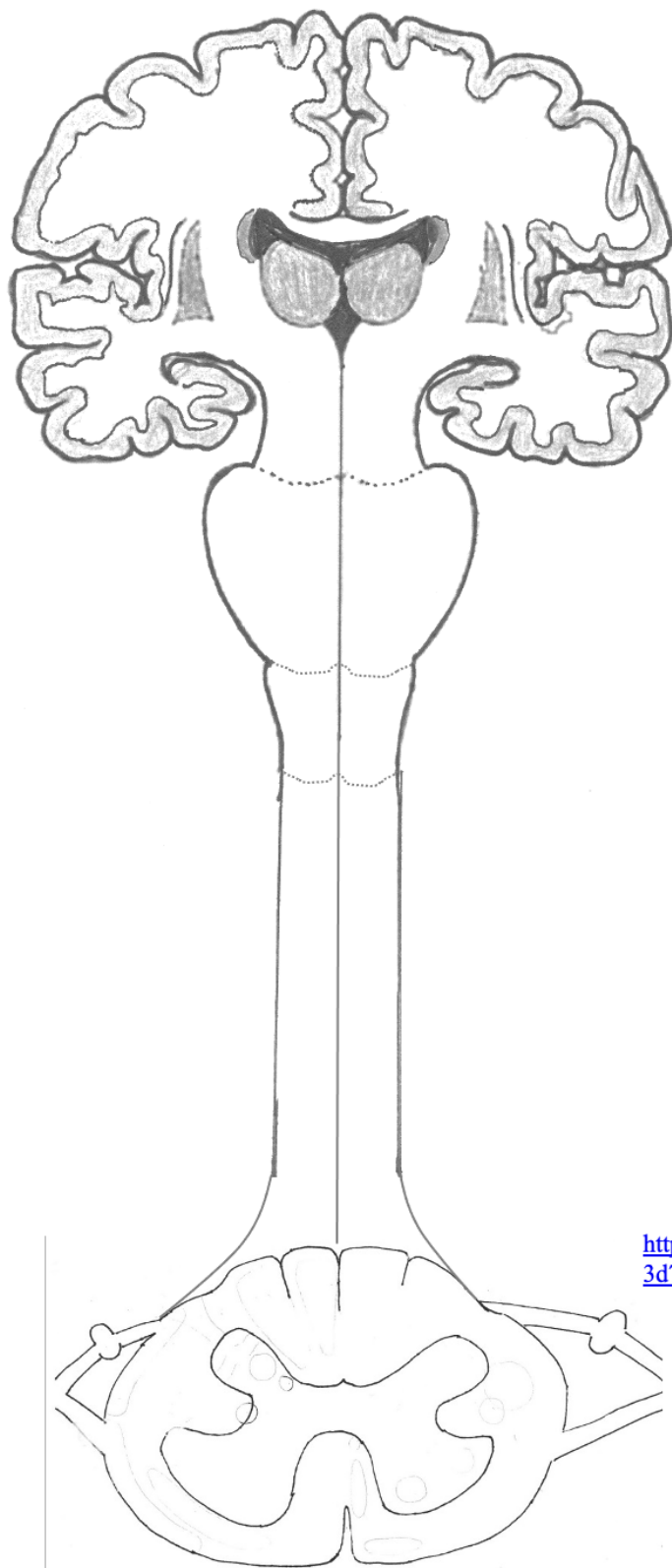
Touch: Central Processing (Ch. 38) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/touch-central-processing/>

Learning Objectives

1. Define somatosensation and discuss the concept of “receptive fields.”
2. Describe the type of information carried in the “Dorsal Column – Medial lemniscus” pathway as well as the types of sensory receptors utilized in this pathway.
3. Draw the DCML pathway on a diagram and identify the location of the decussation.

Terms to Know

- Name: Dorsal Column-Medial Lemniscus. It contains:
 - Fasciculus gracilis or cuneatus (these are the “dorsal columns”)
 - Medial Lemniscus
- Type of information: This is a major ascending pathway that sends sensory information about fine touch, pressure, vibration, and joint position (proprioception) from the body to the brain.
- Circuit: 3 neurons total (the second neuron decussates)
 - Pathway of first-order neuron:
 - Receptors: Meissner’s corpuscles and Pacinian corpuscles are located in the skin. Proprioceptors in the muscles and joints.
 - Somas: located in the dorsal root ganglion (these are pseudounipolar neurons).
 - Axons: enter through the dorsal root. They do NOT cross at the level of the spinal cord. They go immediately into the dorsal columns and head straight up towards the medulla on the ipsilateral side in:
 - Fasciculus gracilis (caudal to T6)
 - Fasciculus cuneatus (T6 and rostral)
 - The axons in the fasciculi will make their first synapse onto the second-order neurons in the caudal medulla
 - Pathway of a second-order neuron
 - Somas are located in nucleus gracilis or nucleus cuneatus.
 - Axons of the second-order neurons immediately decussate (the decussating fibers are called “internal arcuate fibers”). After decussating, the axons travel together in a bundle that looks like a ribbon, called the medial lemniscus.
 - The medial lemniscus travels rostrally to the thalamus, where its axons synapse onto third-order neurons.
 - Pathway of third-order neuron
 - Somas are located in the thalamus.
 - Axons of the third-order neurons travel to the cortex and terminate in the primary somatosensory cortex (Brodmann Area 3,1,2)



Name: _____

Function: _____

Ascending or Descending? _____

Location of decussation: _____

1st order neuron:

- Receptors _____
- Cell bodies _____
- Axons _____

2nd order neuron:

- Cell bodies _____
- Axons _____

3rd order neuron:

- Cell bodies _____
- Axons _____
- Termination _____

<https://sketchfab.com/3d-models/ascending-tracts-3d7e233edbb94cd8b9e1b4047bead951>

Lecture 20: Somatosensation: Pain & Temperature (Spinothalamic)

Tom Finger, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

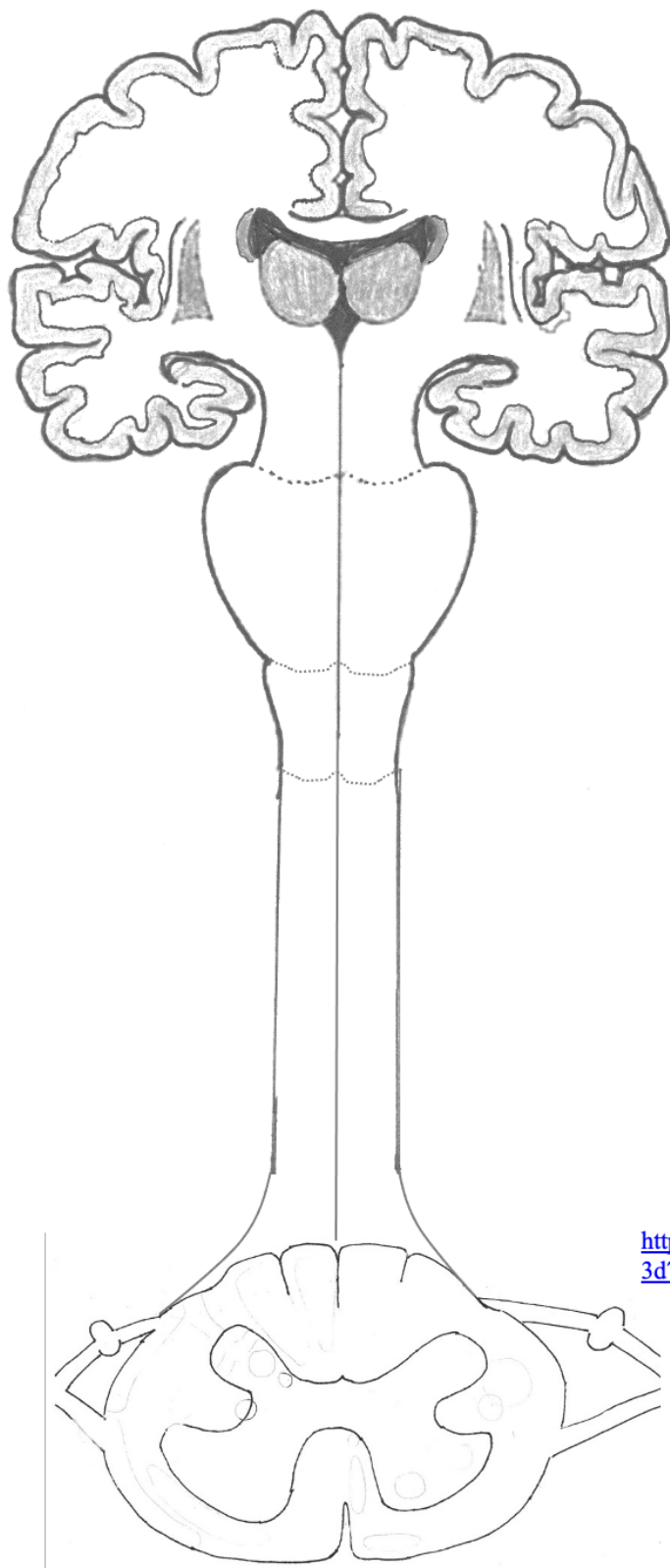
Pain and Nociception (Ch. 39): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/pain/>

Learning Objectives

1. Differentiate nociception vs. pain.
2. Describe the types of receptors and afferent nerve fibers that carry pain and temperature information.
3. Compare and contrast the structure and function of A-delta and C fibers in the conduction of pain.
4. Draw the spinothalamic pathway on a diagram and identify the location of the decussation.
5. Describe how and where pain is interpreted in the brain (what makes pain “feel bad” if it is just action potentials signaling along a somatosensory pathway?)

Terms to Know

- Name: Anterolateral system. It contains many tracts. You only need to know one, which is the Spinothalamic tract
- Type of information: This is a major ascending pathway that sends sensory information about pain and temperature (some crude touch) from the body to the brain.
- Circuit: 3 neurons total (the second neuron decussates)
 - Pathway of first-order neurons
 - Receptors: free nerve endings (nociceptors, thermoreceptors, and some mechanoreceptors)
 - A-delta fibers: myelinated, fast, sharp pain
 - C-fibers: unmyelinated, slow, dull pain
 - Somas: located in the dorsal root ganglion (these are pseudounipolar neurons)
 - Axons: enter the spinal cord through the dorsal root and head to the dorsal horn through Lissauer’s tract.
 - These first-order neurons synapse in the dorsal horn in the substantia gelatinosa
 - Pathway of a second-order neuron
 - Somas are located in the nucleus proprius
 - Axons of the second-order neurons do decussate at the level of the spinal cord in the anterior white commissure of the spinal cord. After decussating, the axons travel in the spinothalamic tract. The way to remember this name is that the axons in this tract go straight from the spinal cord to the thalamus without synapsing, thus they are spino-thalamic.
 - The axons of the spinothalamic tract synapse in the thalamus.
 - Pathway of a third-order neuron
 - Somas are located in the VPL of the thalamus.
 - Axons of the third-order neurons travel to the cortex and terminate in the primary somatosensory cortex (Brodmann Area 3,1,2)



Name: _____

Function: _____

Ascending or Descending? _____

Location of decussation: _____

1st order neuron:

- Receptors _____
- Cell bodies _____
- Axons _____

2nd order neuron:

- Cell bodies _____
- Axons _____

3rd order neuron:

- Cell bodies _____
- Axons _____
- Termination _____

<https://sketchfab.com/3d-models/ascending-tracts-3d7e233edbb94cd8b9e1b4047bead951>

Lecture 21: Corticospinal Pathway

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Execution of Movement (Ch. 44) (corticospinal & corticobulbar tracts):

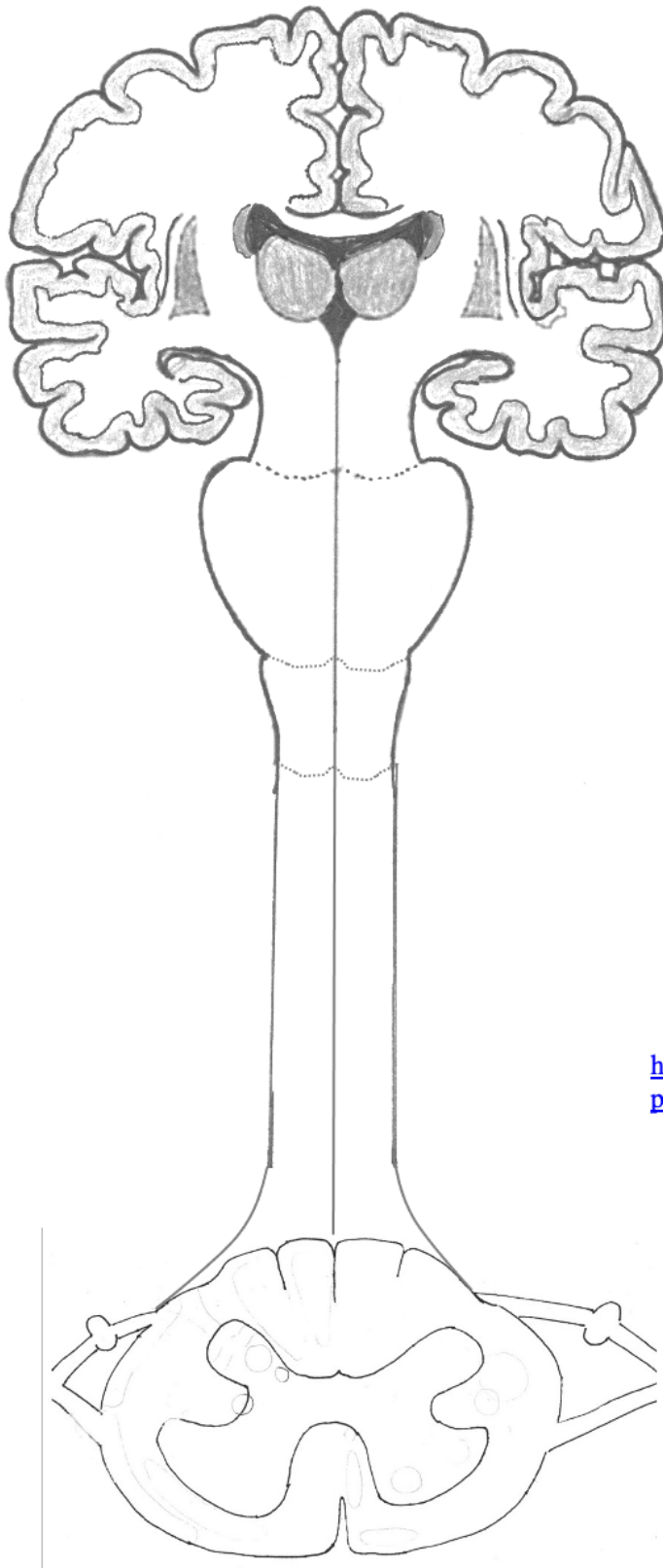
<https://openbooks.lib.msu.edu/introneuroscience1/chapter/execution-of-movement/>

Learning Objectives

1. Describe the type of information carried in the corticospinal pathway.
2. Draw the corticospinal pathway on a diagram and identify the location where this pathway decussates.
3. Identify the parts of the corticospinal pathway in cross-sections of the brain, brainstem and spinal cord.
4. Define upper motor neuron (UMN) versus lower motor neuron (LMN) and identify the location of the soma, axon, and synapse of each.
5. Describe classic features of UMN vs. LMN lesions.
6. Review the homunculus for the primary motor cortex.
7. Describe the role of the Central Pattern Generator in locomotion.

Terms to Know

- Name: Corticospinal Pathway. It contains two main tracts:
 - Anterior Corticospinal Tract (bilateral)
 - Lateral Corticospinal Tract (crossed)
- Type of information: This is the major descending pathway that sends motor commands from the brain to skeletal muscles of the body.
- Circuit: 2 neurons total (rule: the first neuron decussates)
 - Pathway of first-order neuron (or upper motor neuron):
 - Somas in the primary motor cortex (Brodmann area 4; pre-central gyrus)
 - Axons are bundled together as a tract and pass through the following structures:
 - Corona radiata
 - Internal capsule
 - Cerebral peduncle (midbrain)
 - Pons
 - Pyramids (medulla)
 - Spinomedullary junction = decussation of corticospinal tract
 - 80% of fibers decussate and become the lateral corticospinal tract (mostly muscles of the upper and lower limbs)
 - 20% of fibers do not decussate and become the anterior corticospinal tract (mostly axial/postural muscles)
 - Axons then synapse onto alpha motor neurons of the ventral horn
 - Pathway of second-order neuron (or lower motor neuron):
 - Somas are located in the ventral horn of the spinal cord (alpha & gamma LMNs)
 - Axons depart the spinal cord via ventral root to terminate onto skeletal muscle



Name: _____

Function: _____

Ascending or Descending? _____

Location of decussation: _____

Upper Motor Neuron:

- Cell bodies _____
- Axons _____

Lower Motor Neuron:

- Cell bodies _____
- Axons _____
- Termination _____

<https://sketchfab.com/3d-models/corticospinal-pathway-ab008dad830f496abe25a4cf7b84ee0d>

Signs	UMN Lesion	LMN Lesion
Type of Paralysis		
Deep Tendon Reflexes		
Atrophy		
Muscle Tone		
Babinski Reflex		
Muscle Fasciculations		

Lecture 22: Cross-Sectional Anatomy of the Brainstem & Spinal Cord **REVIEW**

Maureen Stabio, PhD – Department of Cell and Developmental Biology

1. Upper
Midbrain

2. Lower
Midbrain

3. Upper
Pons

4. Lower
Pons

5. Upper
Rostral
Medulla

6. Lower
Rostral
Medulla

7. Upper
Caudal
Medulla

8. Lower
Caudal
Medulla

Spinal Cord Sections (slightly enlarged relative to brainstem)

9. Cervical Enlargement

10. Thoracic

*(How do you tell the
difference between upper
vs. lower thoracic?)*

11. Lumbar Enlargement

12. Sacral

Acknowledgements

The brainstem and spinal cord sections are used with permission from Boston University School of Medicine Neuroanatomy Atlas. Special thanks to the Department of Anatomy and Neurobiology. These sections were stained with luxol fast blue and for cells with cresyl violet (method of Kluver and Barrera).

Lecture 23-24: Cranial Nerves and their Nuclei

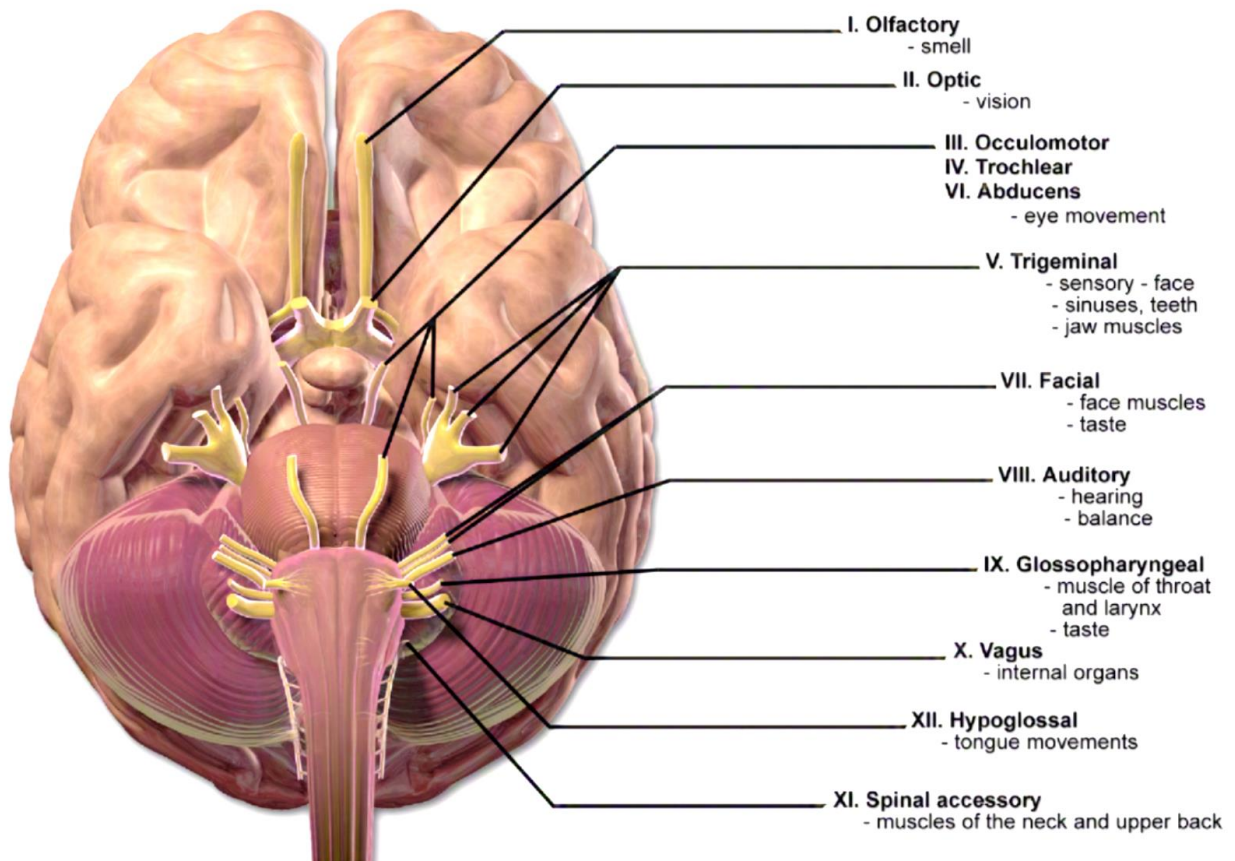
Maureen Stabio, PhD – Department of Cell and Developmental Biology

Learning Objectives

1. List names, numbers, and functions of the cranial nerves.
2. Identify the location of the cranial nerves on the surface of the brainstem and the location of cranial nerve nuclei inside the brainstem.
3. List the afferent & efferent limbs of the jaw jerk reflex, corneal blink reflex, pupillary light reflex & gag reflex.

Terms to Know

- Cranial Nerves
 - I. Olfactory
 - II. Optic
 - III. Oculomotor
 - IV. Trochlear
 - V. Trigeminal
 - VI. Abducens
 - VII. Facial
 - VIII. Vestibulocochlear
 - IX. Glossopharyngeal
 - X. Vagus
 - XI. Spinal Accessory
 - XII. Hypoglossal



#	Name	Type (S,M,B)	Function	How to Assess Clinically
I				
II				
III				
IV				
V				
VI				
VII				
VIII				
IX				
X				
XI				
XII				

- Cranial nerve nuclei (*you do not need to identify starred items in cross-section)
 - In the midbrain (3,4)
 - Oculomotor Nucleus
 - Edinger-Westphal Nucleus
 - Trochlear Nucleus
 - In the Pons (5,6,7)
 - Mesencephalic nucleus of the trigeminal
 - Primary motor nucleus of the trigeminal
 - Primary sensory nucleus of the trigeminal
 - Abducens nucleus*
 - Facial nucleus (note: there is an upper and lower division to this nucleus)*
 - Spinal nucleus of the Trigeminal*
 - At the Pontomedullary junction (8)
 - Spinal nucleus of trigeminal*
 - Vestibular nucleus
 - Cochlear nucleus
 - In the Rostral Medulla (9,10,12)
 - Spinal nucleus of the trigeminal*
 - Superior and inferior salivatory nuclei*
 - Nucleus of the solitary tract
 - Dorsal motor nucleus of Vagus*
 - Nucleus Ambiguus (ventral motor nucleus of vagus)*
 - Hypoglossal nucleus
 - In the Caudal Medulla
 - Spinal nucleus of the trigeminal – ID this nucleus at the caudal medulla only
 - Nucleus Gracilis
 - Nucleus Cuneatus
- Reflexes
 - Jaw Jerk Reflex (Afferent = mesencephalic nucleus V; Efferent = main motor nucleus V)
 - Corneal Reflex (Afferent = spinal nucleus of V; Efferent = motor nucleus of VII)
 - Pupillary Light Reflex (Afferent = optic nerve; Efferent = parasympathetic division of III (Edinger-Westphal nucleus to ciliary ganglia))
 - Gag Reflex (Afferent = glossopharyngeal nerve; Efferent = Vagal division of nucleus ambiguus)

Reflex	Afferent	Efferent	Brainstem Level
Pupillary light reflex			
Jaw jerk reflex			
Corneal reflex			
Gag reflex			

LAB 4: Brainstem & Spinal Cord

MHA Teaching Team

Station 1

Learning Objectives:

1. Review the structural components of the CNS and PNS.
2. Identify gross features and vasculature of the spinal cord.
3. Describe the structure and function of spinal cord horns, roots, & dorsal root ganglia.

