Outline

- Overview of neuroanatomy
- What is "functional" neuroanatomy?
- Neuroanatomy in dementia?
Gross Anatomy

- Frontal Lobe
- Temporal Lobe
- Parietal Lobe
- Occipital Lobe
- Cerebellum
Gross Anatomy-Midline

- Cingulate Gyrus
- Frontal Lobe
- Corpus Callosum
- Brain Stem
- Parietal Lobe
- Occipital Lobe
- Cerebellum
- Brain Stem
Landmarks

- Central Sulcus
- Sylvian Fissure
Landmarks

Longitudinal Fissure

Central Sulcus
Structural Imaging Methods

- CT Scan
- MRI Scan
  - T1
  - T2
  - FLAIR
  - DWI/ADC
  - Not going to talk a lot about these here
Brain CT

- Bone-white
- CSF-dark
- Gray matter lighter than white matter
MRI Cuts

Coronal

Axial or Transverse

Sagittal
MRI sequences

T1

T2

FLAIR
T1 axial cut

- Frontal lobe
- Caudate
- Putamen
- Anterior Commissure
- Insular cortex
- Cerebellum
- Occipital lobe
T2 axial cut—a bit lower

- Eyeball
- Temporal Lobe
- hippocampus
- Brain Stem
Sagittal Cut-Midline

- Frontal lobe
- Corpus callosum
- Occipital Lobe
- Cerebellum
Coronal FLAIR

- Lateral ventricle
- Temporal lobe
- Hippocampus
Functional Neuroanatomy

A bit of history
- Paul Broca/Karl Wernicke
- Phineas Gage case
- Genesis of modern neuropsychology
  - Localization of function by employing behavioral tests—in other words, looking at a person’s cognitive function and dysfunction.
  - One proviso: *though cognitive capacities are somewhat localizable, there is also widespread activation of numerous brain regions.*
Some *general* basics

- In the brain (cerebrum):
  - Anterior is associated with action/behavior
  - Posterior is associated with sensation/perception
    - Like the spinal cord
  - Left side of brain controls right side of body
  - Right side of brain controls left side of body
    - Unlike the spinal cord
More general basics

- Left side tends to be associated with verbal functions
- Right side tends to be associated with nonverbal functions.
Behaviors the Brain controls

- Movement
- Sensation
- Learning/memory
- Language production
- Comprehension
- Visual perception
- Visual integration
- Constructional skills
- Executive functions
- Emotional regulation
- Sleep
- Breathing
- OK—all behaviors controlled by the brain
Cerebellum

Involved in:
1. Motor programming, coordination, postural regulation
2. Cognitive
   1. Executive functions-abstract reasoning, verbal fluency, & planning
   2. Memory and Learning
   3. Spatial abilities
   4. Attention/processing speed
   5. Emotional regulation
*See with alcohol intoxication
1. Loss of tactile sensation or thalamic pain syndrome
2. Memory impairments- anterograde and retrograde
3. Disturbance of time sense
4. Lack of appreciation of deficit
5. Problems with concept formation/executive functioning
6. Emotional flattening/lack of spontaneity or drive

*May see involvement of the thalamus with Wernicke-Korsakoff syndrome
Basal Ganglia

1. Made up of caudate, putamen, globus pallidus
2. Cognition to action—generally characterized by abnormal movements at rest.
3. Procedural memory
4. Trouble with cognitive flexibility
5. Left sided—can produce language disturbance
6. Can produce dramatic personality changes

*Huntington’s disease, Parkinson’s Disease, Wilson’s Disease
Limbic System-Amygdala

1. Involved in emotional processing and learning
2. Hypersexuality
3. Diminished aggression
4. Processing of fear expressions—both perceiving and experiencing

*Kluver-Bucy Syndrome*
Limbic System-Hippocampus

1. Highly interconnected brain structure
2. Very important in memory formation
3. Somewhat modality specific—right: spatial, left: verbal
Occipital Lobes

1. Blindsight-loss of vision, but may be responsive to visual stimuli
2. Anton’s syndrome-denial of blindness
3. Visual agnosia
4. Prosopagnosia
5. Numerous other visual disorders
6. Possible visual field cuts
Parietal Lobes

