Frontal Lobes (~1/3 of cortex)

- Motor control, including speech
- Higher cognitive or executive functions
- Self-regulation (behavioral inhibition, sensitivity to social cues, conscience)
- Initiative

Motor Homunculus (on right)

Anatomy

- The frontal lobe is heavily interconnected with:
  - basal ganglia & other components of the motor system
  - all other lobes of cortex
  - limbic system
- This anatomy should allow you to predict something about the frontal lobe’s functions

Evolution of Frontal Lobe

- From a motor planner/organizer to a higher level behavioral/strategic planner/organizer
- Frontal processing moved from being totally in response to environmental stimuli to being abstract, anticipatory, imaginative, & able to draw upon stored memories & emotions.
Frontal Lobe “Executive Functions”
- Mental representation of the world; working memory
- Forming goals, anticipating consequences
- Considering options; applying knowledge & past emotions ("somatic markers") to make choices/decisions
- Choosing & initiating goal-directed behaviors
- Self-monitoring your responses
- Correcting/adapting behavior in response to feedback or changes in context
- Persistence towards goal despite distraction

Some Causes of Frontal Damage
- traumatic brain injury
- vascular lesions (stroke)
- neoplasms (tumors)
- degenerative diseases that affect frontal pathways (Alzheimer’s, Parkinson’s, Huntington’s, Pick’s disease) and cause dementia
- decreased frontal activity in schizophrenia and major depression

Effects of Frontal Damage
- If motor regions are damaged:
  - Precentral gyrus – decreased fine movements, speed, & strength
  - Premotor region – poor programming of movements
  - Frontal eye fields – poor voluntary gaze, can’t move eyes to visual field contralateral to lesion
  - Vicinity of Broca’s area – impaired production of speech & sign language
- May have loss of sense of smell with orbitofrontal damage

Effects of Prefrontal Damage - Impaired Executive Functions
- Knowledge/intelligence may seem intact (e.g. IQ) but it is not applied effectively
- Less ability to consider options; reduced flexibility, tendency to perseverate
- Difficulty using environmental cues to regulate or change behavior
- Decreased spontaneity, initiative, talking; may appear lazy, unmotivated, depressed (more common after left damage)

Decreased Inhibition
- Problems inhibiting incorrect/ineffective responses & switching to a new strategy
- Perseverates; not responsive to feedback or changes in environment
- Violates rules; takes more risks; less inhibition of emotional responses
- Decreased social inhibitions as well; disinhibited personality; impulsive
- More common after right damage
Orbitofrontal Cortex: Seat of our “Theory of Mind”?

- Ability to decode & be sensitive to others’ states of mind; social understanding
- Conscience; empathy; emotional intelligence; affective decision-making
- Behavior influenced by ‘gut feelings’ or ‘somatic markers’

The Case of Phineas Gage

- Phineas had been a responsible, mild-mannered, church-going family man before his accident.

MRI Reconstructions of Damage

- After: lack of tact, restraint, empathy; decreased conscience, immature, coarse, lack of social graces, irresponsible.
- More common after orbitofrontal or right frontal damage – disrupts link allowing emotional “tags” to influence decisions. May cause Capgras syndrome (think others are imposters).
- Right frontal damage can also impair sense of humor. [Link](http://www.youtube.com/watch?v=sUUP7IYTlqI)

Decreased Temporal or Recency Memory

- Damage to dorsolateral frontal cortex impairs working memory for recency, order, and source or contextual info
- Could affect problem-solving, planning and impair systematic, organized behaviors

Some Neuropsych Tests Used to Test for Frontal Lobe Deficits

- Wisconsin Card Sorting Test; Stroop Test
- Word Fluency Test; Design Fluency Test
- Visual Search Test
- Motor strength, speed and sequencing tests
- Tests for aphasia (language problems); anosmia (loss of smell)
Wisconsin Card Sort Task

Electronic Stylus Maze

Stroop Test

Tower of Hanoi Puzzle
Tests Planning Ability

Are the auditory feedback cues indicating an error used to correct path thru maze?