Learning Tools: Full central nervous system (WET) prosection, spinal cord with cauda equina, and pointers.

Instructions: Observe and appreciate the full central nervous system. The thoughts, movements, sensations and emotions of an individual were all housed here, and we will spend the entire neuroanatomy course working and learning in this space. Please use gloves and treat the tissues VERY GENTLY.

1. First review the components of CNS vs. PNS. Identify key components of the CNS on the full CNS extraction including cerebrum, cerebellum, brainstem, and spinal cord. Notice the optic tract, optic chiasm, optic nerve, eyes and extraocular muscles.
2. Orient in anatomical space one of the individual spinal cords (i.e. a spinal cord without brain attached) and identify dorsal vs. ventral and rostral vs. caudal.
3. Then identify the following structures on the spinal cord.
 - Cervical Enlargement
 - Lumbar Enlargement
 - Cauda Equina
 - Conus Medullaris
 - Filum Terminale
4. Identify the spinal cord arteries and describe the territories they supply.
 - Anterior spinal artery (1)
 - Posterior spinal artery (2)
5. Some structures may not be visible on all spinal cords in the lab. Can you find these?
 - Anterior (ventral) root:
 - What is the structural classification of neurons in the anterior root?
 - What is the functional classification of neurons in the anterior root?
 - What type of information (sensory or motor) is carried by neurons here?
 - Posterior (dorsal) root:
 - What is the structural classification of neurons in the posterior root?
 - What is the functional classification of neurons in the posterior root?
 - What type of information (sensory or motor) is carried by neurons here?
 - Posterior root ganglion (aka: dorsal root ganglion):
 - What anatomical part of the neuron is located in the dorsal root ganglion?
 - What type of glial cells would you find in the dorsal root ganglion?
6. Now observe a cross section from a piece of the spinal cord.
 - Can you identify grey matter vs. white matter?
 - What is found in the white matter vs. grey matter of the spinal cord?
 - Can you identify ventral horns vs. dorsal horns vs. lateral horn?

Station 2

Learning Objectives:

1. Illustrate the anatomical relationships between the spinal cord, vertebral column and spinal nerves
2. explain why this anatomical relationship is important clinically for lumbar puncture.

Learning Tools:

Full Skeleton, Vertebral column plastic model, pipe cleaners, and pointers

Instructions:

Complete the following items #1-6

1. Using the spinal cord model at this station, name and count the vertebral bones. How many of each type (cervical, thoracic, lumbar, sacral and coccygeal) of **bones** are there?
2. Then name and count all the spinal nerves. How many of each type (cervical, thoracic, lumbar, sacral and coccygeal) of **nerves** are there? (remember these come in pairs: L & R)
3. Use a pointer on the skeleton model and indicate where each spinal nerve exits in relationship to its vertebral bone. For example, does C1 nerve exit above or below C2 vertebral bone?
TO DO: As a team, place the pipe cleaners in the vertebral column where the spinal nerves would exit
USE matching colors for each level
4. Use a pointer to point to the vertebral bone corresponding to the location of the conus medullaris of the spinal cord (i.e., where on the vertebral column does the spinal cord end?). Summarize the relationship between the length of the vertebral column and the length of the spinal cord.
5. Simulate a lumbar puncture. Use a pointer to point to the space on the vertebral column where a needle would be inserted for a lumbar puncture. Why is the lumbar puncture performed at this location?
6. What is collected in a lumbar puncture and why? Name one infection and one disorder of the central nervous system that might be diagnosed with the help of a lumbar puncture.

Station 3

Learning Objectives:

1. Draw the pathway of the myotatic stretch reflex using the patellar reflex as an example. Define all parts of this reflex and describe whether it is monosynaptic or polysynaptic.
2. Draw the pathway of the flexor-withdrawal reflex. Define all parts of this reflex and describe whether it is monosynaptic or polysynaptic.

Learning Tools: Reflex hammer, laminated diagrams and dry-erase markers

Instructions:

1). Test your myotatic stretch reflex using a reflex hammer. Break up into pairs. One person will be the tester; the other person should sit on the edge of a table with both legs dangling and totally relaxed. The tester will use the reflex hammer to tap the patellar tendon and elicit the knee-jerk reflex. Then, swap. On the diagram below, draw the pathway for this reflex. Use a (+) for excitatory synapses and a (-) for inhibitory synapses. Label your drawing using the terms listed in the box.

Summary: Stretch Reflex

- Monosynaptic and excitatory onto homonymous muscle
- Disynaptic and inhibitory onto the antagonist muscle.
- **Afferent neuron of stretch reflex** = 1st-order afferent neuron carrying proprioceptive information through the dorsal root to the DCML pathway sends axon collaterals (branches) into the spinal cord at the level of entry to synapse onto alpha motor neurons in the ventral horn
- **Efferent neuron of stretch reflex** = alpha motor neurons in the ventral horn send axons through the ventral root to synapse onto skeletal muscle. This reflex causes excitation (contraction) onto the homonymous muscle (the same muscle from whence the stretch came); this is a monosynaptic circuit. The reflex is also disynaptic and inhibitory onto the antagonist muscle.

2). Compare and contrast the myotatic reflex with the flexor-withdrawal reflex.

Standing next to your table, act out the flexor-withdrawal reflex as if you stepped on a sharp piece of glass with your right foot. Which leg will flex and which leg will extend? On the diagram below, draw the circuit for this reflex and indicate with a (+) or (-) which muscles will contract or relax, respectively. Label all components of the reflex using the terms listed in the box.

➤ Summary: Flexion-crossed extension reflex (AKA: flexor-withdrawal reflex)

- **Afferent neuron** = 1st order afferent neuron carrying pain information through the dorsal root to the spinothalamic pathway sends axon collaterals (branches) into the spinal cord to synapse onto alpha motor neurons on the same side; these neurons also cross through the anterior commissure of the spinal cord to synapse onto alpha motor neurons on the opposite side.
- **Efferent neuron** = alpha motor neurons in the ventral horn on both sides send axons through the ventral root to synapse onto skeletal muscle. This reflex causes contraction of the flexor and relaxation of the extensor on the stimulated leg (where the pain came from). The reflex also causes contraction of the extensor and relaxation of the flexor on the opposite side (to stabilize the standing leg). All circuits are polysynaptic.

Station 4

Learning Objectives:

1. Label the functional divisions of the spinal cord cross-section & differentiate grey matter vs. white matter.

Learning Tools: Diagram & plastinated spinal cord cross-sections

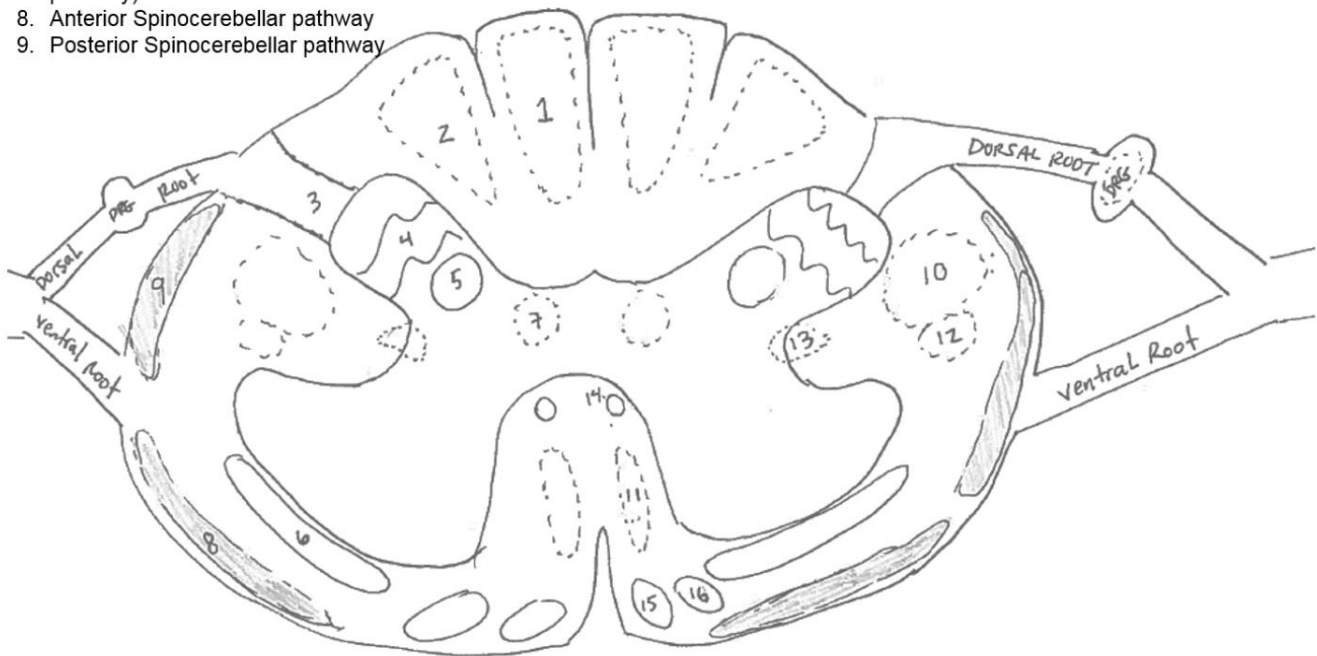
Instructions: First, observe the plastinated spinal cord cross-sections. Can you differentiate grey vs. white matter? Next, fill out the functional divisions on the diagram below. Which parts are “on and off” ramps for pathways and which parts are “elevators” as described in class?

Label structures related to ascending sensory systems on the left

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____
7. Clarke's Column (cerebellar pathway)
8. Anterior Spinocerebellar pathway
9. Posterior Spinocerebellar pathway

Label structures related to descending motor systems on the right

10. _____
11. _____
12. Rubrospinal Tract
13. _____
14. Medial Vestibulospinal Tract
15. Tectospinal tract
16. Reticulospinal Tract



Station 5

Learning Objective: Distinguish the different levels of the brainstem and spinal cord in cross-section.

Learning Tools: Scrambled laminated photographs of spinal cord and brainstem cross-sections

Instructions: Sort the cross-sections from rostral to caudal. Re-scramble when complete.

Station 6

Learning Objective: Explore the Central Nervous System in VR

Learning Tools: Virtual Reality and the VH Dissector

Instructions: Go to the Plastination Lab and follow instructions.

1. Go to the Plastination Lab (Ed1-5101) and open the VH Dissector in the VR system.
2. There are four students in a group and two headsets. So you will work in pairs. Each person will have 5 minutes in the VR headset to explore the CNS.
3. The Visible Man will open by default.
4. Click on "SYSTEMS".
5. Turn off all systems. Turn on (with the eyeball icon) the skeletal and nervous systems.
6. Perform a laminectomy of the lumbar spine. Look for the lumbar vertebrae and dissect the spinous processes to find the conus medullaris.
7. Where did the spinal cord end in this donor? At what vertebral level?
8. Now put the vertebral bones back on.
9. Put a pin where you would place the needle for a lumbar puncture.
10. Explore any other aspects of the nervous system that you would like to explore in VR. HAVE FUN!

LAB 5: Lesion Localization of the Spinal Cord

Maureen Stabio, PhD – Cell & Developmental Biology

Learning Objectives

1. Test two-point discrimination thresholds for the somatosensory (fine touch) system.
2. Review the neuroanatomy of a spinal cord cross-section.
3. Review three major spinal cord pathways (Corticospinal, DCML and Spinothalamic) and identify decussation locations.
4. Compare and contrast clinical presentation of UMN vs. LMN lesions.
5. Demonstrate lesion localization to solve spinal cord cases.

Learning Tools

2-point discrimination tool

PART A: Map your receptive fields

Break into groups of two. Determine the receptive field sizes for the fine touch neurons in your skin using the two-point discrimination tool. One person in the group will be the tester; the other person should be blind-folded (or with closed eyes). The tester should adjust the distance of the sliding section of the tool to make two points. Hold the two points with equal (light) pressure on the skin at various points of the body as listed in the table below. Determine the minimum distance required in order for the two points to be recognized by your partner. Record your data in the table below. Then, swap.

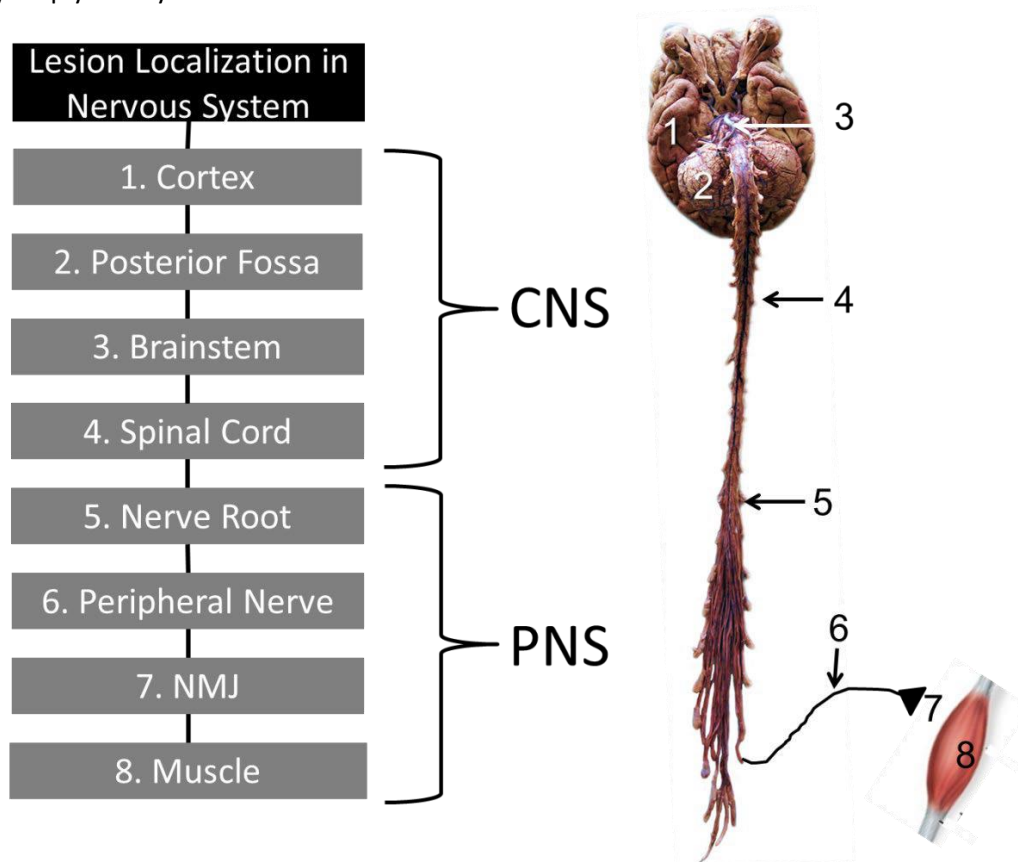
	SUBJECT 1 Minimum threshold distance (mm)	SUBJECT 2 Minimum threshold distance (mm)
Tip of index finger		
Palm of hand		
Forearm (longitudinal axis)		
Back of neck		
Cheek of face		
Leg (calf)		

Questions:

1. What is a receptive field?
2. What part of the body has the lowest two-point discrimination distance value?
3. What part of the body has the smallest receptive fields for fine touch of the skin?
4. What part of the body has the highest sensitivity to fine touch?
5. How does this relate to the somatosensory homunculus?
6. What is the name of the spinal cord pathway that carries this type of somatosensory information to the brain?

Part B: What is Lesion Localization?

Lesion localization in neurology is analogous to finding a light bulb out in a lamp in your home. Where did the problem with the lamp occur? Is the problem with the bulb itself (i.e., a problem with the muscle or neuromuscular junction)? Is the problem with the cord to the lamp (i.e. LMN or peripheral nervous system)? Or is the problem with the circuit breaker or power to the house (i.e. UMN?). This is not a perfect analogy but may help you as you visualize lesion localization.



Part C: How to use a Decision Matrix

To solve lesion localization, there are 5 questions to answer. Answer each question using the matrix below. Read column-by-column starting with the left-hand column.

Step 1: What pathway?	Step 2: Direction of Info Flow?	Step 3: What neurological signs & symptoms?	Step 4: What side?	Step 5: What level?
CST	Descending	UMN = Spastic Paralysis	Are patient's deficits contra or ipsi with respect to the lesion?	Upper body or lower body or both?
		LMN = Flaccid Paralysis		
DCML	Ascending	Loss of two-point discrimination and fine touch (paresthesia)	Are patient's deficits contra or ipsi with respect to the lesion?	Upper body or lower body or both?
Spino-thalamic		Loss of pain/temp		

Part D: Practice Hypothetical Cases

First, we will practice lesion localization with 3 hypothetical cases of lesions occurring in red shaded regions below. Assume these lesions were made with a laser and no inflammation or spreading of damage occurred. These cases are simpler than real-life cases and will help you practice the mental gymnastics of lesion localization and train you to think up and down the spinal cord pathways.

Damage shaded in red	Spinal Cord Level	Pathway Damaged	Expected Symptoms

More Practice with Real Cases on next page

Damage shaded in red	Spinal Cord Level	Pathway(s) Damaged	Expected Symptoms & Name of Disorder
A			
B			
C			
D			
E			
F			

LAB 6: Cranial Nerve Lab

Learning Objective

1. Review the names, numbers, and functions of the twelve cranial nerves.
2. Identify the cranial nerves on the surface of the brainstem (on diagrams, photographs, and models).
3. Identify the location of the cranial nerve nuclei in the brainstem (on diagrams or cross-sections).
4. List the parts of a full neurological exam and perform the Cranial Nerve Exam.
5. Describe the afferent and efferent limbs of cranial nerve reflexes.

Learning Tools

Cranial Nerve Exam Kit (Practice in pairs using the following guide, adapted from “The Precise Neurological Exam” by NYU School of Medicine (<http://informatics.med.nyu.edu/modules/pub/neurosurgery/>)). Use the kit provided for you that includes: odorant, visual acuity eye chart, pen light, 128 Hz tuning fork, Q-tips, and a reflex hammer.

PART A: What is the Neurologic Exam?

The neurological exam is a test used by a physician to assess the health and function of a patient’s nervous system. It can be performed anywhere, but it is typically performed in a doctor’s office during a routine visit. It may also be done following any type of trauma, to follow the progression of a disease or if the patient presents with a neurological complaint. The exam consists of 6 major parts listed below. It may also include a physical exam (vitals) and a review of the patient’s medical history. For today’s lab we will perform the cranial nerve exam only.

Be aware that a full neurological exam consists of all six elements.

1. Mini Mental Status Exam (MMSE) including speech observation
2. **Cranial Nerve Exam (I-XII)**
3. Motor System Exam, including muscle atrophy, tone and power
4. Sensory System Exam, including vibration, position, pin prick, temperature, light touch and higher sensory function
5. Reflexes
6. Coordination, gait, and Romberg’s test.

I. Olfactory Nerve

- a. Function: Special Sensory = smell
- b. How to assess: Evaluate the patency of the nasal passages bilaterally by asking the patient to breathe in through their nose while the examiner occludes one nostril at a time. Once patency is established, ask the patient to close their eyes. Occlude one nostril, and place the odorant (a small bar of soap or a coffee bean) near the patent nostril and ask the patient to smell the object and report what it is. Make certain the patient’s eyes remain closed. Switch nostrils and repeat. Furthermore, ask the patient to compare the strength of the smell in each nostril.

II. Optic Nerve

- a. Function: special sensory = vision
- b. How to assess:
 - Test visual acuity: Test visual acuity by using a pocket visual acuity chart. Perform this part of the examination in a well-lit room and make certain that the patient wears their glasses or contacts if needed. Hold the chart 14 inches from the patient’s face, and ask the patient to cover one of their eyes completely with their hand and read the lowest line on the chart possible. Have them repeat the test covering the opposite eye. If the patient has difficulty reading a selected line, ask them to read the one above. Note the visual acuity for each eye.

- Test visual fields: Next evaluate the visual fields via confrontation. Face the patient one foot away, at eye level. Tell the patient to cover their right eye with their right hand and look the examiner in the eyes. Instruct the patient to keep looking you in the eyes and say "now" when the examiner's fingers enter from out of sight, into their peripheral vision. Once this is understood, cover your left eye with your left hand (the opposite eye of the patient) and extend your arm and first 2 fingers out to the side as far as possible. Beginning with your hand and arm fully extended, slowly bring your outstretched fingers centrally, and notice when your fingers enter your field of vision. The patient should say "now" at the same time you see your own fingers. Repeat this maneuver a total of eight times per eye, once for every 45 degrees out of the 360 degrees of peripheral vision. Repeat the same maneuver with the other eye.
- Assess the optic disc: (skip this one since you will not have an ophthalmoscope. Simply read the following text). Using an ophthalmoscope, observe the optic disc, physiological cup, retinal vessels and fovea. Note the pulsations of the optic vessels, check for a blurring of the optic disc margin and a change in the optic disc's color from its normal yellowish orange.

III. Parasympathetic division of Oculomotor Nerve

- a. Function: visceral motor = parasympathetic to pupillary sphincter and ciliary muscle
- b. How to assess:
 - Perform the pupillary light reflex using the pen light. This will assess the function of both CN II and parasympathetic division of CN III. The afferent component of the reflex is light detection through CN II. The efferent component is parasympathetic innervation to the pupillary sphincter to constrict the pupil. Shining the light in one eye should cause bilateral (consensual) constriction of both pupils. Ask the patient to focus on an object in the distance. Observe the diameter of the pupils in a dimly lit room. Note the symmetry between the pupils. Next, shine the penlight into one eye at a time and check both the direct and consensual light responses in each pupil. Note the rate of these reflexes.
 - You may test for accommodation as well by asking the patient to read the eye chart at various distances (close to nose and at arm's length). Is there any point at which the letters are not in sharp focus?

III, IV & VI. Somatic Motor Divisions of Oculomotor, Trochlear and Abducens Nerves

- a. Function: somatic motor = motor innervation to the extraocular muscles and one eyelid muscle (III = superior rectus, inferior rectus, medial rectus, inferior oblique and levator palpebrae superioris; IV = superior oblique; VI = lateral rectus)
- b. How to assess: Instruct the patient to follow the tip of the penlight (do not turn the light on) with their eyes without moving their head. Move the penlight slowly at eye level, first to the left and then to the right. Then repeat this horizontal sweep with the penlight at the level of the patient's forehead and then chin. Note extra-ocular muscle palsies and horizontal or vertical nystagmus.

Extra at-home work (not tested in this course but will prepare you for gross anatomy next semester):
Watch these videos to review the movements of extraocular muscles:
<https://www.youtube.com/watch?v=6GliSCGkpZ4> <https://www.youtube.com/watch?v=vd70OJ7c1q4>

Examples of cranial nerve palsies can be viewed here:
CN III Palsy: <https://www.youtube.com/watch?v=NAUI-A4qsxs>
CN IV Palsy: <https://www.youtube.com/watch?v=AWJg3Juuhvw>
CN VI Palsy: <https://www.youtube.com/watch?v=3M1GhgV8px0>

V. Trigeminal Nerve

- a. Function: somatic motor = muscles of mastication; somatic (general) sensory = touch of face (includes sensation of cornea)
- b. How to assess:
 - First, test the muscles of mastication. Palpate the masseter and temporalis muscles while you instruct the patient to bite down hard. Also note masseter wasting on observation. Next, ask the patient to open their mouth against resistance applied by the examiner at the base of the patient's chin.
 - Next, test general sensation (touch) of the face. Tell the patient to close their eyes. Use the Q-tip to gently stroke the skin innervated by V1, V2 and V3 on both sides of the face. Ask the patient to report whether they feel the touch at each location.
 - Finally, test the corneal reflex. Use a large Q-tip with the cotton extended into a wisp. Ask the patient to look at a distant object and then approaching laterally, touch the cornea (not the sclera) and look for the eye to blink. Repeat this on the other eye.