Either side:
1. Constructional defects
2. Short-term memory defects
3. Apraxia—ability to perform purposeful movements
4. Disrupted guidance of movements

Left side:
1. Fluent aphasia
2. Loss of semantic features of language
3. Disruption of reading, writing, math
4. Gestural defects
5. Gerstmann syndrome (R/L disorientation, acalculia, agraphia, impaired finger localization)

Right Side:
1. Constructional defects
2. Dressing apraxia
3. Sensory neglect
4. Lost appreciation of facial expressions
Temporal Lobes

Left Side:
1. Aphasia
   1. Posterior-fluent
   2. Anterior-Anomia
2. Verbal memory functioning (medial)
3. Odor Perception

Right Side:
1. Amusia
2. Trouble organizing plans
3. Nonverbal memory functioning (medial)

Also:
1. Emotional disorders
2. Visual field cuts

*Though hippocampus involved in processing newly learned info, it seems that temporal cortex is involved in housing old information.
Frontal Lobes

1. Movement, coordination/planning of movement
2. Language production-nonfluent aphasia
3. Executive functioning-the brain’s supervisor
4. Verbal fluency
5. Online processing of information-working memory-divided attention
6. Emotional dampening/abulia
7. Impulse control, behavioral regulation
8. Odor discrimination
9. Frontal amnesia-not memory problem per se, but inability to spontaneously generate an answer
10. May be florid confabulation
Frontal Lobes, continued

- Apathy, carelessness, and poor judgment
- Diminished abstract reasoning capacity, concrete tendencies
- Perseveration/behavioral rigidity
- Defective self-monitoring, self-awareness
- Problems starting tasks, stopping tasks
White Matter

• Connects regions of the brain together
• Can be associated with problems in attention and diminished processing speed
Typical radiology report

- It seems that every radiology report for dementia care reads: “*Age related cerebral atrophy. Small vessel ischemic disease typical for age. Clinical correlation recommended.*”

- What are we supposed to do with that?
Beware the imaging bias-McCabe and Castel (Cognition, 2008) demonstrated that having a picture of a brain rendered findings more credible.

Mechanic analogy

We need the structure/function correlation

We will spend the rest of the time looking at neuroanatomy, cognition, and specific dementias.
Atrophy

- Reflects the loss of brain tissue—cortical, subcortical, or deep.
  - May lose cortical cell bodies with associated axonal degeneration.
  - Selective atrophy of the white matter following perivascular small vessel insults.

- (potentially) reversible atrophy
  - Alcoholism
  - Anorexia/Starvation
  - Chemotherapeutics
  - Dehydration
  - Marijuana
  - Radiation
  - Steroid use
Cerebral (supratentorial) Atrophy

- Alzheimer disease
- Frontotemporal dementia
- Pick’s disease
- Cruetzfeld-Jakob disease
- Parkinson’s disease
- Multisystem atrophy
- Progressive Supranuclear Palsy
- Cortical-basal ganglionic degeneration
- Lewy Body Dementia
- AIDS
- Multi-infract dementia (vascular dementia)
- Amyotrophic lateral sclerosis
Alzheimer’s disease—behaviorally

- Progressively worsening anterograde memory, eventually joined by retrograde memory and no benefit from reminders
- Later
  - Diminished abstracting capacity/executive dysfunction
  - Anomia
  - Apraxia
  - Constructional deficits
  - Aphasia
- Relatively preserved motor/sensory functions
A 1991 study at the University of Iowa examined the accumulation of plaques and tangles in the Alzheimer’s brain, finding a preponderance of accumulation in the ventromedial (entorhinal cortex) and anterior temporal lobes.

• Disease progression tends to progress from entorhinal cortex, to hippocampus, to cortex.

• As mentioned earlier, the hippocampus and medial temporal cortices are essential in laying down new memories.

