The Brain

Brain Development

Overview

18 days after conception

Primitive streak

- Outer layer of embryo thickens
- Ectoderm forms a plate
- Edges curl up
- Make a neural tube

Neural tube

- Cells inside tube become neurons & glial cells
- Closed tube
- Tube with 3 bulges

1. Forebrain

- Cerebral cortex
- Basal ganglia
- Limbic system
- Thalamus
- Hypothalamus

2. Midbrain

- Superior colliculi = vision
- Inferior colliculi = hearing
- Homeostasis & reflexes

3. Hindbrain

- Medulla oblongata
- Cerebellum
- Pons

Phases

1st Phase

- Symmetrical Division
- 2 identical founder cells
- Radial Glial Cells
- Spread out like tree
- Neurons climb tree to their proper position

2nd Phase

- Asymmetrical Division
- About 3 months
- Divide into neuron & founder cells
- End of cortical development
- Founder cells receive signal (cell death)
Choice 1: Anatomy

Two fists, crossed arms
4 lobes:
   Occipital
   Parietal
   Temporal
   Frontal

Frontal lobes
1. Primary Motor Cortex
2. Pre-motor Cortex

  Supplemental motor
  Connect directly to spine
  Help, don’t know how balance?
  coordination?

Prefrontal Cortex
  Connects direct to spine
  Not fully understood
  Planning?
  Spatial guidance?
  Actions of others?

Posterior Parietal Lobe
3. Pre-frontal Cortex

  Dorsolateral
  fairly new
  develops til 30
  connects to basal ganglia+
  working memory
  damaged in Schiz
  drug abuse
  alcohol
  ?

  Orbitofrontal
  above the eyes
  decision making
  inhibit bad behavior
  gambling
  OCD

  Ventromedial
  risk
  fear
  decision making
  regulation of emotion
Anterior Cingulate Cortex
  Collar around the corpus colosum

Other Regions
  Lingual gyrus
  Fusiform gyrus
  Hippocampal gyrus
  Hippocampus
    Damage to one side = retrograde amnesia
    Damage to both sides = anterograde amnesia

Cerebellum
  Lateral Cortiospinal Tract
    Primary Motor Cortex
    Red nucleus of the midbrain
    Go to medulla oblongata
    Cross contralateral
    Medulla pyramids

Choice 2: Connections
  When neurons reach home
    Connect with each other
    Grow dendrites & axons
    Synapse formation
    Synapse elimination

5 Steps of Neurons

1. Proliferation
   Production of new cells
   Cells along the ventricles divide to become neurons and glia.

2. Migration
   Primitive neurons find their spots
   Chemicals guide cells

3. Differentiation
   Neurons get axon & dendrites
   Makes them different
   Axon grow before dendrites
   During migration

4. Myelination
   Glia cells produce myelin sheaths
   first in spinal cord
   Then in brain
   Lasts til about 30

5. Synaptogenesis
   Continues throughout life
   Forming synapses
Age & Neurons
   Stem cells
   Nose cells always undifferentiated
   Periodically divide & make new olfactory cells

Pathfinding
   Getting axons to their spots
   Chemical Path-finding

Weiss (1924)
   grafted extra leg to a salamander
   axons grew, moved in sync with other legs
   Theory:
   nerves attach to muscles randomly
   variety of messages are sent
   each one tuned to a dif. muscle

Sperry (1943)
   Severed optic nerve axons
   Rotated them 180°
   Grow back to their original target locations in midbrain

Chemical gradients
   Axons attracted by some chemicals, repelled by others
   TOPDV protein is 30x more concentrated in dorsal retina than ventral retina
   axons
   Highest connect to highest
   Lowest concentration axons connect to lowest