VII. Facial Nerve

- a. Function: somatic motor = muscles of facial expression; special sensory = taste anterior 2/3 of tongue; visceral motor = parasympathetic innervation to salivary and lacrimal glands (except parotid)
- b. How to assess:
 - First, test the muscles of facial expression. Initially, inspect the face during conversation and rest noting any facial asymmetry including drooping, sagging or smoothing of normal facial creases. Next, ask the patient to raise their eyebrows, smile showing their teeth, frown and puff out both cheeks. Note asymmetry and difficulty performing these maneuvers. Ask the patient to close their eyes strongly and not let the examiner pull them open. When the patient closes their eyes, simultaneously attempt to pull them open with your fingertips. Normally the patient's eyes cannot be opened by the examiner. Once again, note asymmetry and weakness. Note: When the whole side of the face is paralyzed the lesion is peripheral. When the forehead is spared on the side of the paralysis, the lesion is central (e.g., stroke). This is because a portion of the VII cranial nerve nucleus innervating the forehead receives input from both cerebral hemispheres. The portion of the VII cranial nerve nucleus innervating the mid and lower face does not have this dual cortical input.
 - Next, test taste. Ask the patient to identify a sample of sugar and salt and to identify which is which (note: this test is not usually performed).

VIII. Vestibulocochlear Nerve

- a. Function: special sensory = hearing and balance
- b. How to assess:
 - First, assess hearing by instructing the patient to close their eyes and to say "left" or "right" when a sound is heard in the respective ear. Vigorously rub your fingers together very near to, yet not touching, each ear and wait for the patient to respond. After this test, ask the patient if the sound was the same in both ears, or louder in a specific ear. If there is lateralization or a hearing abnormality, perform the Rinne and Weber tests using the tuning fork.
 - The Weber Test is a test for lateralization. Rap the tuning fork firmly on your palm and then press the butt of the instrument on the top of the patient's head in the midline and ask the patient where they hear the sound. Normally, the sound is heard in the center of the head or equally in both ears. If there is a conductive hearing loss present, the vibration will be louder on the side with the conductive hearing loss.
 - Balance can be tested by a heel-to-toe gait test along a straight line (with eyes closed).

IX & X. Glossopharyngeal and Vagus Nerves

- a. Function of IX: somatic motor = stylopharyngeus; visceral motor = parasympathetic innervation to parotid gland; visceral sensory = visceral sensation from parotid, carotid body, pharynx and middle ear; special sensory = taste posterior 1/3 of tongue; somatic sensory = cutaneous sensation from external ear Function of X: somatic motor = motor to muscles of pharynx (except stylopharyngeus), intrinsic muscles of larynx, muscles of palate (except tensor veli palatini) and striated muscle in superior 2/3 of esophagus; visceral motor = parasympathetic innervation to smooth muscle of trachea, bronchi, digestive tract and cardiac muscle of the heart; visceral sensory = visceral sensation from base of tongue, pharynx, larynx, trachea, bronchi, heart, esophagus, stomach and intestine to left colic flexure; special sensory = taste from epiglottis and palate; somatic sensory = sensation from auricle, external acoustic meatus and dura mater
- b. How to assess:
 - First, perform a swallowing test and voice test. Ask the patient to swallow and note any difficulty doing so. Ask the patient if they have difficulty swallowing. Next, note the quality and sound of the patient's voice. Is it hoarse or nasal? Ask the patient to open their mouth wide, protrude their tongue, and say "AHH". While the patient is performing this task, flash your penlight into the patient's mouth and observe the soft palate, uvula and pharynx. The soft palate should rise symmetrically, the uvula should remain midline and the pharynx should constrict medially like a curtain. Often the palate is not visualized well during this maneuver. One may also try telling the patient to yawn, which often provides a greater view of the elevated palate.
 - Do not perform the gag reflex test on each other. Simply read the instructions on how to perform this test. Use a tongue depressor and touch the pharynx with a Q-tip on both the left and then on the right side, observing the normal gag or cough. Some clinicians omit testing for the gag reflex unless there is dysarthria or dysphagia present by history or examination, or if cranial nerve palsies are present at the medullary level. Note: Roughly 20% of normal individuals have a minimal or absent gag reflex.

XI. Spinal Accessory Nerve

- a) Function: somatic motor = innervation to sternocleidomastoid and trapezius
- b) How to assess:
 - Ask the patient to shrug their shoulders as strongly as they possibly can while the examiner resists this motion by pressing down on the patient's shoulders with their hands. Next, ask the patient to turn their head to the side as strongly as they possibly can while the examiner once again resists with their hand. Repeat this maneuver on the opposite side. The patient should normally overcome the resistance applied by the examiner. Note any asymmetry.

XII. Hypoglossal Nerve

- a) Function: somatic motor = intrinsic and extrinsic muscles of tongue (except palatoglossus)
- b) How to assess:
 - The hypoglossal nerve controls the intrinsic musculature of the tongue and is evaluated by having the patient "stick out their tongue" and move it side to side. Normally, the tongue will be protruded from the mouth and remain midline. Note deviations of the tongue from midline, a complete lack of ability to protrude the tongue, tongue atrophy and fasciculations on the tongue. The tongue will deviate towards the side of a peripheral lesion, and to the opposite side of a central lesion.

CONGRATULATIONS! You have tested the cranial nerves.

PART B: Cranial Nerve Location

Identify the location of the cranial nerves on the ventral and dorsal surface of the brainstem. List both the number and the full name in the key.

VENTRAL BRAINSTEM

DORSAL BRAINSTEM

A.

G.

B.

H.

C.

I.

D.

J.

E.

K.

F.

PART C: Cranial Nerve Reflexes

Review the components of the cranial nerve reflexes by filling out the chart below.

REFLEX	AFFERENT	EFFERENT
PUPILLARY LIGHT REFLEX		
JAW JERK REFLEX		
CORNEAL REFLEX		
GAG REFLEX		

PART D: Cranial Nerve Nuclei:

Color in the location of the cranial nerve nuclei on each brainstem cross-section.

UNIT IV:

SPECIAL SENSES & BRAINSTEM INTEGRATION

Lecture 25: Overview of Special Senses

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

General Principles of Sensory Systems (Ch. 28): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/general-principles-of-sensory-systems/>

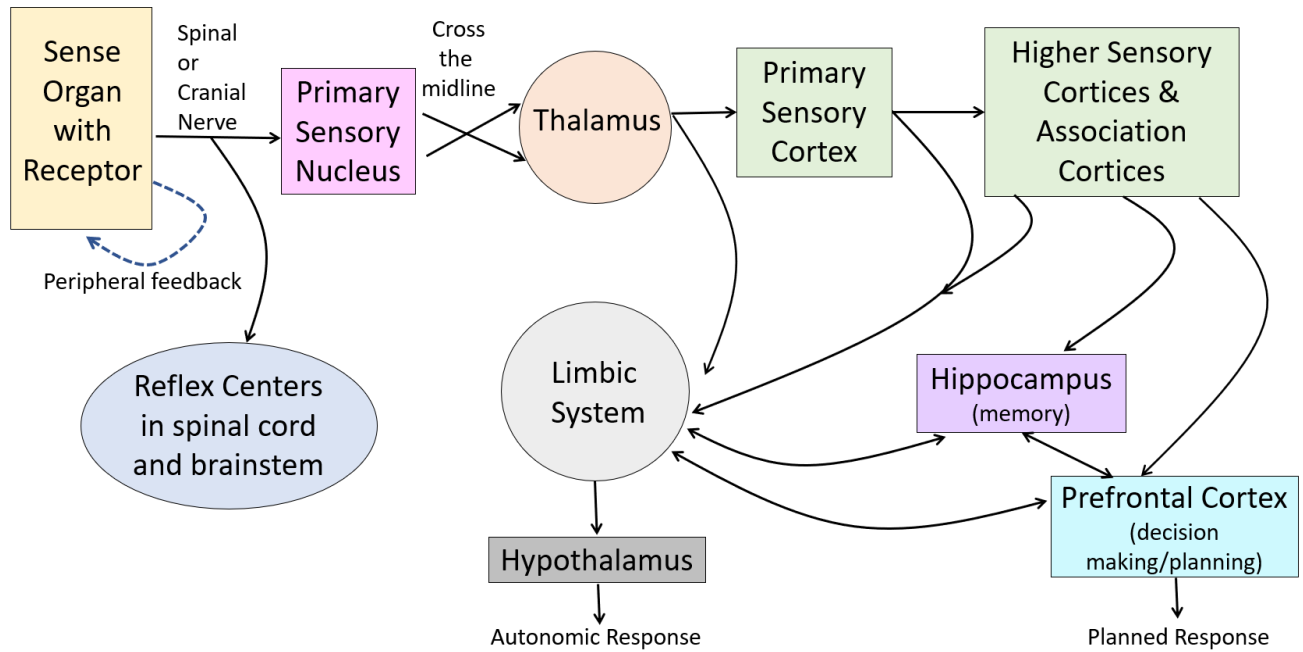
Learning Objectives

1. Outline the general organization of all sensory systems into a table that outlines tissue, receptor, cranial nerve/nuclei, thalamic subnucleus, and cortical targets.
2. Define the terms receptive field, adaptation, cortical topography, and hierarchical organization as related to the special senses.
3. Define the terms: receptor potential vs. action potential and short receptors vs. long receptors.
4. Differentiate central vs. peripheral deficits of special senses.
5. Differentiate the terms reception, sensation, perception, identification, and interpretation according to the organization of sensory pathways.
6. List three strategies the visual system uses to deal with the enormous range of light intensities.

Terms to Know

- **Sensory system terminology**
 - **Reception** = occurs at the sensory receptors (it is the transduction or conversion of environmental energy into a voltage response)
 - **Sensation** = action potentials firing through the sensory pathway
 - **Perception** = occurs in the sensory cortices
 - **Identification** = occurs in the sensory cortices (there are hierarchical centers in higher cortical areas specialized for specific types of identification, i.e., the fusiform face area identifies faces for the visual system)
 - **Interpretation** = emotional tagging or relating the sensory stimulus to past experiences or knowledge, involves the limbic system
 - **Decision Making** = prefrontal cortex and hypothalamus (if autonomic response)
 - **Motor response** = motor pathways
- **Types of Potentials**
 - **Receptor Potential** = sub-threshold graded voltage response that occurs in a sensory receptor. The stimulus intensity is encoded by the amplitude of the voltage response.
 - **Action Potential** = above threshold all-or-none voltage response that occurs in the cranial nerve attached to the sensory receptor
- **Anatomical categories of receptors**
 - **Short receptor** = small, specialized receptor cells that cannot generate an action potential, but instead generate receptor potentials (i.e. Photoreceptors, hair cells, taste receptor cells)
 - **Long receptors** = receptor areas of neurons that are attached directly to the long neuron, such as the free nerve endings (nociceptors) connected to pseudounipolar neurons of the somatosensory system. These can travel long distances and can generate action potentials. In long receptors, the receptor potential occurs at the free nerve ending of the nociceptor; then the action potential is generated in the axon.
- **Deficits**
 - **Central deficit** – occurs in the CNS
 - **Peripheral deficit** – occurs in the PNS

General pattern for sensory system organization



System	Sense Organ (& specialized tissue)	Receptor cell type	Cranial Nerve	Cranial Nerve NUCLEUS	Subnucleus within the Thalamus	Primary Sensory Cortex Location (& in what lobe)	Brod-mann Area
Somatosensory System for the FACE (e.g. Touch & Pain)							
Visual System (seeing)							
Auditory System (hearing) & Vestibular System (balance)							
Gustatory System (Taste)							
Olfactory System (Smell)					(olfaction doesn't go to thalamus)		

Lecture 26: The Visual System

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Visual System: The Eye (Ch. 29): <https://openbooks.lib.msu.edu/introneuroscience1/?p=159>

Visual System: Central Processing (Ch. 30): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/vision-central-processing/>

Circadian Rhythms (Ch. 57): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/circadian-rhythms/>

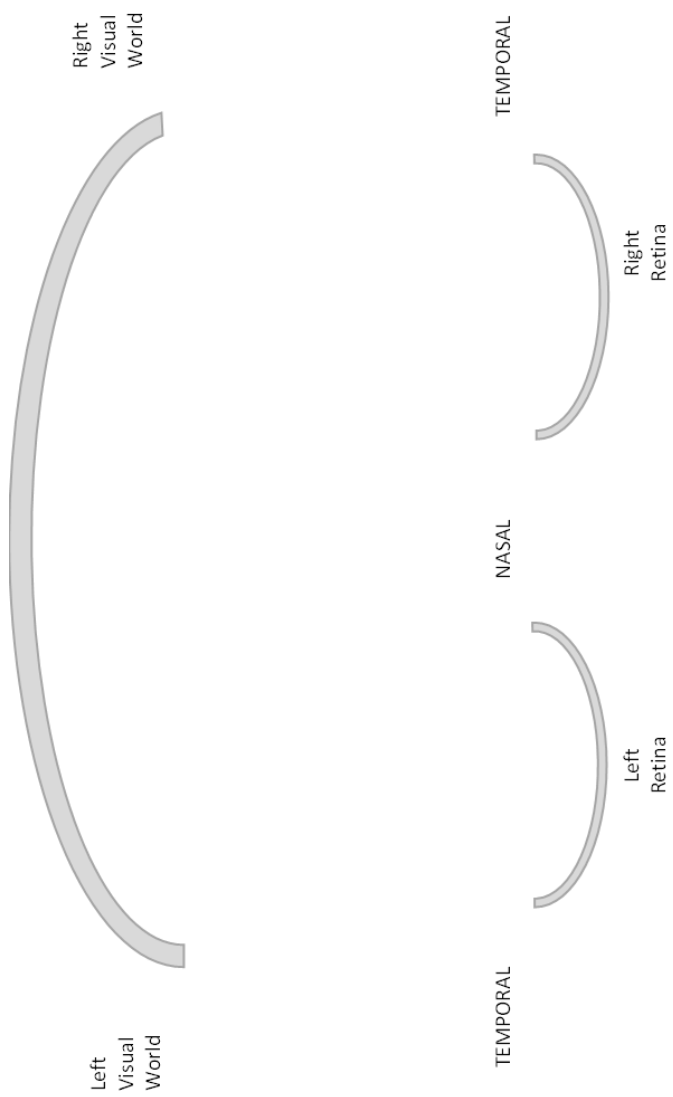
Learning Objectives

1. Describe the anatomy of the eye, including cornea, pupil, iris, lens, sclera, and retina.
2. Define myopia and hyperopia.
3. Draw and label the circuitry of the retina.
4. Compare and contrast rods vs. cones.
5. Discuss the major steps in phototransduction and the result on photoreceptor polarity.
6. Describe the neurobiological basis for trichromatic vision and deficits in this system.
7. Explain the role of ipRGCs and the suprachiasmatic nucleus of the hypothalamus in the regulation of circadian rhythms.
8. Diagram the visual pathway from the retina to the brain, including both cortical targets (V1-V5) and subcortical targets (SCN of hypothalamus, superior colliculus, and Edinger-Westphal nucleus).
9. Describe the visual deficits that occur from lesions at different locations in the visual pathway.

Terms to Know

- Circuitry of Retina
 - Photoreceptors (rods, cones, and melanopsin ganglion cells)
 - Horizontal cells
 - Bipolar cells
 - Amacrine cells
 - Retinal ganglion cells (RGCs)
- Phototransduction
 - Opsin
 - Rhodopsin
 - Cone opsin
 - 11-cis retinal
 - Photoisomerization (cis to trans)
 - G-protein coupled cascade
 - Amplification
 - Dark current
 - Receptor potential (graded)
- Trichromatic vision
 - S-opsin, M-opsin, L-opsin
 - Color vision is accomplished by comparing outputs from the 3 cone types
- Image forming visual pathway

- Optic nerve
- Optic chiasm
- Optic tract
- Lateral geniculate nucleus of the thalamus (LGN)
- Primary visual cortex (BA17; V1; Striate cortex)
- Extra-striate cortices (you do not have to identify these on a brain)
 - V2 = relay signals
 - V3 = debated function
 - V4 = color discrimination
 - V5 = MT (middle temporal area) = motion discrimination
 - IT (inferior temporal gyrus) = visual object (shape) recognition
 - FFA (fusiform face area) = face recognition
 - PPA (parahippocampal place area) = appearance and layout of scenes
- “What” pathway (temporal lobe) vs. “Where” pathway (parietal lobe)
- Subcortical nuclei involved in non-image-forming vision
 - Superior colliculus (visual reflexes)
 - Suprachiasmatic nucleus of the hypothalamus (circadian photoentrainment)
 - Pretectum and Edinger-Westphal nucleus (pupillary light reflex)



Lecture 27-28: The Chemical Senses – Olfactory and Gustatory Systems

Tom Finger, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Olfactory System (Ch. 35): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/olfactory-system/>

Gustatory System (Ch. 34): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/taste/>

Learning Objectives

1. Describe the difference between the terms smell, taste and flavor as properly used in a biomedical context.
2. Review the anatomy and identify the approximate position of olfactory sensory epithelium within the nasal cavity with emphasis on the position of the olfactory sensory neurons (receptor cells).
3. Describe the difference in odorant routes to the olfactory epithelium during orthonasal and retronasal stimulation.
4. Trace the course of the olfactory nerve (cranial nerve 1) from its origin in the olfactory epithelium to its site of termination in the olfactory bulb and describe the way odor information is transmitted from the receptor epithelium to the olfactory bulb.
5. Describe how odors are transduced into a neural signal by the olfactory sensory neurons and discuss how different odors are represented within the receptor sheet and within the bulb.
6. Trace the pathway from the olfactory epithelium to the several olfactory centers in the basal forebrain, and what type of information is extracted in each of these target areas.
7. List the five basic tastes and for each indicate whether transduction is via an ion channel or GPCR and what taste bud cell type is involved in their detection.
8. List the 3 types of lingual taste papillae and indicate which cranial nerve provides gustatory innervation to each of them. Also, list which cranial nerve carries touch and temperature.
9. Describe the different ways neurotransmitters are released from different types of taste cells and identify the key neurotransmitter in this system.
10. Trace the gustatory pathways in the brain, starting with incoming information on the taste nerves.
11. Describe how pain-type nerve endings are implicated in the detection and transmission of information from chemesthetic receptors in the oral cavity.

Terms to Know

- **Introduction**
 - Role of chemical senses in behavior
 - Chemical senses of humans
 - Smell
 - Taste
 - Chemesthesis: trigeminal chemoreception
 - Clinical complaints and how to distinguish between the chemical senses
 - Comparison of chemical senses
 - Receptor cell and sensory end organs
 - Cranial nerve
 - Primary sensory nucleus in the brain
 - Sensations and utilization

- **Olfaction**
 - Peripheral anatomy
 - Orthonasal vs retronasal stimulation
 - Olfactory epithelium
 - A mucus layer and cilia
 - Olfactory receptor neurons
 - Olfactory transduction
 - G-protein coupled receptors
 - Chloride channels
 - Olfactory bulb and representation of odors
 - Glomeruli: chemical map
 - Output cells: Mitral cells
 - Central olfactory pathways
 - Olfactory tract as tract of CNS
 - Piriform cortex, orbitofrontal cortex (conscious appreciation of odor)
 - Entorhinal cortex, hippocampus (odor-evoked memories)
 - Olfactory tubercle and amygdala (limbic/visceral responses to odors)
- **Taste**
 - Taste qualities (bitter, sweet, salty, sour, “umami” [savory])
 - Peripheral taste apparatus
 - Distribution of Taste Buds: lingual vs extra-lingual
 - Lingual papillae
 - Organization of taste buds
 - Diversity of taste transduction mechanisms
 - Ion channels (sour, salty)
 - Second-messenger systems (bitter, sweet, umami)
 - Central Taste Pathways
 - Nucleus of the solitary tract – orotopic map
 - VPM: most medial part
 - Taste cortex (in insular cortex)
 - “flavor” cortex = orbitofrontal cortex
- **Chemesthesis: Trigeminal chemoreception**
 - Free Nerve endings
 - TRP Channels (chemically-sensitive ion channels)

Lecture 29: The Vestibular System

John Caldwell, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Vestibular System (Ch. 33): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/vestibular-system/>

Learning Objectives

1. Identify the environmental stimulus involved in the vestibular system (balance).
2. Identify the external anatomy of the vestibular system, including the sensory receptor organ for the vestibular system.
3. Describe the process of peripheral transduction of vestibular sensory signals.
4. Diagram the vestibular pathway, from the vestibular nerve to central vestibular areas.
5. Identify the vestibular nucleus on a brainstem cross-section and identify its inputs and outputs.
6. Describe the neurocircuitry underlying the vestibulo-ocular reflex (VOR).

Terms to Know

- Vestibular environmental stimulus:
 - Angular acceleration
 - Pitch
 - Yaw
 - Roll
 - Linear acceleration (horizontal and vertical direction and amplitude)
- Vestibular anatomy:
 - Semicircular canals
 - Utricle
 - Saccule
 - Ampulla
 - Cupula
 - Otoliths
- Peripheral transduction of vestibular sensory signals and the vestibular pathway
 - Pathway of first order neuron:
 - Receptors: vestibular receptors called vestibular hair cells are located in the utricle, saccule, and ampulla of the inner ear. Transduction of vestibular signals is via bending of stereocilia on vestibular hair cells. Movement toward the tallest stereocilia (kinocilium) opens K⁺ channels and causes depolarization of hair cells and releases neurotransmitters onto afferent nerve fibers. Movement away from the kinocilium causes the closing of K⁺ channels and a hyperpolarization.
 - Somas: vestibular sensory neurons are bipolar. Cell bodies are found in the vestibular (Scarpa's) ganglion.
 - Axons: axons leaving the inner ear form the vestibular branch of VIII. Leaving the ganglion, axons synapse into one of four vestibular nuclei (superior, inferior, medial, and lateral) or the cerebellum.
 - Pathway of the second-order neuron descending:
 - Somas: located in the medial and lateral vestibular nuclei
 - Axons: leaving the medial and lateral vestibular nuclei terminate in the spinal cord. Involved in balance and posture.

- Pathway of second order neuron ascending:
 - Somas: located in vestibular nuclei
 - Axons: synapse in thalamus near ventral posteromedial nucleus
- Vestibulo-ocular reflex (VOR)
 - There are corresponding head-eye compensations for all directions of head rotation. We will consider what happens during rotation of the head toward the right as an illustration (fig. 1). The reflex described here is driven by vestibular input and can occur in the dark, in the absence of any visual input at all. Rightward head rotation is signaled by horizontal canals; leftward eye rotation is produced by excitation of motor neurons to the left lateral rectus in the left abducens nucleus and excitation of right medial rectus by motor neurons in the right oculomotor nucleus. (There is also inhibition of antagonistic muscles, but these will not be described.)
 - As the head rotates to the right, fluid in the horizontal semicircular canal lags, resulting in deflection of the cupula in both horizontal canals. In the right horizontal canal, the deflection results in depolarization of the hair cells, in the left horizontal canal, the hair cells are hyperpolarized (opposite effects always occur in two paired canals). Excitatory input on the right is transmitted to the right vestibular nuclei. Cells in the vestibular nuclei project by way of the **medial longitudinal fasciculus (MLF)** to excite left lateral rectus motor neurons in the abducens nucleus.
 - What about excitation of the right medial rectus motor neurons? Since the desired end result is to have left lateral rectus and right medial rectus contracting in synchrony, there must be some mechanism to ensure such coordination. This is achieved by having co-activation of abducens motor neurons and internuclear interneurons (about equal numbers of these in the abducens nucleus) whose axons cross over and ascend in the **medial longitudinal fasciculus** to excite right medial rectus motor neurons. Any stimuli that excite abducens motor neurons always also excite these internuclear neurons, thus ensuring co-activation of lateral rectus and contralateral medial rectus. The internuclear neurons serve as "pattern generators" for conjugate horizontal gaze. The axons from these interneurons cross the midline at the level of the abducens nucleus and thus, damage to the MLF on the right side between VI and III damages axons coming from the left abducens nucleus. This damage results in the condition known as Internuclear Ophthalmoplegia (INO).
 - Extra information (**don't need to know**). Any exam questions will be restricted to horizontal eye movements. Conjugate vertical movements are somewhat simpler. The oculomotor nuclei contain motor neurons for the superior and inferior recti, as well as medial rectus and inferior oblique. All one needs to do is simultaneously activate superior motor neurons and inhibit inferior motor neurons for both eyes. So the "pattern generator" for this behavior is relatively simple and doesn't involve long pathways. The interneurons that constitute the pattern generator for upward gaze are located near the superior colliculi and posterior commissure and cells are distributed bilaterally. Timing signals for downward gaze come from deeper in the midbrain, near the dorsomedial edge of the red nucleus, and again are bilaterally distributed.