Hippocampal atrophy
Advanced Alzheimer’s

- Global atrophy
- Hippocampus is *gone.*
Sensorimotor sparing
**Frontotemporal Dementia**

- Atrophy primarily affecting frontal and/or temporal lobes
  - 3 types:
    - frontal variant (40%)
    - semantic dementia (40%)
    - progressive nonfluent aphasia (20%)

- Differentiation from DAT difficult
  - May show more frontotemporal atrophy in frontal variant
  - Semantic dementia may show atrophy of temporal pole and inferolateral gyri
  - Progressive nonfluent aphasia may show perisylvian atrophy
Cortical atrophy in FTD

Frontotemporal Dementia (FTD)

Semantic Dementia (SD)

Progressive Non-Fluent Aphasia (PNFA)
Frontal variant behaviorally

- Generally begins with personality change
  - Disinhibition
  - Mood changes
  - Perseveration
  - Coarsening of behavior
  - *ask if they are craving more sweets*

- Progresses to dementia
Frontal variant-MRI

Normal

Frontotemporal dementia
Semantic Dementia-behaviorally

- Difficulty generating familiar words
- Often a loss of semantic knowledge
- However, speech remains fluent
- Trouble recognizing familiar faces
Semantic Dementia-MRI
Non-fluent primary progressive aphasia

- Hesitant, effortful speech
- Trouble producing speech
- Person eventually becomes mute
- May have difficulty swallowing
- May make paraphasic errors in speech
- Anomia
PNFA MRI
Pick disease

- Characterized by memory loss, confusion, speech dysfunction, social coarsening, apathy and abulia
- Imaging shows anterior temporal lobe predominance + inferior frontal lobe changes.
Pick Disease
Posterior cortical atrophy

Characterized by

- Changes in vision (complain they need new glasses)
- Difficulty recognizing faces and objects
- Diminished spatial awareness
- Trouble perceiving color
- Problems with writing
- Problems with skilled movements
PCA-MRI
Vascular Dementias

- Multiple areas of white matter infarction
  - Hypodense on CT
  - Hyperintense on T2 and FLAIR MRI
- Severe deep gray matter lacunar disease
- Binswanger’s disease
  - May exclusively involve white matter
- CADASIL
  - Severe lacunar disease
  - Subcortical white matter ischemic changes
Vascular Dementia-behaviorally

- Stepwise deterioration
- Problems with memory, but often retained recognition
- Problems with abstract reasoning/executive functioning
- Notably slowed processing speed, diminished attention
- Affective changes-depression, anger, pseudobulbar affect
Vascular disease-MRI
Lacunar Infarcts - deep gray matter
Normal Pressure Hydrocephalus

- Wet, wacky, and wobbly.
- Enlarged ventricles—particularly temporal horns.
- Only 50% of patients improve clinically after shunting.
- Expansion of lateral ventricles leads to strains on white matter and clinical symptoms.
Normal Pressure Hydrocephalus
Transependymal Flow
Deep Gray Matter Disorders

- Huntington’s disease
- Wilson’s disease
- Parkinson’s Disease
Huntington’s disease

- Selective atrophy of the caudate nuclei
  - Frontal horns of lateral ventricles are dilated and rounded.
  - Frontal atrophy may be present
  - Increased signal intensity in the globus pallidus and putamen may appear
In HD, there tends to be changes in:

- **Cognition**: Memory, including procedural memory, executive functioning, processing speed.
- **Behavior**: disinhibition, depression, apathy
- **Movement**: Motor restlessness and chorea
Wilson Disease

- **CT**
  - Atrophy of caudate and brainstem
  - Hypodense basal ganglia and thalami

- **MRI**
  - Hyperintense signal in deep gray matter
  - T1 may show bright hypothalami
  - T2 may show symmetric increased abnormalities in outer rim of putamen, thalami, and globus pallidus
Wilson’s Disease-behaviorally

- Movement disorder-dystonia, chorea, tremor, or parkinsonism
- Personality change-lability, disinhibition, bizarre behavior
- Perhaps psychosis
- May produce dementia over time
Wilson Disease MRI
Recommended Resources

Neuroradiology: The Requisites, Yousem & Grossman

Neuroanatomy Through Clinical Cases, Blumenfeld

The Mental Status Examination in Neurology, Strub & Black