Try your hand at a little Tower of Hanoi
Luria's View of Sensory Processing Deficits

- Primary sensory cortex damaged: Basic sensory awareness
- Secondary sensory cortex damaged: Apperceptive agnosia (impaired recognition of what is sensed because you don’t perceive the “Gestalt”). Can’t copy or match stimulus either
- Tertiary (association) cortex damaged: “Associative agnosia” - deficits in integrating input with other modalities, memory, language, semantic knowledge, etc.; difficulties in naming/using/applying sensory information

Example:
3 levels of Visual Processing

The Parietal Lobe

- Primary and secondary somatosensory cortex – if damaged may experience:
  - Astereognosis – can’t recognize by touch
  - Simultaneous extinction- can’t feel 2 stimuli at once
  - Asomatognosia - loss of body image
  - Finger agnosia: can’t identify/localize fingers
  - Anosognosia – denial of impairments/illness
- Multimodal association cortex (“parieto-occipital-temporal crossroads”)
- Both provide input to motor regions to guide movements [youtube video]

The Parietal Lobe Regions

- Primary and secondary somatosensory cortex – if damaged may experience:
- Multimodal association cortex (“parieto-occipital-temporal crossroads”)
- Both provide input to motor regions to guide movements

Laterization of Parietal Functions

- Left parietal: sensory & integrative processing important for normal language and math
- Right parietal: sensory & integrative processing related to the use of spatial information in perceptual, cognitive & motor behaviors
**Left Parietal Damage**

- Anomia: not able to name things
- Alexia: not able to read
- Impaired grammar
- Agraphia: not able to write
- Acalculia: loss of math abilities
- Impaired left/right discrimination
- Unable to name/recognize fingers
  (these last 4 are known as Gerstmann’s Syndrome & may follow a L. parietal stroke)

http://www.youtube.com/watch?v=9_H2EDf37is&feature=PlayList&p=9C0952DCB4CEA096&playnext_from=PL&playnext=1&index=8

**Right Parietal Damage**

- Contralateral sensory neglect
- “Constructional” apraxia-can’t assemble, build, draw, construct because of visuomotor/spatial impairment
- Perceptual processing impaired more by right parietal damage
- Dressing apraxia (other ideomotor apraxias are associated with left parietal)
- Poor map reading/drawing

**Tests for neglect (line bisection and drawing clock)**

- Asked to copy figures, patient with neglect ignores contralateral visual field

**Some Tests For Parietal Function**

- 2 point discrimination (tactile sensation)
- Seguin-Goddard Form Board (tactile recognition & matching)
- Perceptual tests
  - Copying
  - Gollin Incomplete Picture or Mooney Closure Test
  - Unusual Views Test
  - Match by Function Test; demonstrate or describe
- Semmes extrapersonal orientation test (map reading)
- Kimura box test (motor learning)
**Shape Perception**

Blind-folded subject
Matches shapes to board, then draws board from memory
(Picture is a child’s toy similar to the form-board test.)

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**Copying Problems**

A
B
X
O
3
4

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**Rey-Osterrieth Complex Figure**

- Copy (test of perception)
- Draw from memory (test of visual memory)

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**Incomplete- Pictures Task**

Set 1
Set 2
Set 3
Set 4
Set 5

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**Block Design- WAIS**

Figure 22. ‘Mixed figures,’ used in the assessment of visual agnosia
• Pathway for conscious visual experience
• Also connections to midbrain visual areas for reflexes

Higher Visual Processing

Damage or Dysfunction of Visual Pathways or Cortex

ZZigzag lines of disrupted vision during a migraine aura
Scotoma – loss of a portion of the visual field due to brain damage
Homonymous hemianopsia

Temporal Lobe

Temporal Gyri
Medial Temporal Lobe

- Green = parahippocampal gyrus
- Yellow = lingual gyrus
- Pink = fusiform gyrus

Recall the limbic components (hippocampus and amygdala within temporal lobe)

Temporal Lobe Regions

- Auditory area - superior temporal gyrus (primary & secondary auditory cortex)
- Complex association cortex - middle & inferior temporal gyri (links audition-vision-memory system)
- Limbic region - medial temporal cortex (personality?) & amygdala (emotion) & hippocampus (memory storage process)

Temporal Lobe Epilepsy

- Seizures may cause cognitive or emotional symptoms (deja vu, jamais vu, out-of-body exp, forced thinking, strong emotion, hallucinations, hearing voices, speech changes)
- Temporal lobe seizures may trigger aggression, even murder
- Associated with “temporal lobe personality traits”

Temporal Lobe Personality

- Qualities that may be associated with TL abnormalities:
  - humorlessness; paranoia; feel threatened
  - overemphasis on details/minutiae; verbose
  - egocentric, “sticky” personality, sense of destiny
  - strong religiosity, focus on good vs evil
  - aggressive outbursts (temporal lobe pathology has been found in brains of several mass murderers and has been used as a defense by others)

Ramachandran Part 1 & 2 & 3

- http://www.youtube.com/watch?v=wlFi6IV42Ag
- http://www.youtube.com/watch?v=DBbaE00hs
- http://www.youtube.com/watch?v=sUUP2iYTLol