Lecture 30: The Auditory System

Tom Finger, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Auditory System: The Ear (Ch. 31): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/auditory-system/>

Learning Objectives

1. Identify the environmental stimulus involved in audition (hearing).
2. Review the anatomy of the auditory parts of the ear: external, middle, and inner ear, including the sensory receptor organ.
3. Describe the process of peripheral transduction of auditory signals, starting with pressure waves impacting the tympanic membrane and ending with a signal going to the brain.
4. Diagram the central auditory pathway, from the auditory nerve to central auditory areas, identify the components of this pathway on brainstem cross-sections, and describe where in the system the direction of sound is computed and what features of the stimulus are important for this computation.
5. Describe how the auditory system localizes sound around you – including mechanisms and central nuclei involved.

Terms to Know

- Auditory Environmental Stimulus:
 - Amplitude
 - Frequency
- Auditory Anatomy: (review from gross anatomy)
 - Pinna/Auricle
 - Tympanic membrane
 - Tensor tympani muscle and stapedius muscle
 - Oval window
 - Round window
 - Ossicles
 - Cochlea & Organ of Corti
- Peripheral transduction of Auditory Sensory Signals
 - Receptor cells: Auditory receptor cells, called inner hair cells, are short (axonless) receptor cells located in the Organ of Corti within the cochlea. Transduction of auditory signals is via bending of stereocilia on the apical part of inner hair cells. This causes depolarization of hair cells, releasing neurotransmitters onto the sensory terminals (dendrites) of the afferent nerve fibers within the Organ of Corti. Outer hair cells are motile and serve to amplify the vibrations of the basilar membrane. In situations where hair cells die (e.g., getting old or various diseases), a cochlear implant can restore many aspects of hearing by directly stimulating the sensory endings of the nerve, thereby bypassing the hair cells.
 - First-order neurons: Sensory nerve endings in the cochlea arise from bipolar auditory ganglion neurons situated in the spiral ganglion of the cochlea.
 - Axons: axons leaving the spiral ganglion form the cochlear branch of CN VIII (Vestibulocochlear nerve). Upon entering the rostral medulla, these axons synapse in a tonotopic map (high-low frequency sounds) in the ipsilateral cochlear nucleus. This tonotopic organization is maintained throughout all central auditory nuclei.

- Central Auditory Pathways
 - Somas: Located in the cochlear nucleus
 - Axons: Most axons that leave the cochlear nerve nucleus decussate to cross the midline in the trapezoid body, but many do not cross the midline. These outputs from the cochlear nucleus terminate in both the ipsilateral and contralateral superior olivary complex (SOC, including the SO nucleus), which computes a map of auditory space by comparing the timing and amplitude of signals coming from the 2 ears.
 - The axons leaving the SOC project to the following while maintaining a tonotopic organization: SOC -> lateral lemniscus (LL) -> Inferior colliculus (IC) -> Medial geniculate body (MGB; Thalamus) -> auditory cortex (acoustic area of temporal lobe cortex); Brodmann areas 41 and 42
 - Note that the cochlear nucleus processes auditory information from only the ipsilateral ear, whereas all auditory information after that is processed bilaterally. Hence, if hearing is lost in only one ear, the damage must be at the level of the ear, the nerve, or the cochlear nucleus. Damage to structures beyond the cochlear nucleus results in decreased hearing ability in both ears, with the loss being more pronounced in the contralateral ear, e.g. if the auditory cortex on the R side is damaged, hearing will be decreased more in the L ear than the R ear, but both sides will be affected.

Lecture 31-32: Brainstem Pathways & Reflexes (AKA Cranial Nerve Pathways)

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Learning Objectives

1. List the cranial nerves that have a motor component and identify how they get their motor command from the brain (draw the corticobulbar pathway). Be sure to indicate which of these CN motor nuclei get bilateral vs. contralateral innervation from the corticobulbar tract.
2. Draw all three pathways of the trigeminal system.
3. Compare upper motor neuron lesion vs. lower motor neuron lesion in CN VII and the clinical significance of this anatomy to facial palsies (include a definition of Bell's palsy in your own explanation).
4. Name the two nuclei that control salivation, as well as their associated cranial nerves and salivary glands.
5. Describe the clinical deficits that can be observed in a lesion to the hypoglossal nucleus.
6. Compare and contrast the function of the vagal nuclei (dorsal motor nucleus of X, nucleus ambiguus and solitary nucleus).

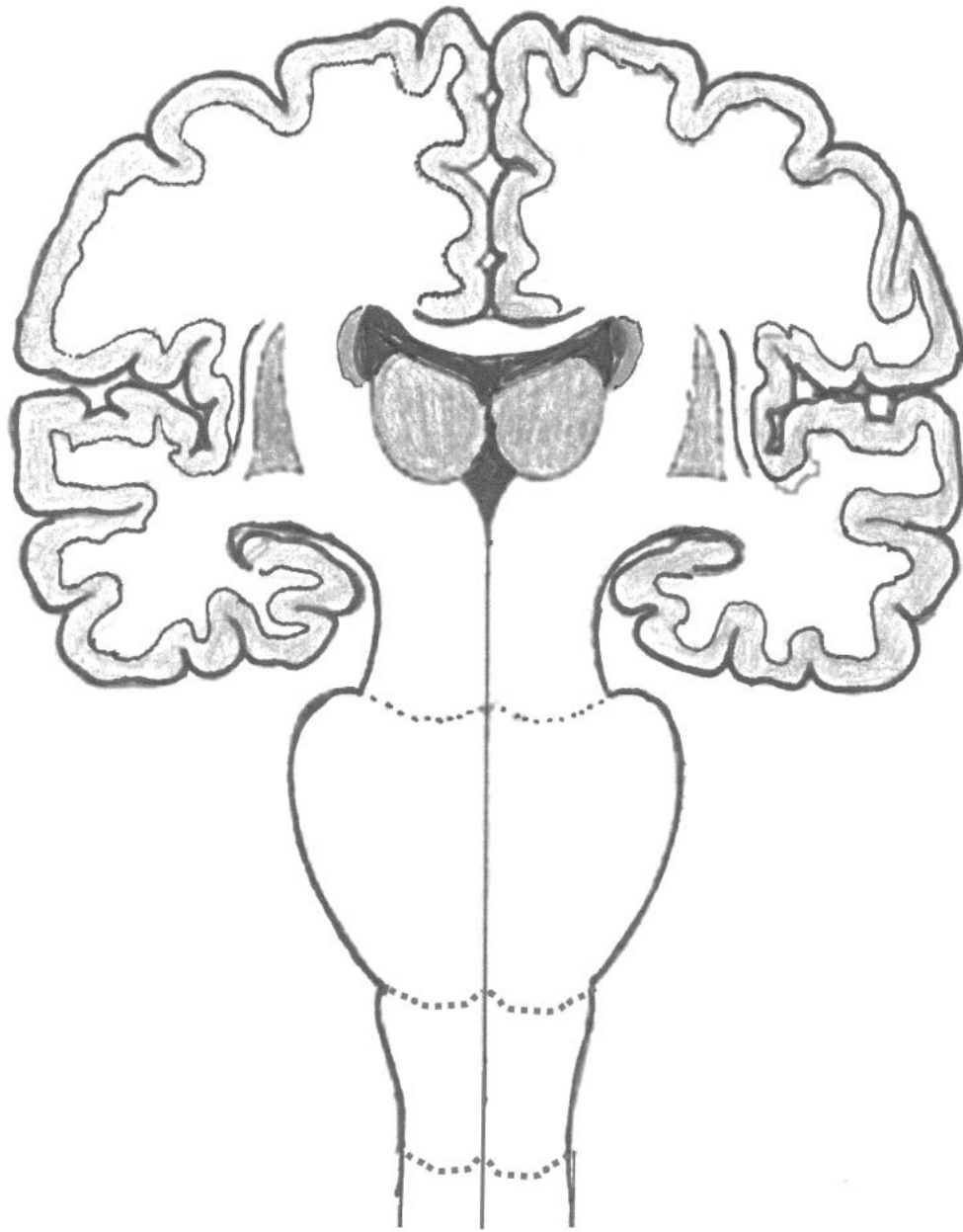
Terms to Know

- Pathways
 - Trigeminal Pathway
 - Trigeminal Fine Touch Pathway
 - Trigeminal Pain Pathway
 - Trigeminal Motor Pathway (part of corticobulbar)
 - Corticobulbar Pathway

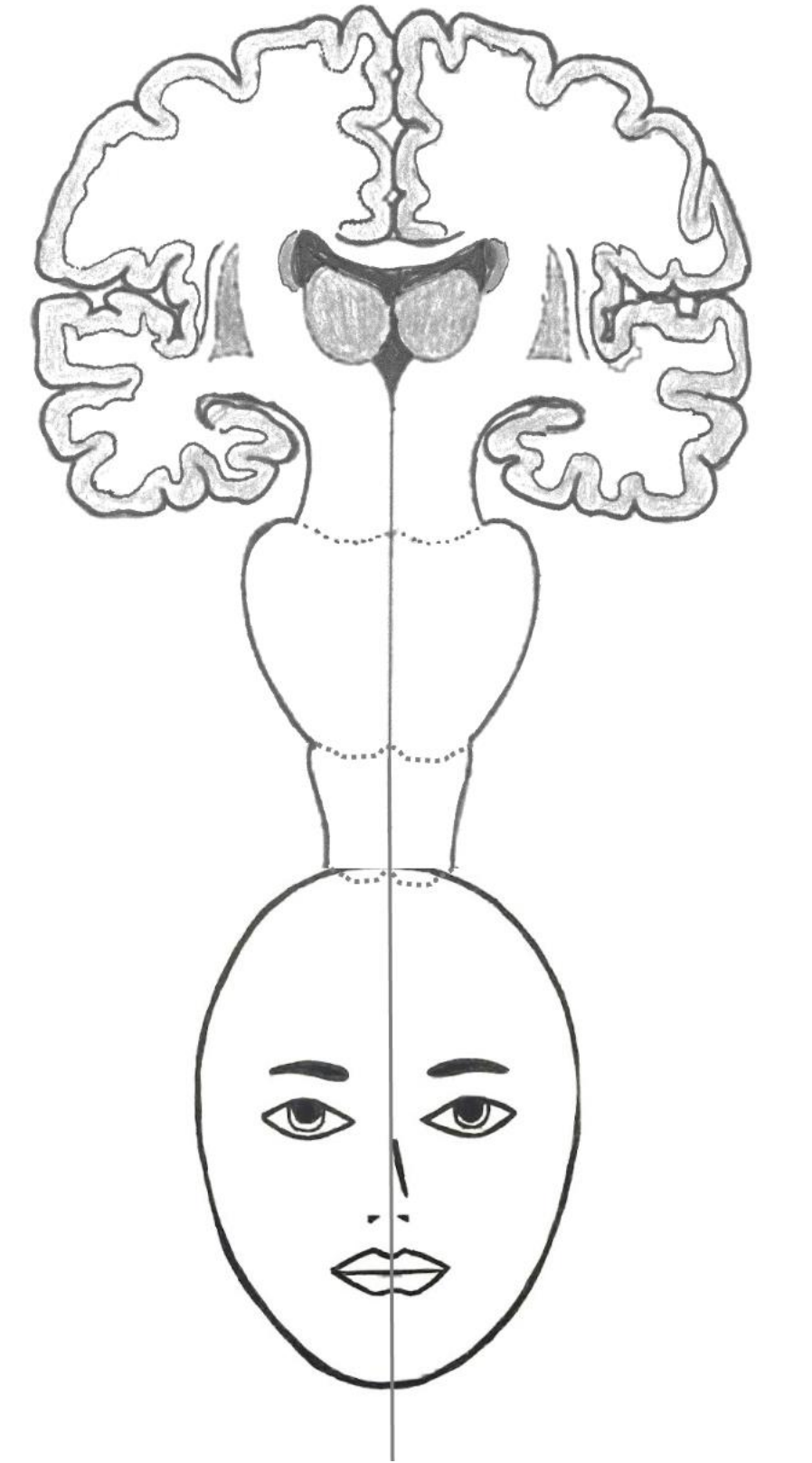
- Provides bilateral innervation to all motor nuclei except the lower division of facial motor nucleus
 - Note that while innervation to the hypoglossal nucleus is bilateral, there is contralateral dominance. Thus, UMN lesions to hypoglossal pathway will lead to weakness but not paralysis.
 - Provides contralateral innervation to the lower division of the facial motor nucleus
- Medial Longitudinal Fasciculus
 - CN III, IV, VI and VIII (vestibular only)
 - Integrating head movements and eye movements
- Vagal Nuclei
 - Dorsal motor nucleus of X = Preganglionic parasympathetic X to heart, thoracic and abdominal viscera
 - Nucleus ambiguus = LMN from X to soft palate, pharynx, larynx (important for swallowing, phonation, movement of uvula and gagging). This nucleus is shared and contains motor fibers of glossopharyngeal (IX) to the stylopharyngeus (helps in swallowing and phonation)
 - Solitary nucleus = receives visceral and special sensory fibers (taste) from VII, IX, and X

Corticobulbar Pathway

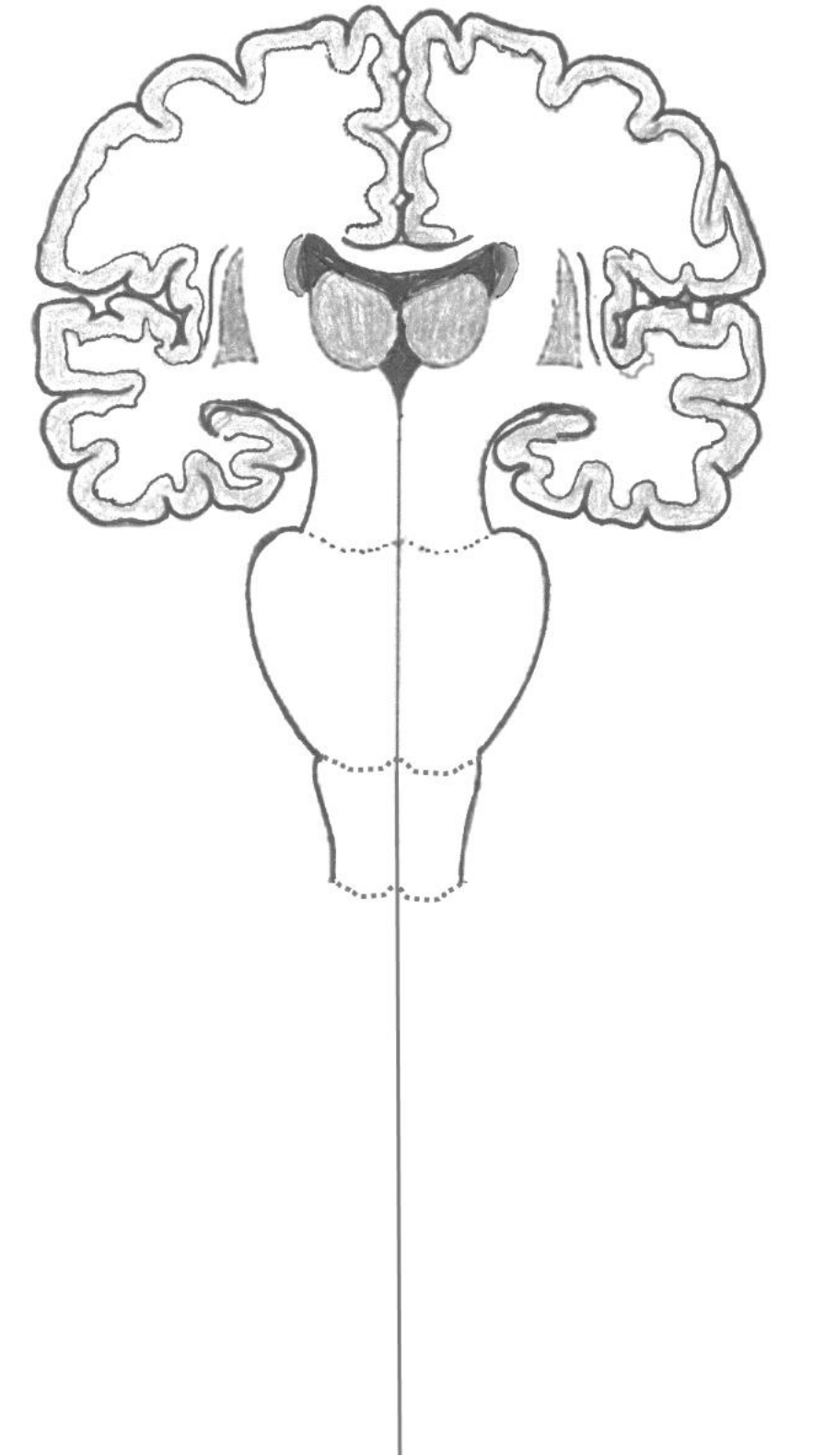
Corticobulbar pathway to XII (Hypoglossal)



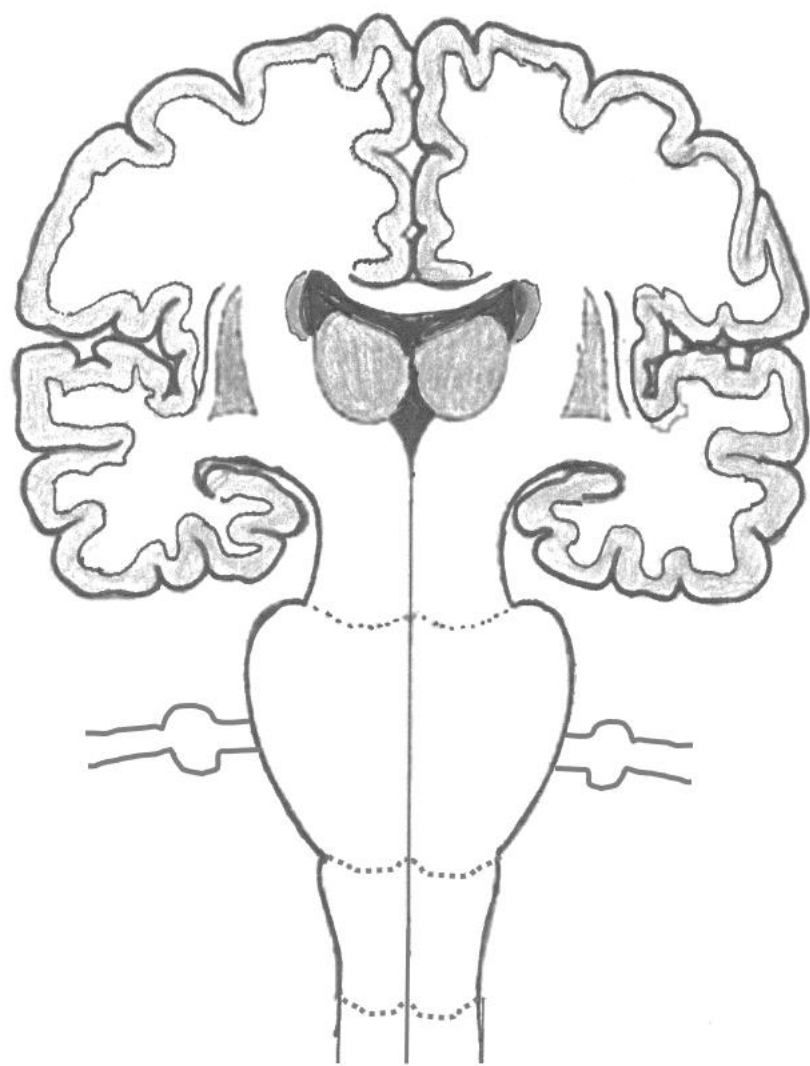
Corticobulbar Pathway to VII (Facial)



Corticobulbar Pathway to V3 (Trigeminal Motor Pathway)

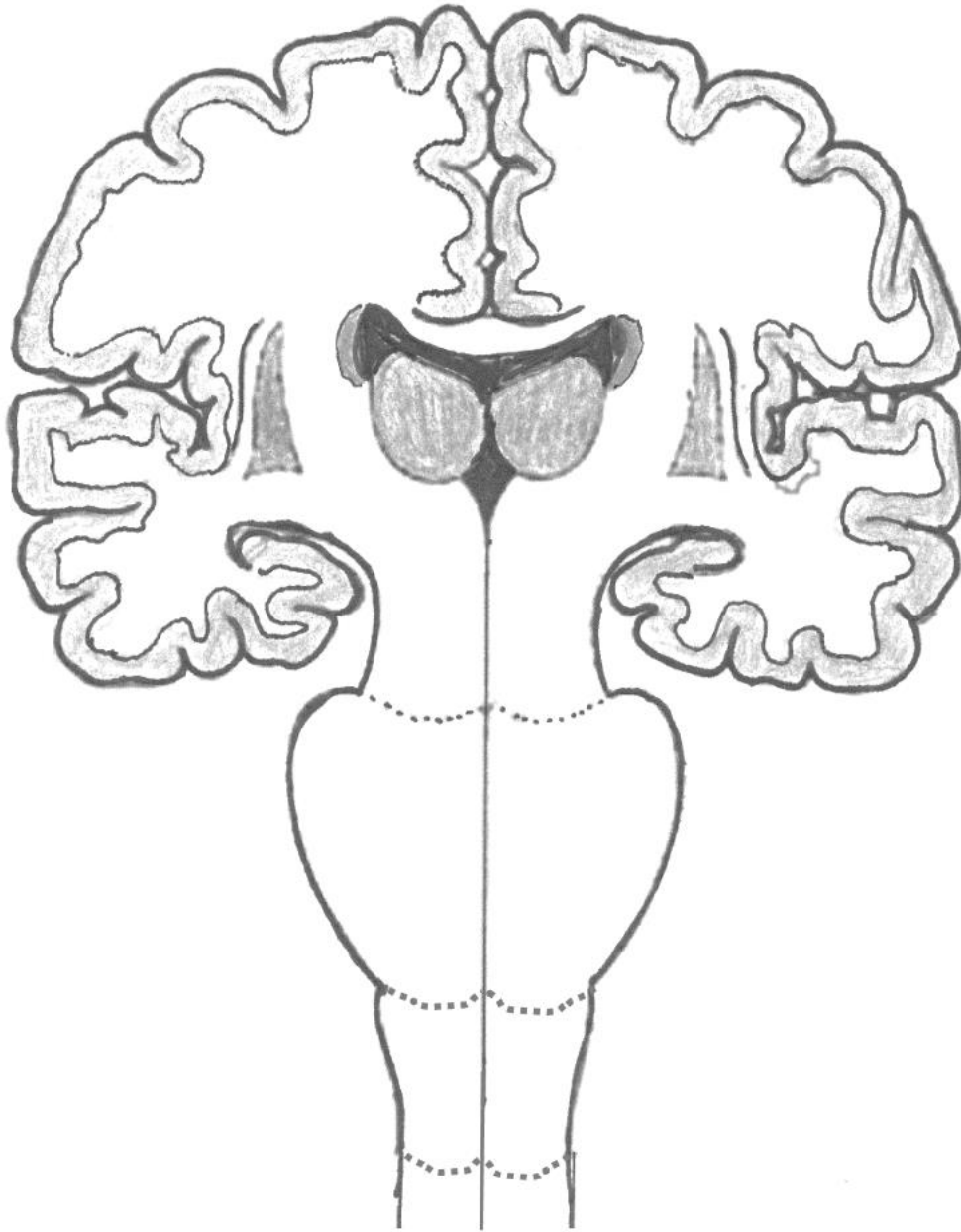


Trigeminal Sensory Pathways

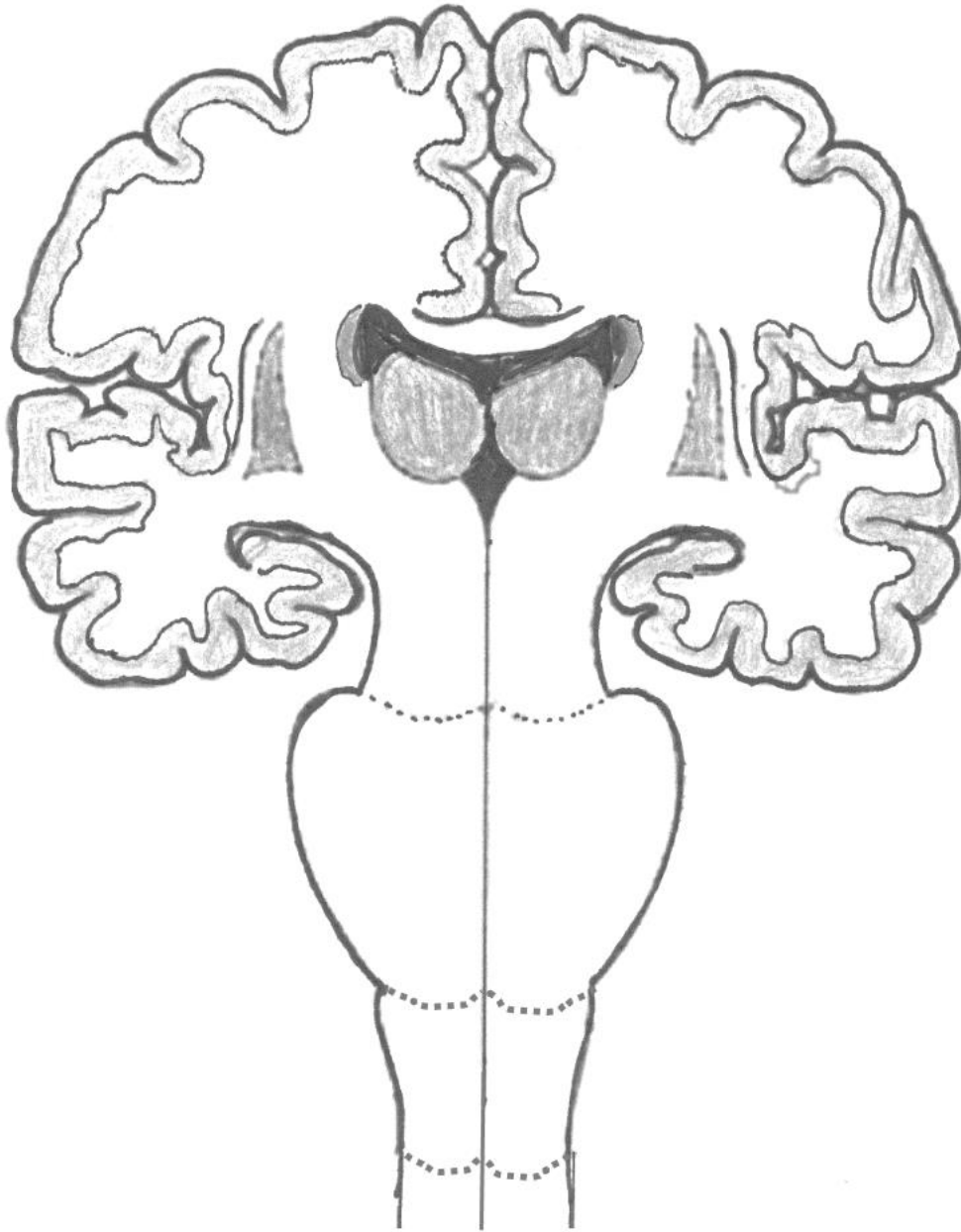


	BODY	FACE
Motor Command to Skeletal Muscles		
Fine Touch, <u>Pressure, Vibration</u>		
Proprioception		
Pain & Temp		

Corticobulbar Pathway to III, IV, VI (Extraocular Muscles)



MLF Pathway



LAB 7: Special Senses Lab

Cory Gritton, PhD, & MHA Teaching Team

Learning Objectives

1. Recall the pathways for the visual, auditory, vestibular, gustatory and olfactory systems.
2. Practice ultrasound scanning skills.
3. Recognize the anatomical structures of the eye with ultrasound.
4. Solve clinical cases related to the special senses and brainstem.