Neural Darwinism
   During development
   Synapses form randomly
   Selection process keeps some and rejects others
   Chemical guidance
   Neurotrophic factors
   Muscles & synapse survival
   produce & release NGF (nerve growth factor)
   Not enough NGF, axons degenerate and cell bodies die
   Neurons automatically die
   don’t make synaptic connection
   Apoptosis = cell death
   Neurotrophin
   promotes survival & activity
   Similar to NGF
   BDNF
   brain-derived neurotrophic factor
   most abundant neurotrophin in cortex
   Make more than enough
Neurotrophins are also used in adult brains
More axon & dendrite branching
Deficiencies of neurotrophins lead to cortical shrinking and brain diseases

Cortex Differentiation
Different parts of cortex
Different shapes
Shape and functions depend on input received
Transplant immature neurons
Become like neighbors
Transplant later
Some new, some old attributes
Experience fine tunes
Redesign our brain to fit (within limits)
Enriched environments
Thicker cortex
More dendritic branching
Best enrichment = activity

Transfer
Far transfer = do well in one, do well in other tasks
Near transfer = practice task, do better on that task only
Train the brain – doesn’t work

Neural Plasticity
Blind from birth
better at discriminating objects by touch
increased activation in occipital lobe (vision) doing touch tasks
Use occipital cortex for Braille (sighted people don’t)
Concept of straight
Learn to read as adults
More gray matter in cortex
Thicker corpus callosum

Music Training
Pro musicians
Bigger temporal lobe (30%)
2x greater response to pure tones (in auditory cortex)
Violin players
larger area devoted to left fingers in the postcentral gyrus

Musician’s Cramp
Practice too much
Fingers get jerky, clumsy & tired
expanded representation of each finger overlaps neighbor

Writer’s Cramp
Spend all day writing
Fingers get jerky, clumsy & tired

**Overruling reflexes**
- Antisaccade task
  - Object appears in periphery
  - Must look in opposite direction
  - Top-down processing overruling reflex
  - Improves with age unless
    - Very young; hard to look away from attention getter
- ADHD

**Age & Neurons**
- At 30, frontal cortex begins to thin
  - Much individual variation
- 60+
  - Synapses alter more slowly (learn)
  - Hippocampus gradually shrinks
  - Compensate by using more brain areas

**Choice 3: Under the Brain**
- Thalamus
- Hypothalamus
- Pituitary
- Pons
- Basal ganglia: 4 structures
- Amygdala
  - left & right
  - memory consolidation
  - strength of emotion impacts memory strength

**Optic Chiasm**
- Transfer neural info
  - left fields to right side
  - inside switches
  - cross your nose

**Path**
- eye
- optic nerve
- optic chiasm
- optic tract
- LGN
- occipital lobe
NOT IN LECTURE

Developing brain vulnerabilities
  Toxic chemicals
  Malnutrition
  Infections
  Teratogens
    Environmental factor
    Interfere with development
  Medication, drug, alcohol or substance
  Disease

Critical Periods
  Implantation = common blood supply
    whatever’s in mother’s blood crosses
    10 to 14 days after conception
    3.5 to 4.5 weeks
  Closure of the neural tube
  Central nervous system vulnerable throughout pregnancy

3 Major Substances
  Alcohol
  Phenytoin
  Chickenpox

1. Fetal alcohol syndrome
  Best known non-genetic cause of mental retardation
  (3 in 1,000)
  Infant brains are especially sensitive to alcohol
  Suppress release of glutamate
  brain’s main excitatory
  neurons receive less excitation and undergo apoptosis
  Alcohol broken down more slowly
  immature liver
  Alcohol levels remain high longer
  Worse when born to alcoholic mothers
drink more than four to five drinks/day
  No amount of alcohol is safe

2. Phenytoin (or Dilantin)
  Anti-convulsive
  used to treat epilepsy (seizure disorder)
  10% chance of birth defects
  Fetal Hydantoin Syndrome
  If taken in the first trimester

3. Varicella (chickenpox)
  Highly infectious disease
  95% of Americans have had it
  90% of pregnant women are immune
  1 out of 2,000 develop during pregnancy
A. If in pregnancy (week 1-20)
2% chance of defects
“congenital varicella syndrome“
Scars
Malformed and paralyzed limbs