- Visual agnosia
- Prosopagnosia
- http://www.youtube.com/watch?v=XLGXAi5pN00&feature=related
- Capgras syndrome
**Effects of Temporal Damage**

- Auditory impairment; word deafness, Wernicke’s aphasia
- Visual agnosias; prosopagnosia; impaired selective attention
- Impaired storage of new memories
- Emotional & personality changes

**Visual Agnosia**

- Patient could copy but could not identify the pictures being copied

**Broca’s (aka Motor or Anterior) Aphasia**

- Damage in vicinity of inferior frontal gyrus
- Patient not articulate, not fluent
- Speech slow, difficult, & much reduced (“telegraphic speech” - nouns & verbs)
- Comprehension relatively intact, except for grammatical words, endings, and meaning which relies on word order (recall frontal lobe involvement in sequencing & temporal memory)

http://www.youtube.com/watch?v=f2iMEbMnPm

**Recognizing Faces**

Inability to recognize faces = prosopagnosia. May be accompanied by inability to make other fine visual discriminations (makes of car, types of flowers, etc.)

**Wernicke’s (aka Sensory/Receptive or Posterior) Aphasia**

- Temporal lobe language area damaged
- Speech is fluent, but nonsensical
- Reduced comprehension of language
- Anomia, confusion of phonemes, paraphasias (use wrong word or made up word)
Dementia: More Than Memory Loss

- Cognitive deficits (in memory, reasoning, understanding, language, perception, organization & control of behavior) not due to clouded consciousness
- Impaired social/occupational functioning
- Decline from previous level of functioning
- Over 100 causes; about 30% of dementias are reversible (due to endocrine problems, vitamin deficiency, medications, CSF pressure, etc.)

Most Common Dementias

- ~50% Alzheimer’s Disease
- ~20% Vascular dementias
- ~10%-20%? Dementia with Lewy Bodies & PD
  - (up to 40% those with AD also have Lewy bodies)
- ~10% Fronto-temporal dementias
- Other diseases causing dementia (AIDS, Huntington’s, Creutzfeld-Jakob and others); Dementia pugilistica
- Each dementia associated with some distinctive neural and behavioral symptoms

Alzheimer’s Disease

Diffuse progressive degeneration in cortex, hippocampus, amygdala, ACh nucleus basalis of Meynert associated with increasing numbers of neurofibrillary tangles of abnormal tau proteins inside of neurons & abnormal plaques of amyloid proteins outside of neurons.

Misdiagnosis not infrequent.

Current drug treatment in mild-mod. Cases is aimed at boosting ACh but is not as successful as drugs which boost DA in Parkinson’s
- Cognex (tacrine)
- Aricept (donepezil)
- Exelon (rivastigmine)
- Reminyl (galantamine)

In mod-severe cases may use Namenda (memantine) to block excitotoxicity of excess glutamate.
Vascular Dementias (several types)

- Most often seen in those with risk factors for stroke or cardiovascular problems, but can also have a genetic or disease-related cause (lupus, vasculitis) or profound low blood pressure
- May occur suddenly or gradually
- May not progress
- May be associated with more focal deficits
- Less likely to affect personality, emotional control

Lewy Body Dementia (LBD)

- About 30%-50% of Parkinson’s disease patients develop dementia. In about ¼ of these cases it is Lewy Body Dementia, in the others it is Alzheimer's.
- Cortex & substantia nigra affected
- LBD produces similar symptoms to AD but more day to day fluctuation in cognitive function with variations in alertness & attention, visual hallucinations, Parkinson's motor signs
- DA-promoting Parkinson's drugs or Alzheimer’s drugs may help

Lewy bodies contain abnormal protein alpha-synuclein. Lewy bodies may also be seen in Alzheimer’s disease.

Fronto-temporal Dementia

- 70% show unilateral degeneration, often in dom. hemisphere
- Abnormal tau protein & neurofibrillary tangles
- One variety (Pick's disease) assoc. with swollen neurons and “Pick bodies”
- *Appears earlier (40-60), may run in families
- *Loss of restraint & personality changes before memory problems;
  spatial abilities preserved

Folstein mini mental status exam

1. Name
2. Age
3. Sex
4. Address
5. Marital status
6. Occupation
7. Religion
8. Educational level
9. Nationality
10. Whether able to write
11. Whether able to read
12. Whether able to calculate
13. Whether able to understand
14. Whether able to speak
15. Whether able to hear

1. Orientation
   a. Time
   b. Place
   c. Person
2. Registration
3. Attention & calculation
4. Recital
5. Language
6. Writing
7. Comprehension
8. Construction

* Folstein mini mental status exam