Instructions

We will break into groups of 6 students each and rotate through the stations, spending approximately 20-25 minutes at each station.

- STATION A: Ultrasound of the eye (Dr. Gritton, Muazzam Ramziddin, Sloan Fridrich)
- STATION B: Visual System Pathways and Lesions (Dr. Stabio)
- STATION C: Chemical Senses (Erika Alor)
- STATION D: Auditory & Vestibular Systems (Dr. Caldwell)

Station A: Ultrasound of the Eye

Instructions: Follow the steps and answer the review questions below using yourself and your US partners as human models. You and your US partners should take turns using the probe and compare your answers. Facilitators are here to assist you, but this is meant to be a hands-on experience.

****No contact lenses if you wish to scan your eye** Wear appropriate clothing and accessories to facilitate live peer scanning of the eye.**

Attempt to identify all of the structures below, one at a time. Practice applying gel, holding the probe, and moving the probe to visualize each structure. Position the structures in the center of the screen. Identify the right and left side of the patient on the on-screen image.

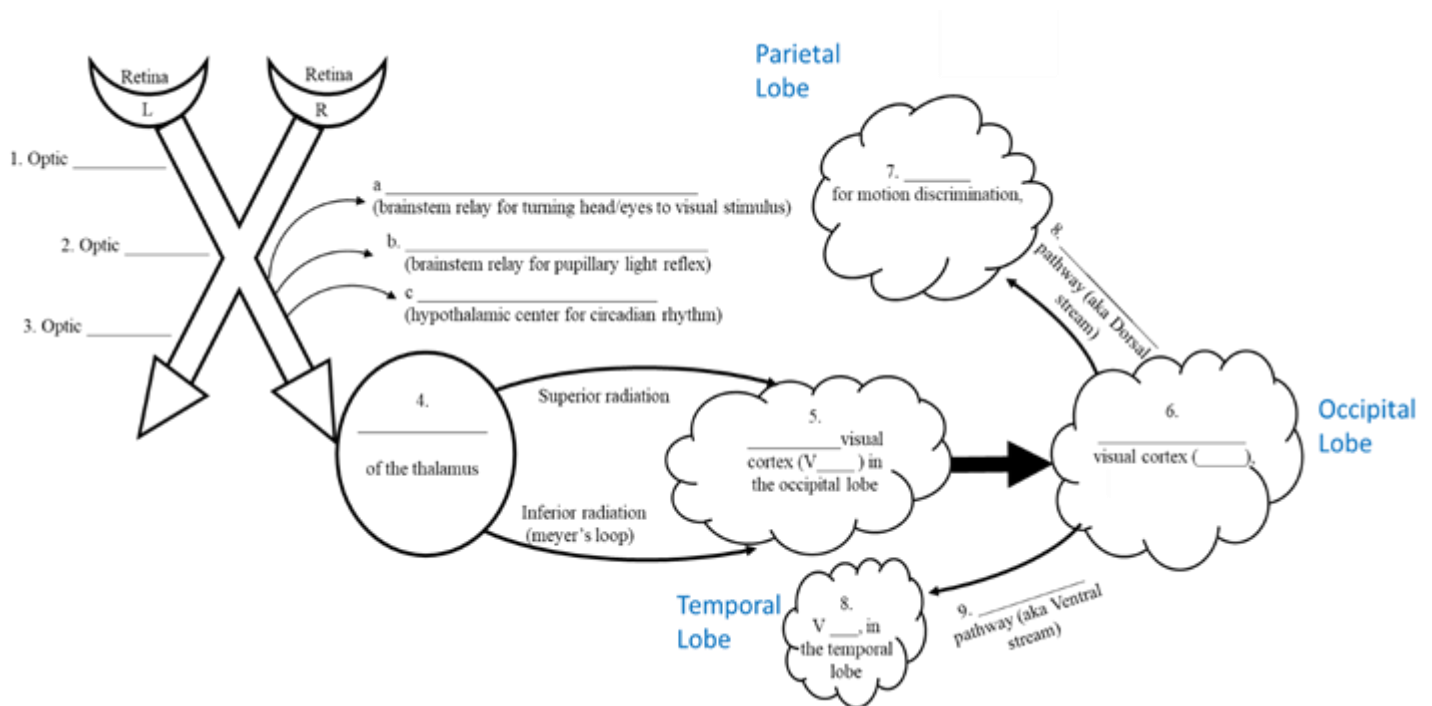
Reminder: As a precaution, we will not be scanning the eyes of volunteers with contact lenses currently in place.

1. Place a generous helping of ultrasound gel on the closed eyelids. Stand or sit at the head of the model and gently place the probe over the closed eye in the short axis view (Figure below).
2. Recall the path of light through the structures of the eye from superficial to deep.
3. Identify the following structures: Cornea, Iris, Pupil, Lens, Anterior chamber, Posterior chamber, Vitreous Chamber, Retina, Optic nerve
4. What do you expect a lens dislocation to look like? What about a retinal detachment? A vitreous hemorrhage?

5. Measure the width of the optic nerve. _____ **mm** (Adult standard diameter is 4.9-5.8mm) What is the clinical importance of this measurement?
(Remember, 1 view is NO view. Clinically, measurements must be taken in at least 2 axes)
6. List the layers of the optic nerve sheath from superficial to deep.
7. With the probe still positioned to ultrasound one of the model's eyes, perform a pupillary light reflex using a flashlight or phone. With the probe positioned over one closed eye, move the light over the model's other, open eye.
What muscle is responsible for the action that you observe? _____
What cranial nerve receives the afferent input in this reflex? _____
8. What cranial nerve elicits the efferent response in this reflex? _____
What muscle performs the opposing response? _____
Is this muscle action controlled by sympathetic or parasympathetic fibers? _____
9. Have the model look toward their right, then have the model look towards their left by moving their eyes only. Identify the cranial nerves that control extraocular muscles and what actions they elicit.

STATION B: The Visual Pathway

First, complete the visual system pathway schematic below. *Note: only one side of this bilateral pathway within the cortex is shown in the block diagram below.* Next, shade in black the area of the visual field that would demonstrate loss of vision from a lesion to each part of the pathway.

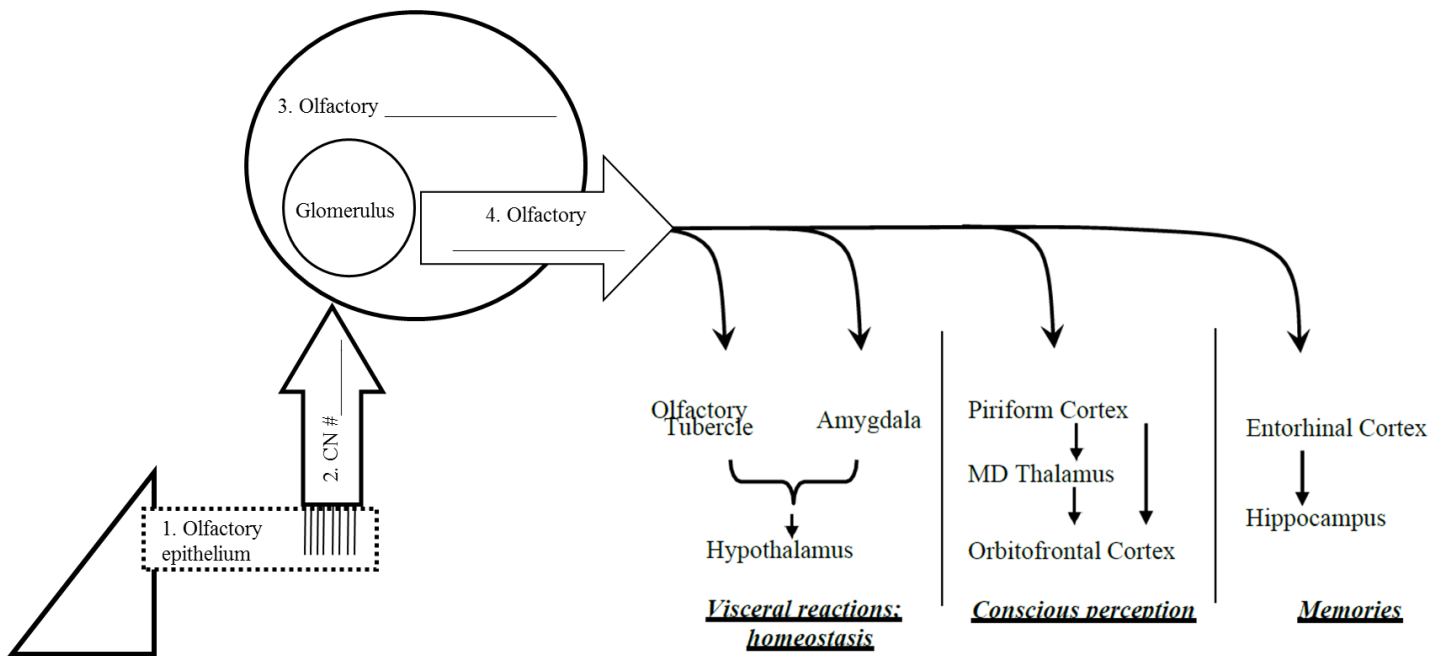


Station C: Chemical Senses

Gustatory Pathway

Gustatory pathways are bilateral above the medulla (but heavier ipsi than contra).

The Olfactory Pathway



Next, solve and describe the following case: A 75-year-old man presents to his Primary Care Provider complaining of loss of appetite and an unintentional weight loss. This started a few months ago after falling off his bicycle and hitting his head on the street. Although he was wearing a helmet (good for him), he seemed dazed and was evaluated for a concussion, which was negative. He denies any current nasal congestion, cough, or sinus pressure. He denies nausea, vomiting, difficulty swallowing or changes to bowel habits. “I am trying to eat enough, but I just don’t enjoy food the way I used to. It’s like it has no taste.”

1. The patient says, “Food has no taste anymore.” But is this truly a “taste deficit” or something else? Define the word “taste” as applied in a biomedical context.
2. How would you evaluate whether the patient has a taste deficit or something else? What cranial nerves would you be testing for taste loss and for the something else? What would you expect if you asked the patient to taste a Peppermint Altoids? What sensations might the patient report?
3. After performing the cranial nerve exam, you determine that the patient has lost his sense of smell. How did this occur and what was damaged? And will the patient recover this sense?

Station D: Auditory & Vestibular Pathways

Auditory Pathway

Review Questions:

1. What part of the basilar membrane is most sensitive to high frequency sounds?
2. What part of the basilar membrane is most sensitive to low frequency sounds?
3. Damage to what part(s) of the pathway would cause “peripheral hearing loss”?
4. Damage to what part(s) of the pathway would cause “central hearing loss”?
5. What nucleus is the first to receive input from both ears and is important for sound localization?

Vestibular Pathway

Review Questions:

1. The ampulla of the semicircular canals detects what type of motion? _____
2. The saccule is most sensitive to what type of motion? _____
3. The utricle is most sensitive to what type of motion? _____
4. Vestibular information (1) feeds into many places. It descends to the spinal cord for the _____ tracts. It goes to the _____ nucleus in the brainstem (2a). It also goes to the _____ (2b) through the inferior cerebellar peduncle. It also feeds into the MLF for the VOR, and it ascends to the thalamus and from the thalamus to cortex (3) where it synapses onto multiple targets.

LAB 8: Brainstem Cases Lab

Maureen Stabio, PhD – Department of Cell & Developmental Biology

Learning Objectives

1. Distinguish the orientation of the brainstem in CT/MRI vs. histological cross-section.
2. Explain the deviation of the tongue upon a cranial nerve XII lesion.
3. Differentiate Bell's palsy from a UMN lesion of the facial pathway (i.e. ischemic stroke to the face area of the primary motor cortex)
4. Recall the trigeminal pathway and the potential symptoms of lesions in this pathway.
5. Describe the classic triad of symptoms due to an uncus herniation compressing the midbrain.
6. Identify all innervation to the tongue and differentiate taste sensation from somatosensation.

***Important FYI: Spatial Orientation is flipped in histological vs. radiological images. You will be tested on the histological sections of the brainstem, but these clinical cases include MRI images.

Case 1

A 25-year-old male presents to the emergency room with dysarthria and minor difficulty in swallowing. He tells the doctor, "It feels like my tongue is thick in my mouth." When asked to stick out his tongue for examination, his tongue is significantly deviated to the right side (Figure 1) and shows atrophy and weakness. When the patient rested his tongue on the floor of his mouth, minor fasciculations were observed. Sensation of the face is intact bilaterally, but he complains of weakness on the left side of his body. The patient was sent for the imaging shown in Figure 2 below.

Figure 1: Patient's tongue deviation

Figure 2: Axial (left panel) and coronal (right panel) T1 MR images reveal a large schwannoma (T)
Before you answer the MCQs, make a list of relevant deficits.

1. The compression of which cranial nerve is causing deviation of the tongue to the right?
 - i. Right hypoglossal nerve
 - ii. Left hypoglossal nerve
 - iii. Right glossopharyngeal nerve
 - iv. Left glossopharyngeal nerve

2. At what level of the brainstem is the hypoglossal nerve?
 - i. Pons
 - ii. Medulla
 - iii. Midbrain

3. Why is the patient's sensation of the face intact?
 - i. Sensation of the face is carried by the facial nerve, which is in the pons, rostral to lesion.
 - ii. Sensation of the face is carried by the facial nerve, which is in the medulla, caudal to lesion.
 - iii. Sensation of the face is carried by the trigeminal nerve, which is in the midbrain, rostral to the lesion.
 - iv. Sensation of the face is carried by the trigeminal nerve, which is in the pons, rostral to the lesion.

4. Why does the patient also present with weakness on the left side of the body?
 - i. The schwannoma is compressing the right cerebral peduncle
 - ii. The schwannoma is compressing the left medial lemniscus
 - iii. The schwannoma is compressing the right pyramid
 - iv. The schwannoma is compressing the left pyramid

5. The motor deficits of the tongue are due to a(n) _____ lesion, and the motor deficits in the leg are due to a(n) _____ lesion.
 - i. LMN; LMN
 - ii. LMN; UMN
 - iii. UMN; LMN
 - iv. UMN; UMN

Case 2

A 52-year-old female was admitted to the hospital after experiencing nausea, dizziness, and sudden onset headache. She has hemi-paralysis of facial muscles in the lower right quadrant and difficulty speaking. The patient has a history of hypertension and diabetes mellitus. You perform the cranial nerve exam. At rest, the right side of her mouth droops. When you ask her to smile, she is only able to lift the left corner of her mouth (Figure 1).

However, when you ask her to raise her eyebrows and crinkle her forehead, she can do so without difficulty. No fasciculations are present in facial muscles, and the corneal reflex is intact.

Before you answer the MCQs, make a list of relevant symptoms and draw on the diagram below with x marks to indicate the areas with deficits.

LIST INTACT FUNCTIONS	LIST THE DEFICITS

6. A CT scan is ordered. In the meantime, based on your knowledge of neuroanatomy, is the hemiparesis of facial muscles due to an upper motor neuron (UMN) lesion or a lower motor

- neuron (LMN) lesion?
- i. LMN
 - ii. UMN
 - iii. Poisoned beets
 - iv. Bear attack

7. Why can she still crinkle her forehead and raise her eyebrows bilaterally?
- b. Bell’s palsy tends to have “eyebrow sparing” due to bifurcation of peripheral facial nerves to both sides of the face.
 - c. The trigeminal motor nucleus in the pons was spared.
 - d. Bilateral innervation from the cortex onto the superior division of the facial motor nucleus in the pons spares the eyebrow muscles.
8. The patient has most likely suffered an ischemic stroke in which vessel?
- e. Right middle cerebral artery
 - f. Left middle cerebral artery
 - g. Left anterior cerebral artery
 - h. Right superior cerebellar artery

Case 3

A 68-year-old woman presents with a 3-month history of severe, unilateral facial pain on the right side. She describes sudden episodes of "electric shock-like" pain lasting seconds, affecting her cheek and upper jaw. The pain is triggered by chewing, touching her face, or brushing her teeth, and she is completely pain-free between episodes. Physical examination reveals normal cranial nerve function, though light touch to the right upper lip triggers a typical pain episode.

9. Which of the following cranial nerves is causing the sensation of pain?
 - a. Facial Nerve
 - b. Trigeminal Nerve
 - c. Hypoglossal Nerve
 - d. Glossopharyngeal Nerve
10. Where is this cranial nerve found on the surface of the brainstem?
 - e. Midbrain
 - f. Pons
 - g. Medulla
11. Which nucleus in the brainstem carries this pain information?
 - h. Facial Motor Nucleus
 - i. Solitary Nucleus
 - j. Hypoglossal Nucleus
 - k. Spinal Nucleus of V
 - l. Main Sensory Nucleus of V

Case 4

A 16-year-old male was riding his skateboard after high school one day. He was not wearing a helmet, crashed during a kick-flip and cracked his skull on a set of concrete steps. He was rushed to the emergency room. Neurologic exam reveals right eye deviation "down and out" (to the right) and a dilated right pupil. Visual acuity examination is normal. Peripheral and central vision are both intact in both eyes. He has weakness in the left arm and leg with a Babinski reflex on the left foot. Somatosensory functions appear unaffected. What is the probable cause?

12. Ptosis (drooping of the eyelid) of the right eye with a dilated right pupil would be caused by damage to what cranial nerve?
 - a. Right CN III
 - b. Left CN III
 - c. Right CN VI
 - d. Left CN VI
13. Where is this cranial nerve located?
 - a. midbrain
 - b. pons
 - c. medulla

14. The patient also has motor deficits in the left limbs. Are these due to a UMN or LMN deficit?
- a. LMN
 - b. UMN
15. At this point you should have determined that the left limb motor deficits are due to a lesion somewhere along the corticospinal tract. But, where? Hint: look at the anatomical relationship between the affected cranial nerve and the brainstem.
- a. motor cortex
 - b. internal capsule
 - c. cerebral peduncles
 - d. pyramids

Based on your answers above, you should have identified three anatomical clues (skull fracture, eye movement problem, and foot problem) that play a role in this case. At what location do all three of these structures appear in closest proximity to one another? If you determine this you will know the location of the lesion.

16. What is the most likely explanation of the patient's symptoms?
- a. Lateral Brainstem Syndrome
 - b. Epidural hematoma caused an uncal herniation that is compressing the right midbrain
 - c. Three concurrent ischemic strokes: right PCA, right MCA, and basilar artery

Case 5:

A 52-year-old woman returns to her dentist after noticing absent sweet and salty taste perception on the tip of her tongue, which began after a difficult lower molar extraction (wisdom tooth removal). Taste on the back of the tongue is intact. Tongue movement was normal.

17. Which cranial nerve was damaged from the tooth extraction to account for the loss of taste?
 - a. Trigeminal
 - b. Facial
 - c. Hypoglossal
 - d. Glossopharyngeal

18. What cranial nerve provides innervation of general somatosensation to the tip of the tongue?
 - e. Trigeminal
 - f. Facial
 - g. Hypoglossal
 - h. Glossopharyngeal

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LAB 9: Disorders of Perception

Maureen Stabio, PhD – Department of Cell & Developmental Biology

Learning Objectives

1. Recall the distinctions between reception, sensation, perception, and interpretation along the sensory pathway.
2. Distinguish "disconnection syndromes" from "destruction syndromes," and explain how each produces a mismatch between sensory input and perceptual output.
3. Describe the neuroanatomical basis of phantom limb syndrome, blindsight, Capgras delusion, and hemispatial neglect, with attention to the specific pathway or circuit disrupted in each.
4. Define anosognosia and identify which of the above syndromes it commonly accompanies.
5. Discuss the concept of the brain as maintaining a standing internal model of the body, space, or familiarity, and explain how this model can persist or reassert itself despite absent or contradictory sensory input.

Instructions

Break into groups of 4-5 students each and solve all cases using approximately 15-20 minutes per case. Each group will be selected to present one case to the class at the end of lab. Be prepared to answer all questions for each case. As you work through the following four cases, recall the differences between reception, sensation, perception and interpretation – where along each sensory pathway do these different functions occur?

Case 1:

A 33-year-old male, a former infantryman in the U.S. Army, was five months into a deployment when he was injured at a checkpoint in a vehicle-borne IED attack, losing his right arm below the elbow. Shortly after the loss of his limb, the patient reports experiencing the vivid sensation that his missing limb was still present. He also comments that the phantom will often occupy a habitual posture with the forearm in a pronated position, occasionally changing posture spontaneously. The patient states that his phantom limb will sometimes assume a fixed, painful posture that feels like his hand is clenched tightly. These episodes are distressing because the clenching spasms in the phantom hand cause the patient to experience severe pain that is accompanied by the sensation of fingernails digging into his palm. The episodes are reported to last up to several hours before the patient is able to voluntarily unclench his phantom hand.

He claims that when his face is touched he can also feel his phantom arm being touched. To test this, a cotton swab was used to brush the patient's face at randomly selected points while he closed his eyes. When the cotton swab brushed against his right lower face he reported feeling his phantom hand being brushed as well. Stimuli applied to other parts of his body did not produce the same mislocalized sensation in the phantom hand. A magnetoencephalography (MEG) and MRI study is shown below. Note that it is not shown in the standard imaging orientation convention.

Figure 1. A top-down view of a combined MEG and 3D rendered MRI. Note that the right hemisphere is normal and shows the following somatotopic organization: face (red), hand (green), upper arm (blue). The left hemisphere shows that the face area has extended into the hand territory (Ramachandran and Hirstein, 1998).

Questions

1. The sensation of a phantom limb is very common in amputees. According to Ramachandran, what is the neurological basis for this?

2. What pathways would normally be involved to prevent you from clenching so tightly?

3. Provide a possible explanation for the results of the MEG/MRI and how this accounts for sensation in the patient's face being mislocalized to his phantom hand.

4. How does Ramachandran treat phantom limb pain, and what is the proposed mechanism for why this works?

Case 2

A 53-year-old male suffered a medial right occipital lobe stroke nine months ago. The patient reports that he is unable to see anything in his left field of vision. Neurological examinations indicated that the patient has a left homonymous hemianopia (Figure 1). However, upon further testing, the patient demonstrated that he possessed some residual visual abilities within his cortically blind field defect. While his non-blind field was obscured, he could manually locate objects in his blind field, demonstrated orientation discrimination and motion detection despite not being aware of seeing anything in his left field of vision. To further determine any residual visual abilities in his blind field, basic detection and discrimination skills were tested. The patient could indicate whether or not a $7^\circ \times 7^\circ$ visual angle object was present in his blind field with near perfect accuracy when asked, although he insists that he was just guessing and that it must be by chance. His accuracy was only 90% for smaller figures. The patient was also able to discriminate between X's and O's and horizontal vs. vertical bars in his blind field when the figures were large (~78% and 73% respectively). Again, the patient adamantly denied being able to see the objects and that he was only guessing. However, testing shows that the patient could utilize information in his blind field and identify items at a level of performance that was above chance alone.

Figure 1. Patient's field defect, a left hemianopia, as measured in a visual fields test. The locations that are marked in black are where the brightest stimulus elicited no response (Trevethan, Sahraie, and Weiskrantz, 2007).

Questions

1. Given the history of the patient's left visual field defect, what area of the occipital lobe was damaged to result in his blind field defect?
2. Despite being consciously unaware of visual stimuli in his blind field, the patient still demonstrates some residual visual ability. What is this condition called?
3. What part of the visual pathway is spared to allow for this residual visual ability?
4. To what area of the brain does this pathway carry information?
5. How did they assess blindsight in the patient shown in the Ramachandran video?

Case 3

An 85-year-old right-handed woman suffered a hemorrhagic stroke two years ago that caused extensive damage to the right hemisphere. Neurological examination indicated hyperreflexia and spastic paralysis of her left upper and lower limbs. There was a paralysis of her left lower facial muscles that quickly resolved. She was also found to have left-sided neglect but denies her unilateral neglect as well as her hemiplegia. After undergoing physical therapy the patient is still unable to walk or use her left upper limb. The patient was discharged to permanent nursing home care. She continues to deny her hemiplegia and believes that her left side is fully functional, claiming full physical independence. However, the nursing home staff has reported that the patient, on various occasions, said that her left hand had disappeared. There was also evidence that her left-sided neglect and inattention have persisted. She was referred for neurological testing and a structural brain scan.

Neuropsychological Assessment

The patient was alert and oriented to person, place, and time. She knew the date and the city and country she lived in. She could understand and follow test instructions and had no oral or written comprehension deficits, nor did she have any expressive language deficits. Short-term and long-term memory appeared within the normal range for her age. However, she showed severe left-sided neglect while trying to copy simple figures (Fig. 1c). Spontaneous drawing was also affected by her unilateral neglect, drawing only the right side of a daisy (Fig. 1a) and cramming all the numbers of a clock in the upper right quadrant of a clock face (Fig. 1b). When asked to produce a spontaneous drawing or to write a sentence, the patient used her right hand and only used the far-right side of the sheet of paper that was placed in front of her.

Sensory assessment showed visual and auditory left extinction, and that the patient was insensitive to touch over the left upper and lower limbs. When asked to lift her hands she only lifted her right hand. When the patient was asked whether she could move her left arm, she said, "Yes, but it's better if I rest it." When the patient was asked to get up and greet a visitor at the door, she said, "I could get up to meet them, but the doctor says I should rest." She denies any problems on her left side and claims normal sensation in her left limbs. When asked if her doctor's assessment of left-sided paralysis was correct, she denied any paralysis. She was asked to look at her left leg so she could see that she had no control over it; she acknowledged this but was unable to explain why she had suddenly lost control. When she was unable to perform a bimanual task when asked, she would insist that she did not know why she was unable to complete the requested action. Due to the results of her neuropsychological exam, an MRI was ordered with results shown in Figure 2.

Figure 1. Spontaneous drawing of (a) a daisy; (b) a clock face (Venneri and Shanks, 2004).

Questions

1. Based on the MRI, what lobe of the brain is affected by the stroke on what side?
2. Based on this information, which artery was involved in the stroke?
3. How does the location of the lesion correlate with the patient's signs and symptoms?
4. Why does the patient have paralysis on the left side?

Figure 2. Axial T2-weighted MRI image set (Venneri and Shanks, 2003).

5. On a separate sheet of paper, draw the pathway that is involved in the patient's unilateral hemiplegia and mark where this pathway is disrupted. Is this UMN or LMN damage?
6. The patient has a left-sided visuospatial neglect and does not appear to be aware of her deficits. What are these conditions called?
7. How does this case compare to the one presented in the Ramachandran video? Describe the case and how Ramachandran assessed the patient's condition.
8. Hemispatial neglect almost always occurs for the left visuospatial world. Why don't people get neglect of the right visuospatial world?

Case 4

A 75-year-old woman is suffering from face misidentification after sustaining a head injury one year ago. She now believes that her husband has been replaced by an impostor. She uses cues from her husband's clothing or physical appearance to justify her delusional belief, arguing that this impostor is wearing her husband's watch and has the same birthmark. Occasionally, she searches for her "missing" husband. She is anosognosic about her delusion and will minimize her behavioral disturbances. Interestingly, the husband reports that she can recognize him by voice on the phone, and that the delusion only occurs with the visual modality. Except for her husband, the patient's ability to recognize faces is not altered. Her neurological exam is normal and there are no signs of visual field deficits. A CT scan of the brain reveals no obvious abnormality. However, an fMRI scan reveals unusual activation, shown in Figure 1.

Questions

1. The patient misidentifies her husband's face and holds the delusional belief that he has been replaced by an impostor. What is this unusual condition called?
2. What is the role of the fusiform face area?
3. What areas of the brain show unusual activation patterns in the fMRI above?

Figure 1. Neural activity to the partner's face in a control (A) and the patient (B). Note the absence of activity when the patient is shown her husband's face. Posterior cingulate cortex (PCC), posterior superior temporal sulcus (pSTS) and supramarginal gyrus (SMG) (Thiel et al., 2013). Fusiform face area activation was normal.

4. What do those areas do (as it relates to facial recognition) and how do they interact to allow you to recognize someone as familiar?
5. How does the unusual neural activation in the fMRI explain why the patient is able to recognize faces but still misidentify her husband?
6. Describe a similar case as it was presented in the Ramachandran video.

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Special Thanks to Wei Szeto for the original draft of this lab guide.

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Case 3 adapted from:

1. Venneri A, Shanks MF. Belief and awareness: Reflections on a case of persistent anosognosia. *Neuropsychologia* 2004;42(2):230-8.

Case 4 adapted from:

1. Hirstein W, Ramachandran VS. Capgras syndrome: A novel probe for understanding the neural representation of the identity and familiarity of persons. *Proceedings of the Royal Society B: Biological Sciences* 1997 03/22;264(1380):437-44.
2. Jedidi H, Daury N, Capa R, Bahri MA, Collette F, Feyers D, Bastin C, Maquet P, Salmon E. Brain metabolic dysfunction in Capgras delusion during Alzheimer's disease: A positron emission tomography study. *American Journal of Alzheimer's Disease and Other Dementias* 2013 June 30.
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UNIT V:

MOTOR, LIMBIC, & INTEGRATIVE SYSTEMS

Lecture 33: Motor Systems Overview

John Caldwell, PhD – Department of Cell and Developmental Biology

Remote Lecture

Please watch the video for this lecture on Canvas while you enjoy your holiday week!

Learning Objectives

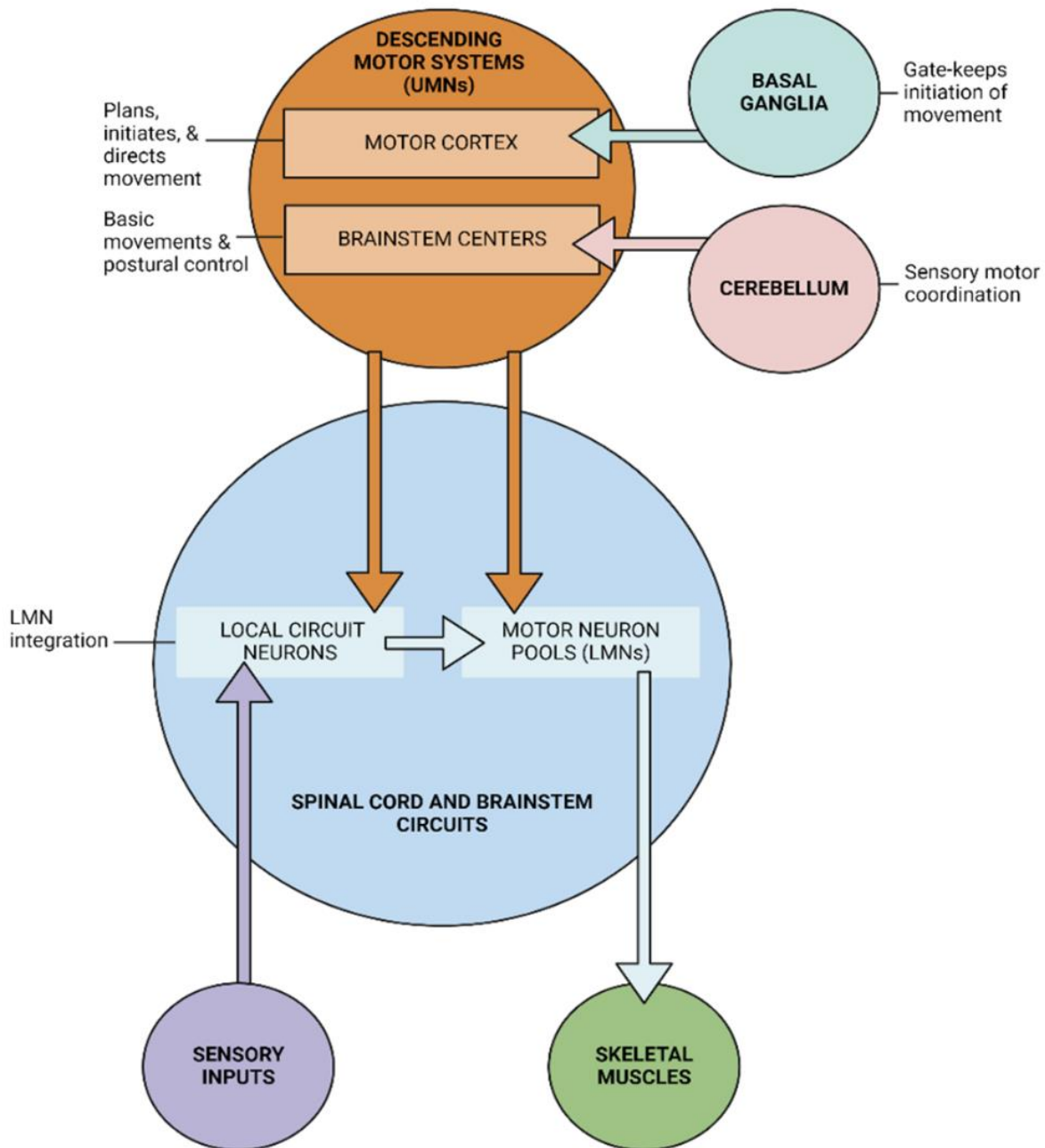
1. List and describe the function of three major motor systems.
2. List and describe the function of three additional motor systems important for the control of muscle tone and coordination.
3. Define pyramidal (pathways that go through the pyramids) vs. extrapyramidal (everything else) motor systems.
4. Review the differences between upper and lower motor neurons.
5. Compare and contrast the medial and lateral pathways from the cortex and brainstem that control lower motor neurons.

Terms to Know

- Three main motor systems
 - Motor cortex (corticospinal or corticobulbar tracts)
 - Cerebellum (modulatory)
 - Basal Ganglia (modulatory)
- Additional subcortical/brainstem motor systems for control of muscle tone, posture, and coordination
 - Red nucleus (rubrospinal pathway)
 - Reticular formation (reticulospinal pathway)
 - Vestibular nuclei (vestibulospinal pathway)
- UMN vs. LMN
 - UMN lesion
 - Spastic paralysis
 - Minimal atrophy
 - Increased deep tendon reflexes
 - No fasciculations
 - Increased muscle tone
 - LMN lesion
 - Flaccid paralysis
 - Severe atrophy
 - Absent deep tendon reflexes
 - Muscle fasciculations
 - Decreased muscle tone
- Lateral and Medial pathways
 - Lateral: important for fine movements of the limbs (especially digits of the hand)
 - Lateral corticospinal tract (from primary motor cortex)
 - Rubrospinal tract (from the red nucleus)
 - Medial Pathways: important for posture and equilibrium
 - Anterior corticospinal tract
 - Vestibulospinal tract (from brainstem vestibular nuclei)
 - Reticulospinal tract (from brainstem reticular formation)
 - Lateral pathways are purely contralateral. Medial pathways can bilaterally innervate spinal motor neurons.

Review of UMN vs. LMN

Signs	UMN Lesion	LMN Lesion
Type of paralysis	Spastic	Flaccid
Atrophy	Minimal (from disuse)	Severe
Deep tendon reflexes	Increased	Absent
Muscle fasciculations	Absent	Present
Muscle tone	Increased	Decreased



Lecture 34: Basal Ganglia (aka Basal Nuclei)

John Caldwell, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Basal Ganglia (Ch. 45): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/basal-ganglia/>

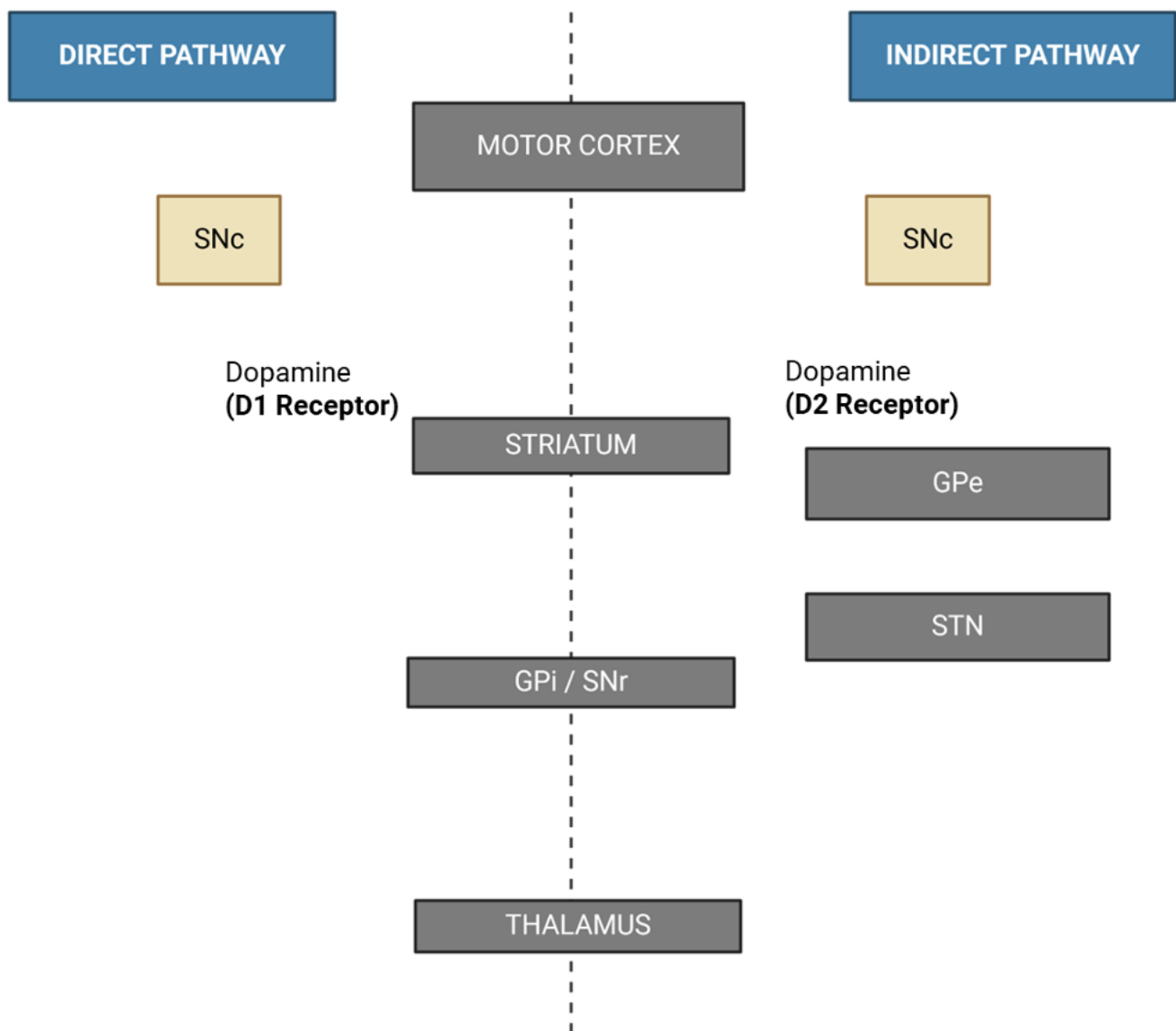
Neurodegenerative Diseases: Motor (Ch. 46) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/neurodegenerative-diseases-motor/>

Learning Objectives

1. Describe the major motor function of the basal ganglia (basal nuclei).
2. Review the anatomical structures that make up the basal ganglia.
3. Describe the basic circuitry through which the basal ganglia influence motor activity in the CNS (if the basal ganglia are damaged on one side, are the symptoms on the ipsilateral or contralateral side relative to the damage).
4. Draw and describe the direct pathway circuitry with an excitatory/inhibitory synapse indicated.
5. Describe the anatomical components and effects of dopamine on the direct pathways (in other words, what is the effect of adding dopamine and how does it work in the direct pathways?).
6. Describe the type of movement that is observed when the subthalamic nucleus is damaged (does this make sense from what you know about the functional output of the subthalamic nucleus to the GP internal?).
7. Discuss the role of the basal ganglia in repetitive motor behaviors (i.e., OCD and Tourette's).
8. Define the neurobiological basis of Parkinson's disease and Huntington's disease (i.e., what basal ganglia structures are affected?).
9. Compare basal ganglia disease with other motor neuron diseases such as polio or ALS.

Terms to Know

- Anatomical structures
 - Telencephalic structures:
 - Caudate
 - Putamen
 - Globus Pallidus (internal and external)
 - Note: "striatum" refers to the caudate and putamen; "lenticular nucleus" refers to the putamen and globus pallidus
 - Diencephalic and brainstem structures:
 - Subthalamic nuclei
 - Substantia nigra (remember this structure makes dopamine)
- Pathways
 - Direct
 - Indirect
 - (You should know the final output of both pathways (as in this table), but you are only responsible for memorizing the exact circuitry of the direct pathway)
- Diseases that involve the Basal Ganglia
 - Parkinson's disease
 - Huntington's disease
 - OCD



Basal Ganglia	Direct Pathway	Indirect Pathway
Without Substantia Nigra		
With Substantia Nigra		

Lecture 35: Cerebellum

Christy Niemeyer, PhD – Department of Neurology

Learning Objectives

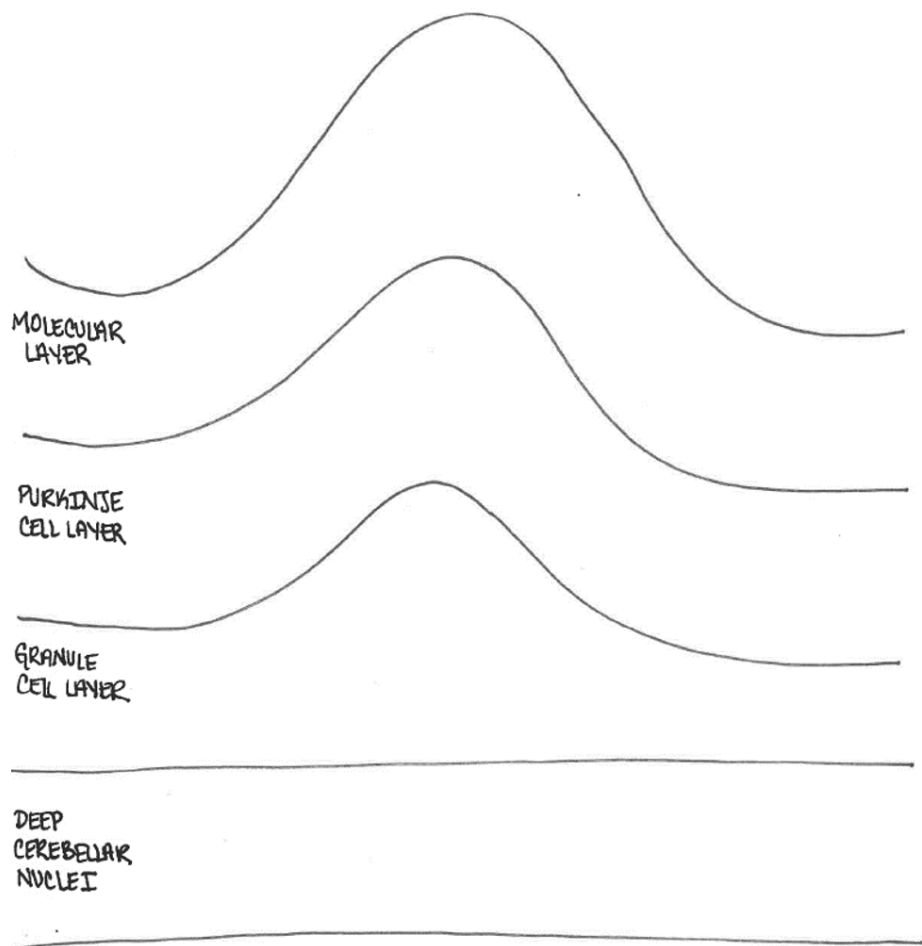
1. Compare and contrast cerebrum and cerebellum in terms of the following: volume, percentage of total neurons in the brain, number of peduncles (and their names), and number of histological layers of cortex.
2. List the major functions of the cerebellum.
3. Define folia and arbor vitae.
4. Describe the anatomical and functional divisions of the cerebellum.
5. Review the function of the cerebellum circuitry in motor learning/adaptation.
6. Review the difference between feedback and feedforward control of movement.
7. From prework video: describe the cells that make up the cerebellum and diagram their circuitry.

Terms to Know

- Anatomical organization of the cerebellum
 - Folia = “leaves” or grey matter (analogous to gyri of the cerebrum)
 - Arbor Vitae = “tree of life” or white matter
 - Hemispheres (left and right)
 - Vermis
 - Anterior lobe
 - Posterior lobe
 - Flocculonodular lobe
 - Deep cerebellar nuclei: Dentate, Emboliform, Globose & Fastigial (“Don’t Eat Greasy Food”)
 - Superior, Inferior, & Middle Cerebellar Peduncles

- Functional organization of the cerebellum
 - Cerebrocerebellum
 - “Neocerebellum”
 - Consists of lateral portions of the cerebellum
 - Important for the planning of movement
 - Spinocerebellum
 - “paleocerebellum”
 - Consists of vermis and paravermis
 - Important for the proper execution of movement
 - Vestibulocerebellum
 - “archaeocerebellum”
 - Consists of the flocculus and the nodulus
 - Important for equilibrium and coordinated eye movements (vestibulo-ocular reflex)
- Functional contribution of cerebellar circuitry to motor adaptation
 - Delay eyeblink conditioning/Prism Goggle experiment
 - Where does the conditioned stimulus come from?
 - Where does the unconditioned stimulus come from?
 - Where does the “teaching signal” come from?

- Histological organization of the cerebellum (3 layers) (please watch the pre-lab video to complete the diagram below on the histology of the cerebellum.)
 - Molecular layer
 - Dendrites of Purkinje cells
 - Unmyelinated axons (parallel fibers) of granule cells
 - Basket cells
 - Stellate cells
 - Purkinje cell layer
 - Purkinje cell bodies – only output of cortex; inhibitory (GABA)
 - Granule cell layer
 - Granule cells – extremely small and numerous
 - Golgi cells
- Input fibers to the cerebellum
 - Mossy fibers – contact the granule cell layer
 - Climbing fibers – contact Purkinje cells



Lecture 36: Limbic System

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

UBC Limbic System: https://www.neuroanatomy.ca/syllabi/Limbic_2024.pdf

Emotions: Overview (Ch. 58): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/emotions-overview/>

Emotions: Fear (Ch. 59): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/studying-fear/>

Memory Systems (Ch. 66): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/memory-systems/>

Psychiatric disorders (Ch. 62-63): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/mental-illness-depression/>

Learning Objectives

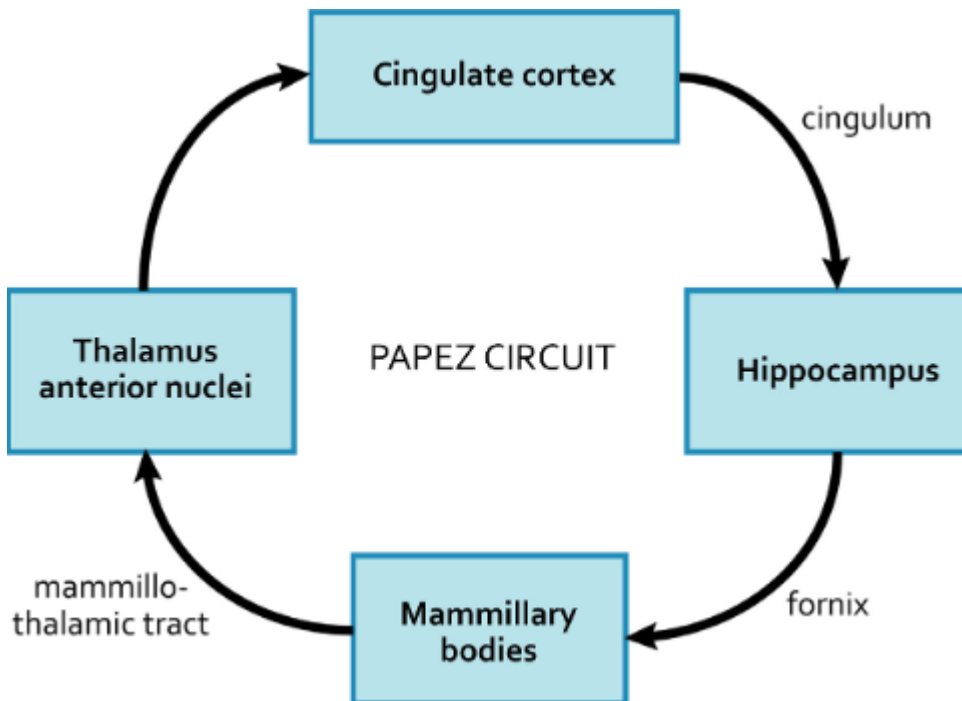
1. Describe the function of the limbic system and identify the structures classically included in it.
2. Draw and label the Papez circuit, trace the flow of information through its components, and describe its role in memory.
3. Compare and contrast anterograde versus retrograde amnesia using clinical case examples, and localize each pattern to specific points of circuit disruption.
4. Describe the neuroanatomical basis of Wernicke-Korsakoff syndrome (mammillary body and thalamic involvement) and relate its symptoms of confabulation and anterograde amnesia back to the Papez circuit.
5. Describe the role of the amygdala in regulating fear, aggression, and emotional processing, and explain how it and other limbic components integrate environmental input to generate an emotional response.
6. Define Kluver-Bucy syndrome, including its neuroanatomical basis (bilateral amygdala/temporal lobe damage).
7. Define Urbach-Wiethe disease, including its neuroanatomical basis (amygdala calcification) and symptoms, and use it as a natural-experiment contrast to Kluver-Bucy.
8. Define the Duchenne smile and describe the neuroanatomical basis of emotionally-driven (involuntary) facial expression.
9. Define a "mood disorder" and compare and contrast the HPA-axis dysfunction hypothesis with the stress-diathesis hypothesis as competing (or complementary) explanatory models.

Terms to Know

- Major Limbic Structures (*Objective 1*)
 - Amygdala
 - Hippocampus
 - Parahippocampal gyrus
 - Cingulate gyrus
 - Anterior nucleus of thalamus
 - Mammillary bodies of hypothalamus
- Papez Circuit (*Objective 2*)
 - Hippocampus
 - Fornix
 - Mammillary body
 - Anterior nucleus of thalamus
 - Fibers from thalamus to cingulum
 - Cingulum
 - Entorhinal cortex and parahippocampal gyrus back to hippocampus
 - Cingulate gyrus

- Memory Circuit Dysfunction (*Objectives 3–4*)
 - Anterograde amnesia
 - Inability to form new memories after lesion
 - Classic case: patient H.M. (bilateral hippocampal resection)
 - Retrograde amnesia
 - Loss of memories formed before lesion
 - Often temporally graded
 - Wernicke-Korsakoff syndrome
 - Cause: thiamine (Vitamin B1) deficiency
 - Lesion site: mammillary bodies, dorsomedial thalamus
 - Wernicke's triad (acute): confusion, ataxia, ophthalmoplegia
 - Korsakoff syndrome (chronic): confabulation, anterograde amnesia
- Amygdala Circuit (*Objective 5*)
 - Amygdala subnuclei
 - Basolateral nucleus — sensory input
 - Central nucleus — output
 - Major connections
 - Sensory thalamus and cortex (input — fear conditioning)
 - Hypothalamus (output — autonomic response)
 - Periaqueductal gray (output — freezing behavior)
 - Prefrontal cortex (top-down regulation)
 - Fight, flight, or freeze response
- Amygdala Circuit Dysfunction (*Objectives 6–7*)
 - Kluver-Bucy syndrome
 - Lesion: bilateral amygdala / anteromedial temporal lobe
 - Hyperorality
 - Hypersexuality
 - Docility / loss of fear
 - Visual agnosia ("psychic blindness")
 - Urbach-Wiethe disease
 - Lesion: bilateral amygdala calcification (selective)
 - Selectively blunted fear recognition/response
 - Other emotions and cognition largely intact
 - Contrast point: more selective than Kluver-Bucy's broader temporal damage
- Emotional Expression (*Objectives 8–9*)
 - Duchenne smile
 - Zygomaticus major (mouth) + orbicularis oculi (eyes)
 - Involuntary, genuine emotional expression
 - Pathway: limbic / extrapyramidal
 - Voluntary (pyramidal) smile
 - Pathway: corticobulbar (pyramidal)
 - Direct cortex → facial nucleus route
 - "Smile on command"
 - Clinical correlate
 - Central facial (UMN) lesion, e.g., stroke
 - Voluntary smile lost, emotional smile can remain intact — two separate pathways
- Mood Disorders (*Objective 9*)

- Definition
 - Persistent disturbance in mood (depressive, manic, or both)
 - Causes clinically significant distress or impairment
- HPA-axis dysfunction hypothesis
 - Hypothalamus (CRH) → Pituitary (ACTH) → Adrenal gland (Cortisol)
 - Chronic stress → sustained cortisol → impaired feedback, hippocampal changes
- Stress-diathesis hypothesis
 - Diathesis (genetic/biological vulnerability) + environmental stressor
 - Explains variable onset — not everyone under stress develops a mood disorder
 - Complements, rather than competes with, the HPA-axis model
- Full Circuit Integration (*Synthesis*)
 - Papez circuit lesion → memory circuit dysfunction (Wernicke-Korsakoff)
 - Amygdala circuit lesion → emotion circuit dysfunction (Kluver-Bucy, Urbach-Wiethe)
 - Mood disorders → distributed dysfunction across both circuits



The Classic Papez Circuit from the UBC Vancouver Limbic System Module
https://www.neuroanatomy.ca/syllabi/Limbic_2024.pdf

Lecture 37: Mesolimbic & Mesocortical Pathways

Erika Alor, MS – Department of Cell & Developmental Biology

Optional Foundational Reading

Motivated Behavior: Reward Pathway (Ch. 49): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/motivation-and-reward/>

Drug Addiction (Ch. 51): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/drug-addiction/>

Psychiatric Disorders: Schizophrenia (Ch. 65) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/mental-illness-schizophrenia/>

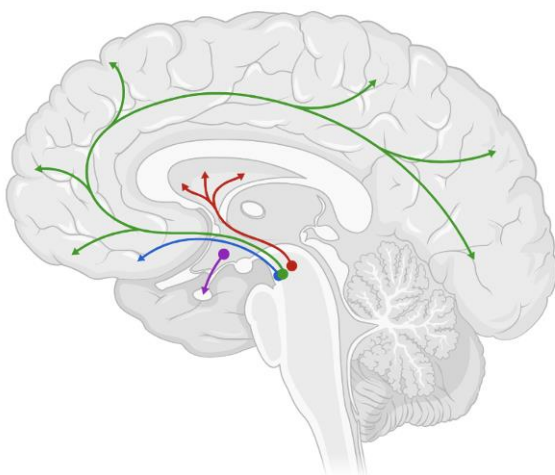
Learning Objectives

1. Describe the structural components, projections, and functions of the 3 dopaminergic pathways: nigrostriatal, mesolimbic and mesocortical.
2. Describe the role of dopamine in the anticipatory response.
3. Define reward and the reward pathway, and discuss how it relates to addiction.
4. Define two hallmark characteristics of addiction: tolerance and withdrawal.
5. Describe an example of how the brain changes with addiction.
6. Describe how cannabis is potentially harmful to adolescents.
7. Compare and contrast diseases associated with dopamine dysregulation, distinguishing conditions caused by dopamine deficiency (e.g., Parkinson disease) from those associated with dopamine excess or dysregulated signaling (e.g., schizophrenia, substance use disorder).
8. Describe the dopamine hypothesis of schizophrenia; differentiate positive, negative, and cognitive symptoms.
9. Describe mesolimbic hyperactivity underlying positive symptoms and mesocortical hypoactivity underlying negative and cognitive symptoms.

Terms to Know

- Dopaminergic Pathways (*Objective 1*)
 - Nigrostriatal pathway
 - Substantia nigra pars compacta
 - Striatum (caudate + putamen)
 - Function: motor control
 - Mesolimbic pathway
 - Ventral tegmental area (VTA)
 - Nucleus accumbens
 - Ventral striatum, amygdala, hippocampus
 - Function: reward, motivation, emotion
 - Mesocortical pathway
 - Ventral tegmental area (VTA)
 - Prefrontal cortex
 - Function: cognition, executive function, mood regulation
- Reward and Addiction (*Objectives 2–5*)
 - Reward and reinforcement
 - Motivation
 - Liking vs wanting
 - Anticipatory dopamine response
 - Addiction

- Tolerance
- Withdrawal
- Reward Pathways (*Objectives 2–3*)
 - Mesolimbic pathway
 - Ventral tegmental area (VTA)
 - Nucleus accumbens (NA)
 - Ventral striatum
 - Medial forebrain bundle (MFB)
 - VTA
 - NA
 - Amygdala
 - Septal nuclei
 - Prefrontal cortex
- Adolescent Cannabis Use (*Objective 6*)
 - Endocannabinoid system and dopamine interaction
 - Adolescent prefrontal cortex still developing
 - Risks: altered reward circuitry, cognitive effects, psychiatric risk
- Diseases of Dopamine Dysregulation (*Objective 7*)
 - Dopamine deficiency
 - Parkinson disease (nigrostriatal pathway)
 - Dopamine excess / dysregulated signaling
 - Schizophrenia (mesolimbic/mesocortical pathways)
 - Substance use disorder
- Dopamine Hypothesis of Schizophrenia (*Objectives 8–9*)
 - Mesolimbic hyperactivity → positive symptoms
 - Hallucinations
 - Delusions
 - Mesocortical hypoactivity → negative and cognitive symptoms
 - Negative: flat affect, avolition, alogia
 - Cognitive: memory, attention, executive function deficits



Dopaminergic Pathways

Nigrostriatal • Voluntary movement production

Mesocortical • Cognition
• Memory & learning
• Motivation

Mesolimbic • Emotion
• Perception
• Reward

Tubero-infundibular • Sensory processing
• Hormonal regulation
• Maternal nurturing

Lecture 38: Brain Waves, EEG, & Sleep

John Caldwell, PhD – Department of Cell and Developmental Biology

Optional Foundational Reading

Sleep and EEG (Ch. 56): <https://openbooks.lib.msu.edu/introneuroscience1/chapter/sleep-and-eeeg/>

Circadian Rhythms (Ch. 57) <https://openbooks.lib.msu.edu/introneuroscience1/chapter/circadian-rhythms/>

Learning Objectives

1. Discuss the EEG and its role in discovering REM and non-REM sleep states.
2. Describe the two major controls of sleep: circadian rhythm and sleep deprivation.
3. Explain the neural basis for both wake and sleep states, including key brain structures and neurotransmitter systems.
4. Explore the physiological need for sleep (what does it accomplish for the body?).
5. Define disorders of sleep and discover their neural correlates.
6. Describe the neurobiological mechanism by which caffeine promotes wakefulness.

Terms to Know

- EEG, the electroencephalogram, and what it is measuring (synchronicity)
- REM and Non-REM sleep:
 - REM sleep is an “active, hallucinating brain in a paralyzed body.”
 - Non-REM sleep is “an idling brain in a moveable body.”
- Four brain regions are associated with the Reticular Activating System (RAS) and the neurotransmitters they release and their targets in the brain (where they release NTs)
 - LC = Locus Coeruleus, releases norepinephrine
 - Raphe = raphe nuclei, releases serotonin
 - SN = substantia nigra, releases dopamine
 - The midbrain releases histamine
- The VLPO initiates NREM sleep by releasing the inhibitory NT GABA onto nuclei associated with the RAS
- The neural basis for the “switch” between REM and NREM states (no need to memorize nuclei)
- The retinohypothalamic pathway: retinal ganglion cells contain the photopigment melanopsin and project directly to the suprachiasmatic nucleus of the hypothalamus. It’s how light entrains circadian rhythms.
- SCN rhythmically secretes vasopressin
- The pineal gland acts downstream of the SCN and upstream (probably initiates) adenosine signaling
- Physiological need for sleep
 - Beta-amyloid clearance
 - Learning consolidation in REM
 - Increased flow of CSF during sleep to remove metabolites
- Sleep disorders
 - Narcolepsy
 - Sleep apnea

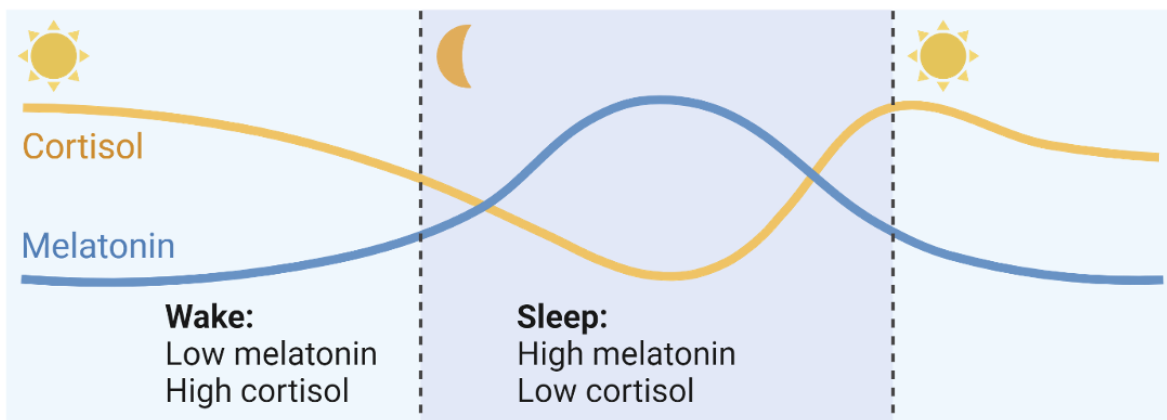
- REM behavior disorder
- Circadian rhythm disorders
- Insomnia (a symptom, not a disease)

What is Circadian Rhythm?

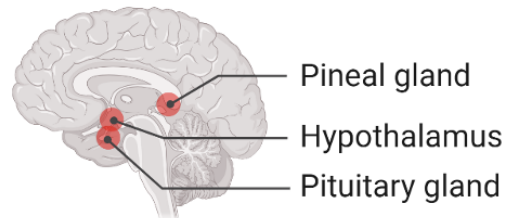


Circadian rhythm is part of the body's **internal clock**; it follows a 24-hour schedule and regulates the sleep-wake cycle.

During the 24-hour cycle, our hormone levels fluctuate in response to light, particularly **melatonin** and **cortisol**.



Regions of the brain that regulate the circadian rhythm include **pineal gland, hypothalamus** and **pituitary gland**.



Lecture 39: Neuropathology Overview

Brad Pawlikowski, PhD – Department of Pediatrics

Learning Objectives

1. Review the “VITAMIN-C” mnemonic of neuropathology.
2. Recall the difference between an obstructive and a hemorrhagic stroke.
3. Recall the difference between encephalitis and meningitis.
4. Define concussion, traumatic brain injury, and chronic traumatic encephalopathy.
5. Define neurodegeneration; match the disease with the part of the nervous system where neurons die and any proteinopathy involved for the following: Alzheimer’s disease, Parkinson’s disease, Huntington’s disease, and Amyotrophic Lateral Sclerosis.
6. Define dementia and list the types of diseases that cause dementia.
7. List two main categories of metabolic disorders (toxicities vs. deficiencies).
8. Compare and contrast multiple sclerosis and Guillain-Barré Syndrome, including the glial cells involved and the tissues affected.
9. Describe the cells involved in the different types of neoplasms.

Terms to Know

- **Vascular**
 - Obstructive Stroke
 - Hemorrhagic Stroke
- **Infectious**
 - Encephalitis
 - Meningitis
 - Neurocysticercosis
 - Other viruses (Rabies, Herpes simplex, Polio, Zika, COVID-19)
- **Trauma**
 - Concussion, TBI, and CTE
 - Spinal Cord injury
- **Age-Related/Degenerative**
 - Alzheimer’s disease (AD)
 - Parkinson’s disease
 - Huntington’s disease
 - Amyotrophic Lateral Sclerosis (ALS)
- **Metabolic**
 - Toxic (heavy metals, industrial chemicals, environmental pollutants, drugs of abuse, ethanol, chemotherapeutic drugs, and ionizing radiation)
 - Vitamin Deficiency
- **Inflammatory**
 - Multiple Sclerosis (MS)
 - Guillain-Barré Syndrome (GBS)
- **Neoplastic**
 - Pituitary adenoma
 - Medulloblastoma (neuron)
 - Meningioma (meningeal cell)
 - Ependymoma (ependymal cell)
 - Glioma (glial cell) – Astrocytoma, Oligodendroglioma, Schwannoma
- **Congenital and Developmental** (congenital disorders taught in MHA Embryology course)

Lecture 40: Emerging Technologies

Maureen Stabio, PhD – Department of Cell and Developmental Biology

Learning Objectives

1. Discuss the NIH BRAIN Initiative, including the goals and motivations for this project.
2. Differentiate neurostimulation (“write-in”) from brain-computer interface (“read out”).
3. Explain how deep-brain stimulation (DBS) works and list the two main deep-brain targets.
4. Describe responsive neural stimulation (RNS) and how it works to prevent seizures.
5. Describe Vagus Nerve Stimulation and list its FDA-approved uses.

Terms to Know

- Advancements in Imaging & Mapping the Brain
- Nerve Regeneration & Stem Cell Technology
- “Neuroprosthetics”
 - **Brain Computer Interface**
 - Recording neural activity to control external effector
 - “Read out”
 - **Neuromodulation**
 - Electrical stimulation to change neural activity
 - “Write in”

LAB 10: Motor System Disorders

Prepared by John Caldwell, PhD, and Christy Niemeyer, PhD

Learning Objectives

1. Distinguish basal ganglia disorders from cerebellar disorders by clinical presentation (Parkinson's, Huntington's vs. cerebellar cases).
2. Localize a motor system lesion and its sidedness from signs alone (including laterality).
3. Connect the underlying pathophysiology of a motor disorder (neurodegeneration, genetic inheritance, or dopamine receptor dysfunction) to its clinical symptoms and treatment.

Instructions

Break into groups and solve all cases using approximately 15-20 minutes per case. Each group will be selected to present one case to the class at the end of lab. Be prepared to answer all questions for each case.

Case 1

A 49-year-old man visits his physician to renew his prescription. As he sits in the waiting room, he is observed to have a tremor in his hands and fingers. His face is unexpressive, and he makes few movements. When he is invited to enter the physician's office, he seems to have difficulty standing up. The patient walks slowly into the office, and his arms do not swing appreciably.

When he talks to the physician, his speech is monotonous, but he shows no intellectual deficit.

1. In what ways does this man's tremor differ from that of a patient with Multiple Sclerosis (demyelination of axons in multiple tracts)? What is the name of the disorder in this case?
2. Why are the movements so few and slow?
3. Are the phasic stretch reflexes altered?
4. What part of the nervous system is involved in this disease?

Case 2

A 46-year-old female living with her nuclear family in the rural area of Rajasthan, India presented to a health clinic with complaints of abnormal, dance-like movements, which were non-repetitive and non-periodic. She stated that the onset of this symptom was associated with a fall she had ~11 years ago. These abnormal movements initially began in her left hand and eventually progressed to both hands. Over time, the movements sequentially involved her upper limbs, lower limbs, neck, and face--and were finally seen as a tremor in her lips and tongue.

During the initial stage of this disease, the patient also reported depressive symptoms such as sleep disturbances, low moods, loss of interest, irritability, and fatigue. As the disease progressed, she began to experience psychotic episodes involving constant suspicion/distrust, aggressiveness, poor hygiene, complete detachment, and even auditory hallucinations. The patient has a positive family history of abnormal movements in both her father and grandfather, who died at the ages of 62 and 80, respectively.

5. This is a movement disorder with behavioral/emotional symptoms. What do the motor symptoms suggest for a diagnosis?
6. What type of a genetic defect (autosomal dominant or recessive) is suggested by her family history?
7. The gene defect can now be identified. What are some ethical concerns associated with having the test done to determine whether someone has the mutated gene?

Case 3

A 26-year-old woman presented to her physician with complaints of clumsiness in her right arm. She also stated that her right hand had a tremor when she attempted to insert the key into the ignition of her car. This woman's friend noted that, while walking on the sidewalk, she tended to reel to the right and repeatedly bumped into her. Her friend also noticed that the woman's speech was slurred for particular sounds and that she spoke more slowly and deliberately. Upon physical examination, the physician noted that this woman's right shoulder was slumped lower than her left shoulder. There was evidence of hypotonia and looseness on the right side when her arms and legs were passively moved. When she was asked to touch her nose with her right index finger, the woman's right hand displayed tremor and her finger overshot the target. When asked to do the same task with her left hand and finger, there was no abnormality present.

8. What is a possible cause of the lesion in this case?
9. Where is this lesion located?
10. What side of the body is affected relative to the abnormal symptoms?
11. What are the clinical terms to describe each of the woman's symptoms?
12. Symptoms of clumsiness, intention tremor, muscle incoordination, and hypotonia involving the muscles on the right side can be explained by all the following EXCEPT:
 - a. The right cerebellum is affected
 - b. The voluntary muscles on the right side of the body are not receiving the correct nervous control
 - c. The sequence of firing of the alpha motor neurons controlling the muscles on the right side is not normal
 - d. The efferent axons, which travel directly from the cerebellum to the lower motor neurons, are not functioning
 - e. The cerebellum is not sending information to the motor cerebral cortex to inhibit the agonist muscles and stimulate the antagonist muscles

Case 4

A 24-year-old female was admitted to the hospital complaining of dizziness and the inability to walk steadily for more than 20 years, but who now had additional nausea and vomiting in the past month. The woman is married and has one daughter, whose pregnancy and delivery were described as uneventful. Her parents have no history of neurological disorders. Her four sisters and one brother are described as neurologically normal.

According to this patient's mother, she was 4 years old before she could stand unassisted and did not begin to walk unassisted until the age of 7, with a persistently unsteady gait. She never ran or jumped. Her speech was not intelligible until ~6 years of age and she did not enter school at that time. A neurological examination revealed this woman was cooperative and fully oriented. A verbal analysis test revealed her word comprehension and expression remained intact and she has no sign of aphasia but does show mild to moderate signs of dysarthria. The patient has a mild voice tremor with slurred pronunciation and her voice quality is slightly harsh. Ataxia is present, and there is evidence of heel-knee-tibia impairment. The patient experienced mild to moderate dysmetria in reaching her nose when she was administered the finger-to-nose test. Pronation-supination rapid alternating movements were slightly irregular and slowed. While she is able to walk without support, her gait is moderately unsteady. The patient has difficulty with tandem gait (walking a straight line, heel-to-toe, as seen in a sobriety test) and relatively reduced gait speed. There is no focal paresis (muscle weakness) present, but her muscle tone is mildly increased.

Evaluation of the sensory system showed no abnormalities, she had no deformities of the fingers, and her complete blood count and urinalysis were normal.

1. Where is the location of the lesion in this case?
2. Is this a result of trauma or is it congenital?
3. If it is congenital, why is she able to perform better than someone with a cerebellar lesion?
4. Are the abnormal symptoms present on the left or right side?

Case 5

A 16-year-old male presented to the psychiatric outpatient department with a 6-year history of repetitive involuntary blinking, shoulder shrugging, sniffing, vocalization, and whistling. The intensity and persistence of the symptoms waxed and waned spontaneously, but he stated he was never completely symptom-free. Symptoms tended to be less severe in the summer and when engaged in physical activity, but these symptoms would be exacerbated when he was fatigued, stressed, or sitting idle. He could only voluntarily suppress the tics for a few minutes at a time, but this was associated with severe stress and restlessness. Over the past year, new symptoms had emerged (grimacing and foot tapping). He feels increasingly embarrassed in public, his school grades are deteriorating, and he finds himself feeling irritable throughout the day. Due to his loss of self-esteem, he has not been attending school for 2 months. The patient's family had helped him seek treatment 4 years prior (2 years after the initial onset of symptoms). At that time, he was treated with 10 mg/d Haloperidol in divided doses. Haloperidol is used as an antipsychotic medication and acts by blocking dopamine D2 receptors. This medication seemed to resolve the patient's symptoms, and he remained in remission for 1 year. However, the patient stated he stopped the Haloperidol treatment because of unfavorable side effects (restlessness,

tremors, slurred speech, motor retardation). His tic symptoms re-emerged 3 months later. The physician decided to start this patient on Aripiprazole, a partial agonist of the dopamine D2 receptor, which works by reducing dopamine neurotransmission, mainly in the mesolimbic and mesocortical pathways. This drug does not solely act on dopamine receptors—it also has effects on serotonin receptors. After steadily increasing the dosage of Aripiprazole over a period of 6 weeks, the patient’s symptoms disappeared. He is currently stable.

1. What is the diagnosis for this patient?
2. The medications prescribed are antipsychotics that are D2 receptor antagonists. What pathway for movement utilizes D2 receptors?

Optional video: “I have Tourette’s, but Tourette’s doesn’t have me” — an interesting HBO documentary film that interviews children with T.S. You can watch the trailer on YouTube if you are interested in a brief introduction: https://www.youtube.com/watch?v=7_dBRDvkbTU

References:

- *A Rare Case of Huntington’s Disease Associated with Depression and Psychotic Features.* Pankaj Kumar, Supriya Suman, Pratima Singh, Ranjeet Kumar *International Journal of Health Sciences and Research* Vol.10; Issue: 4; April 2020
- *Cerebellar Agenesis.* Feng Yu, Qing-jun Jiang, Xi-yan Sun and Rong-wei Zhang (2015). *A new case of complete primary cerebellar agenesis: clinical and imaging findings in a living patient.* *Brain* 138; 1–5

LAB 11: VITAMIN-C Neuropathology Lab

MHA Teaching Team

Learning Objectives

1. Examine radiological, histological, & gross features of various vascular, infectious, traumatic, age-related, metabolic, inflammatory, neoplastic & congenital neurological disorders.

Instructions

You will work in groups to rotate through 8 stations that examine and review pathological disorders that we have discussed in this class. Please read the following guidelines before you begin:

- **HUMAN TISSUES:** The human tissues you will examine in this lab come from the Pathology Department collection directed by Dr. BK DeMasters through the hospital autopsy signed consent donation program for use in education and research. Plastinated brains come from the Modern Human Anatomy Program plastinated collection directed by Dr. Maureen Stabio. These brains were donated to the University of Colorado Anschutz Medical Campus through the Colorado State Anatomical Board for the purpose of education and research. The giving of one's body for the purpose of education is an immeasurable gift, and each donated body has been treated and preserved with the highest ethical standards. Please treat photos and specimens with the utmost gentleness and respect.
- **SAFETY:** The wet tissues have been stored in 10% formalin and rinsed in water 3 times for >48 hours. Please wear proper PPE; a mask is recommended. The plastic specimens have been preserved through plastination, a tissue preservation process in which the water and lipids are replaced with silicone, and thus you do not need to wear gloves to touch them unless you feel more comfortable. If you are wearing gloves to touch the plastinates, make sure they are clean/dry gloves.
- **MINDFULNESS:** Learning about neurological diseases and seeing pathological brain specimens can bring up many feelings. The lab may stir up fascination and curiosity as well as feelings of sadness for the victims and their families. It is OK to carry both of these feelings simultaneously. Should you experience a strong emotional response, please feel free to step out of the room or do what you need to do for self-care in that moment (without penalty).

Treat brain tissue with gentleness and respect.
It is a gift to you for your education.

Station 1: Vascular

Case:

Severe, widespread Cerebral Amyloid Angiopathy, bifrontal intracerebral hemorrhages and left subdural hematoma and histological hallmarks suggestive of AD. (Bucket #A21-63)

Clinical Presentation:

A 75 y/o male presented to the ED with sudden onset severe headache, confusion, and cognitive changes. His family reported that they had noticed slowly progressive increasing depression, agitation, and personality changes over the last 6 months. Head CT and MRI demonstrated large bifrontal intracerebral hemorrhage. He was transferred to the hospital and admitted to the NICU for several days. After transfer to inpatient rehab, he had acute onset of not speaking and increased headache, and repeat imaging found two new smaller areas of hemorrhage in the bilateral frontal lobes. He spent several more days in the ICU; workup included additional MRI with and without contrast that was positive for bifrontal intracerebral hemorrhage, with evidence of differing chronicities of hemorrhage (some old; some new).

Imaging:

Observe the CT and MRI images.

1. What is observed in the MRIs? Is the blood epidural, subdural, intraventricular or intraparenchymal? Does there appear to be just one bleed or multiple bleeds?

Etiology & Mechanism:

What caused these bleeds? The patient was negative for suspected underlying glioma. An MR venogram was negative for dural venous sinus thrombosis. Thus, after ruling out tumor and dural thrombosis and after additional review with the medical team, the attending physician recorded that the bleeds were most likely related to hypertension or to cerebral amyloid angiopathy.

2. What does 'Cerebral Amyloid Angiopathy' mean?

Neuropsych Testing:

Neuropsych testing found global cognitive dysfunction but was most notable for consistent impairments in executive functioning, particularly in areas such as initiation and set-shifting as well as perseverative and stimulus-bound behaviors. In addition to severely impaired semantic fluency, mild to moderate impairments were noted across nearly all other domains, including processing speed, learning/memory and language functions, with the exception of simple attention and visuospatial abilities. The patient enrolled in rehab and made significant improvement in physical, occupational, and speech/cognitive therapies over the course of a year. He continued to gain independence and could perform all of his ADLs, although he continued to have cognitive deficits.

3. How do the cognitive deficits align with the location of bleeds? (Recall the main functions of frontal lobe)

Gross Neuropathology:

The patient was relatively stable for 2 years and passed away at home. Autopsy was performed.

4. What can you observe at a gross level?

Histopathology:

5. Observe the histology slide. The information given from the autopsy suggests the patient's brain had 'suggestive' (but not definitive) histological hallmarks of Alzheimer's Disease. How would you link those histological hallmarks to the information given to you about the patient?

EXTRA Course Review from Unit 1:

View the brain with Basilar aneurysm: In what vessel was the aneurysm?

Recall the appearance of different bleeds in the brain by filling out the chart below:

Station 2: Infectious

Case: Sepsis

Clinical Presentation:

A 60-year-old woman with history of cirrhosis, diabetes type II, minimal healthcare follow-up, and no daily medications, presents with altered mental status. Her spouse reports that she has been more fatigued than usual for the past week. Last night, she seemed more confused but was able to converse with their son and eat dinner. Today patient stayed in bed for most of the day. Her spouse reports that she kept saying she would get out of bed, but never actually did. Patient is awake, has increased work of breathing, not verbally responsive, not following commands, but is purposeful in all four extremities. The patient has septic shock due to an infection with *Staphylococcus aureus* that originated in the heart (aka: septic emboli as a result of infective endocarditis). The infection has spread to the brain. The patient unfortunately did not survive and the brain was removed at autopsy.

Gross Brain Specimen

Observe the specimen and describe visible neuropathology. Brain with multifocal embolic hemorrhagic infarcts, embolic petechiae, micro-abscesses (note: “petechiae” are the pinpoint spots you can see throughout the brain).

1. What causes petechiae? (what type of vessels are breaking?)
2. If there is bleeding, why is this brain in the “infectious” category instead of the “vascular” category?

Final Summary

The patient had acute bacterial endocarditis of the aortic valve with *Staphylococcus aureus* with systemic embolic phenomenon affecting the kidney, spleen, skin and brain, resulting in infectious microabscesses and petechial hemorrhages. The persistent bacteremia was associated with septic shock and disseminated intravascular coagulation. Bacterial endocarditis can predispose the brain toward developing abscesses, and the autopsy report for this patient noted that several micro-abscesses were found in regions of brain not obscured by the infarct.

Station 3: Trauma

Case: Traumatic Brain Injury Clinical

Presentation:

Phineas Gage was a railroad worker in the mid-1800s. He survived an accidental blasting explosion that involved an iron rod piercing his skull and damage to vast portions of his brain. Although Gage survived, his personality and demeanor were completely altered due to the incident. Before the accident, Gage was known as being responsible, trustworthy, and socially conscious. After his recovery, people noticed he became irreverent, impatient, irresponsible, and prone to using intense profanity (which he had not used before the incident). His friends said they could not recognize any of his old personality.

1. **Let's first review our terminology related to brain injury: First define TBI.** Also, note for clarity that "concussion" is a form of mild Traumatic Brain Injury (mTBI) resulting from a direct or indirect impact to the head or body. Concussion and mTBI are synonymous and interchangeable.
2. What lobes/structures were damaged due to the location of the injury and how would damage to these explain his deficits?
3. Speculate on what white matter tracts were likely damaged.
4. Today, Gage would be described as having an executive function disorder due to the injury. What is executive function?
5. Some patients who sustain multiple TBIs (most studied in football players and boxers) will develop CTE. What does this stand for?
6. Now view the Chronic Traumatic Encephalopathy (CTE) (virtual histology slides). What are the neuropathological features of CTE (what is the name of the protein)?

Gross Neuropathology (CTE only, not useful in concussion or TBI):

7. Observe and note visible neuropathology from photographs of brain specimens at autopsy. What is abnormal?

Station 4: Age-Related/Degenerative

Case: Alzheimer's Disease

Clinical presentation:

The patient is a 67-year-old woman who was evaluated 15 years ago for one to two years of progressive memory problems and cognitive difficulties. Her husband reported that “she would turn on the stove and remove food items out of the fridge but then leave the kitchen and forget the stove was on and leave perishable food on the counter”. The most concerning incident was that she did not arrive to pick her grandchildren up from school, which she had done for several years prior. The children called their grandfather, and the patient was found driving around the neighborhood close to the house and could not remember where she was supposed to be going. Clinical evaluation demonstrated memory and cognitive impairments. As her disease progressed, she experienced worsening anxiety and had difficulty sleeping.

Imaging:

1. What findings are most notable on imaging?

Gross Neuropathology:

2. What are the gross findings for this case?

Time course:

3. What is the typical temporal profile? In other words, what regions of the brain are impacted early in disease vs. later in disease?

Histopathology:

4. What abnormal protein accumulates in the brain? What accumulates intracellularly (inside neurons) vs. extracellularly (outside neurons)?

One more case:

We will next show you a brain from a patient with Parkinson's disease (PD), which is another age-related degenerative disease.

5. Observe the visible pathology in the midbrain. What structure is damaged/lost in patients with Parkinson's disease?
6. What neurotransmitter is thus affected and how does this relate to pharmacological treatments for Parkinson's disease?
7. How can this structure be recognized from a gross anatomical view? What would look different about it in a patient with Parkinson's?

Station 5: Metabolic

Case: Chronic Alcohol Abuse Clinical

Presentation:

A 73-year-old woman presented to the emergency department with her family who noticed worsening gait disturbances and repeated falls with unknown head strike. Upon arrival, she has normal mental status and cognitive function but is complaining of a headache. She denies any personal or family history of neurological disorders. However, her exam was remarkable for postural instability, an ataxic gait, gaze nystagmus (involuntary eye movements) and a tremor in both arms. Her family mentions she has consumed a pint of peppermint schnapps daily for over 2 decades and that she never eats enough. Labs, CT and MRI imaging are ordered.

Alcohol acts directly as a neurotoxin and indirectly on other physiology to damage the brain in both cases of acute and chronic use. Acute intoxication can cause cerebral edema. Chronic abuse leads to brain atrophy and neuronal loss.

1. Observe the MRI and the gross brain images. What area of the brain shows the most visible atrophy?
2. Atrophy of the cerebellum would suggest a loss of which kind of cells? Where in the cerebellum are these found?
3. How does this correlate to the patient's symptoms?

The toxic effects of chronic alcohol can be direct or secondary to nutritional deficits (particularly thiamine = vitamin B1). Using the classical "triad" of symptoms: confusion, ataxia and nystagmus, the patient was diagnosed with Wernicke's encephalopathy: a degenerative brain disorder caused by the lack of thiamine (vitamin B1).

If time, review the circuitry of the cerebellum by drawing it below:

Station 6: Inflammatory

Clinical Presentation:

A 23-year-old female with no medical history develops blurry vision in her right eye that progresses over 4 days. She has pain associated with eye movement and some sensitivity to light. She attributes symptoms to stress. But the pathology is in her right optic nerve. Symptoms resolve over 2 weeks. One year later: She experiences lightning-type zaps when she flexes her neck. This lasts 1 week and then resolves. She follows up with her primary care physician. MRI of the brain is ordered.

1. What do you observe on the MRI?
2. What is the diagnosis?

Histopathology:

3. What does the histology look like?

Gross Neuropathology:

4. Unfortunately, she was lost to follow up and never pursued treatment for her MS. If she died from complications related to long-standing MS (after 50 years of disease), what might her brain look like? Work in groups to review the various MS brain specimens at your table.

Look for these key gross pathological features:

- ✓ Enlargement of ventricles
- ✓ Thinning of corpus callosum
- ✓ Widespread white matter lesions

Station 7: Neoplasms

Case: Meningioma

Clinical Presentation:

The patient is a 62-year-old woman who presented to care for visual changes in her left eye and has experienced severe right-sided headaches involving the back of her head for over a decade. She underwent an MRI as part of the workup.

Imaging:

Observe the MRI image.

1. What is abnormal?

Etiology & Mechanism:

2. Meningioma is a tumor arising from the meninges. What three tissues comprise the meninges?
3. What region of the brain is being compressed? Explain why that would cause headaches and visual changes.

Gross Neuropathology:

4. Observe and note visible neuropathology. Note that this is a different brain than the one presented in this case. Where was the meningioma in this case?

Treatment:

5. Can a meningioma be successfully treated with surgery?

Next, check out the additional tumor specimens & fill out the chart below to review

- ✓ Metastatic Melanoma
- ✓ Glioblastoma
- ✓ Cerebellar Tumor

Name	Cellular Origin – required info	Extra Info
Astrocytoma (highest grade = Glioblastoma)		Common and highly malignant
Oligodendroglioma		Relatively rare, slow growing, most often in frontal lobe
Schwannoma		Classically at cerebellopontine angle and benign
Meningioma		Common and typically benign
Medulloblastoma		Most common malignant brain tumor in childhood, often cerebellar
Ependymoma		Most common in 4 th ventricle
Pituitary Adenoma		Tunnel vision
Pinealoma		Can compress cranial nerve IV

Station 8: Congenital

Case: Partial agenesis of corpus callosum

- 1.** What type of tract is the corpus callosum (commissural, projection, association)? Knowing that, what flow of information is the corpus callosum normally responsible for?
- 2.** Some individuals born with agenesis of the corpus callosum can be prone to suffering from epileptic seizures throughout their life. Can you recall some of the treatments for seizures discussed?

Neuro Glossary

Greek and Latin meanings of many neuroscience words

Prefixes

a = no/not (absence of)
Angi = blood vessel
Dys = impaired or difficult
Epi = upon/above/outside of
Hemi = half
Hyper = over or increased
Hypo = under or reduced
Ipsi = same
Para = alongside of/abnormal
Peri = around/surrounding
Sub = beneath

Suffixes

-algia = pain (i.e., neuralgia)
-arthria = joint or articulation
-geusia = taste
-itis = inflammation (i.e., myelitis)
-kinesia/-kinesis = movement
-metria = measurement
-oma = tumor (i.e., meningioma)
-opsia = vision
-osmia = smell
-pathy = suffering/disease/disorder (i.e., neuropathy)
-phagia = swallowing (or eating)
-phasia = speaking
-plegia = paralysis (i.e., quadriplegia)
-tonia = muscle tension

Terms

Accumbens = lie down
Acusis = hearing
Afferent = to carry (towards)
Ageusia = loss of sense of taste
Agnosia = no knowledge
Agraphia = inability to write
Akathisia = unable to sit still
Akinesia = no movement
Amacrine = no long fiber
Ambiguus = doubtful
Amnestic = memory loss
Amygdala = almond
Anarthria = no articulation (meaning no articulation of language/speech - inability to make sounds due to paralysis of muscles of tongue, vocal cords, or larynx)
Analgesia = no pain

Anesthesia = no sensation
Aneurysm = widening
Anosmia = without sense of smell
Aphagia = no eating; inability to swallow
Aphasia = no language/ speech
Aqueduct = water canal
Arachnoid = spider web-like
Arbor vitae = tree of life
Astrocyte = star-like cell
Ataxia = no order/arrangement (unsteady/swaying walk, often with feet planted widely apart)
Atrophy = wasting of body tissues
Axon = axis, axle
Auditory extinction = Inability to perceive auditory stimuli on the contralesional side when presented with simultaneous stimuli
Basilar = base
Bouton = button
Brachium = arm
Bradykinesia = slowness of movement (by reduced speed and progressive decrement in amplitude during repetitive movements)
Callosum = hard, tough
Caudal = towards the tail
Cauda Equina = horse tail
Caudate = tail
Cephalon = relating to the head (i.e., brain)
Cerebellum = little brain
Cerebrum = brain
Coeruleus = blue
Cervical = neck
Chiasm = a crossing
Chorea = dance
Choroid = membrane-like
Cingulate = belt (i.e., the gyrus that forms a belt around the corpus callosum)
Cistern = reservoir
Clastrum = barrier
Colliculus = little hill (i.e., those in the tectum of midbrain)
Commissure = joining together (i.e., bundle of axons in CNS crossing hemispheres)
Contra = opposite
Conus = cone
Corona = crown
Corpus = body
Cortex = bark (outer layer)

Cuneate = wedge
Decussation = crossing
Dementia = away from mind
Dendrite = tree
Dentate = notched, having teeth
Diplopia = double vision
Dorsal = towards the back
Dura = hard
Dysarthria = impaired articulation (slurred or slowed speech that is difficult to understand due to weakness of muscles of tongue, vocal cords or larynx).
Dysphagia = impaired swallowing
Dysdiadochokinesia = an impaired ability to perform rapid, alternating movements
Dysgeusia = impaired perception of taste (often persistent bad taste)
Dyskinesia = impaired or abnormal involuntary motion/movement
Dysmetria = impaired measurement (misjudgment of distance to an object)
Dysosmia = impaired sense of smell
Dystonia = twisting movements/postures
Efferent = carry out (away)
Emboliform = plug-like
Falx = sickle
Fasciculus = little bundle
Fasciculations = involuntary muscle twitches of a single motor unit
Filum = thread
Fimbria = fringe
Fissure = cleft or slit
Flocculus = tuft of wool
Folium = leaf
Foramen = opening (or hole)
Fornix = arch
Fossa = trench
Fovea = pit, depression
Funiculus = little cord
Fusiform = spindle-shaped
Ganglion = knot, swelling (collection of somas in PNS)
Gelatinosa = gelatinous
Genu = knee
Glia = glue
Globus pallidus = pale ball
Glossal = tongue
Gracile = slender
Gyrus = ring, circle (refers to bump on cortex)
Habenula = rein
Hemi = half
Hemianesthesia = loss of sensation on one side of body
Hemianopsia = loss of vision on one half of the visual field

Hemineglect = unawareness of one side of space
Hemiparesis = muscle weakness on one side of body
Hemiplegia = paralysis on one side of body
Hippocampus = sea horse
Homonymous hemianopsia = a visual deficit that is present on the same side of both eyes
Hyperkinesia = increase in muscular activity or movement
Hyperreflexia = exaggerated reflexes due to loss of control from higher brain centers; typically indicates UMN damage
Hypokinesia = decrease in muscular activity or movement
Hyporeflexia = decreased reflexes due to loss of innervation of the lower motor neuron onto the muscle.
Hyposmia = reduced or diminished sense of smell
Hypotonia (flaccidity) = reduced tone of muscles, limpness
Infundibulum = funnel
Ictal = literally means "blow" and refers to a seizure
Insula = island
Interictal = between seizures
Lamina = layer, thin plate
Lemniscus = band or ribbon
Lenticular = lens-shaped (i.e., the putamen + globus pallidus)
Limbic = border
Locus = point or spot
Mater = mother
Medulla = innermost, marrow
Meninges = membrane
Myoclonus = involuntary movements caused by muscle contractions (positive) or sudden loss of muscle tone (negative)
Neuron = nerve
Nerve = bundle of axons in PNS
Nigra = black
Nucleus = collection of somas in CNS
Nystagmus = involuntary, rapid movements of the eyes
Oculomotor = eye movement
Parosmia = altered perception of smell in the presence of an odor, usually unpleasant
Paroxysmal = sudden attack of a disease
Peduncle = stem-like/stalk (i.e., a bundle of axons that travel together)
Pellucidum = transparent
Phantomia = perception of smell without an odor present
Pia = soft
Pineal = pinecone
Pinna = wing
Piriform = pear-shaped

Plexus = a braid
Pons = bridge
Post-ictal = after a seizure
Ptoxis = drooping/falling of upper eyelid
Pulvinar = cushion, pillow
Putamen = shell
Quadrigenina = four twins
Ramus = branch
Raphe = seam
Rectus = straight
Restiform = rope-like
Reticular = net-like
Rigidity = constant muscle stiffness and increased resistance to passive movement across a joint. The increased muscle tone/resistance is felt uniformly throughout the range of motion when, for example, the examiner tries to move the neck, arm or leg when the patient is completely relaxed.
Rostral = towards the beak (nose)
Rubro = red
Sclera = hard
Sella turcica = Turkish saddle
Septum = wall, partition
Sinus = a hollow space
Solitarius = solitary, alone
Soma = body
Spasticity (spastic paralysis) = abnormal muscle stiffness and increased tone (prolonged contraction of entire muscle groups) that is velocity dependent, meaning the faster a muscle is passively moved, the more resistance to movement will be felt.

Spine = thorn
Splenium = bandage
Stellate = star
Strabismus = deviation of eye movement
Striatum = striped (i.e., the caudate + putamen)
Substantia = substance
Sulcus = groove
Tardive = characteristic lesion is late in appearing
Tectum = roof
Tegmentum = covering
Temporal = time
Tentorium = tent
Thalamus = inner chamber
Trabecula = little beam
Tract = bundle of axons in CNS
Tremor = oscillation, tremble, shiver
Trigeminal = three twins
Trochlear = pulley
Tubercle = swelling
Uncus = hook
Vagus = wandering
Velum = covering
Ventricle = small cavity
Vermis = worm
Vesicle = blister, bladder
Visual extinction = inability or difficulty perceiving visual stimuli on the contralesional side when presented with simultaneous stimuli
Vitreous = glassy

Adapted from:

- <https://faculty.washington.edu/chudler/neuroroot.html>
- https://www.wikidoc.org/index.php/List_of_medical_roots_suffices_and_prefixes
- <https://neurology.uams.edu/neurology-clinical-services/movement-disorders/glossary-of-movement-disorder-terms/>
- <https://www.aans.org/en/Media/Glossary-of-Terminology>