B. Newborn period
5 days before to 2 after birth
About 25% newborns become infected
About 30% of infected babies will die if not treated

Parental use of:
- Cocaine or cigarettes
- ADHD
- Antidepressant drugs
- Heart problems
- Birth Defects
- 3-5% of newborns
- Leading cause of infant mortality
- Majority have no known cause

Blood-Brain Barrier

Paul Ehrlich, 1800’s
Injected blue dye into animals
All tissues turned blue EXCEPT brain and spinal cord
Keeps most chemicals out of brain

Why need BBB?
Brain has no immune system
Neurons can’t replicate-replace
No way to fix damage
Viruses that do enter kill you
Rabies
Neural disorders last whole life
Chicken pox-shingles

How it works
Keeps out harmful chemicals
Keeps out medications
Cancer med
Dopamine for Parkinson’s
Astrocytes form layer around brain blood vessels
may be responsible for transporting ions from brain to blood
Semi-permeable
Endothelial cells line capillaries
Small spaces between each
Some things can move between them
Loosely joined in body, large gaps
Tightly joined in brain, blocking most molecules
Large molecules can’t easily pass thru
Molecules with a high electrical charge are slowed down
Protects the brain

**What can cross passively**
Small uncharged molecules
- Oxygen & carbon dioxide
Molecules dissolve in fats
capillary walls are fats

**What can cross actively**
An active transport system
protein-mediated process
uses energy to pump chemicals
E.g., burn glucose for energy

**Broken by:**
Hypertension (high blood pressure)
Development (not fully formed at birth)
High concentrations of some substances
Microwaves & radiation
Inflammation
Brain injury
Infections
Alzheimer’s disease
endothelial cells shrink
makes gaps
harmful chemicals enter

**Nourishing Neurons**
Almost all need glucose
Practically only nutrient that crosses blood-brain barrier in adults
Ketones can also cross but are in short supply.
If you can’t use glucose
Korsakoff’s syndrome
thiamine (vitamin B1) deficiency
inability to use glucose
neuron death
severe memory impairment

**Head Injury**

**Open or Closed**
Open head injury (penetrating)
Object enters brain
Closed head injury (skull not broke)
Concussion
Most common traumatic injury
Brain gets rattled
Causes
- Car, train, airplane accident
- Fall
- Assault
- Sports

Symptoms
- Can show immediately or develop slowly
- Unequal pupil size
- Headaches
- Obvious
- Object sticking out of head
- Fluid draining from nose-ears
- Clear or bloody
- Coma or unconscious
- Paralysis
- Seizures

Sort Of Obvious
- Slurred speech
- Blurred vision
- Lack of coordination
- Memory loss
- Stiff neck
- Vomiting more than once; children often vomit once

Not So Obvious
- Irritability (especially children)
- Mood or personality changes
- Drowsiness
- Confusion
- Loss of hearing, vision, taste or smell
- Low breathing rate
- Memory loss
- Symptoms improve, then get worse

Get immediate help if
- Loss consciousness, even briefly
- Severe headache or stiff neck
- Vomits more than once
- Behaves abnormally
- Unusually drowsy

Do
- Call 911
- Make sure breathing
- Assume spinal cord injury
- If normal breathing but unconscious
  - Stabilize head and neck
  - Hands on both sides of head
If bleeding
   Press clean cloth on wound
   If soaks through, don’t remove it
   Put another cloth over it

DO NOT
   Don’t wash deep head wound
   Don’t move or shake
   Don’t remove helmet
   Don’t pick up child
   Don’t drink alcohol (48 hours)

If skull fracture
   Don’t apply pressure to bleeding site
   Don’t remove debris from wound
   No aspirin
   Aspirin & ibuprofen can increase risk of bleeding

If vomiting
   Roll the head, neck & body as one unit

Sleeping
   Wake every 2 to 3 hours, check alertness
   ask simple questions: “What is your name?”

Occipital Lobe

Overview

Five Steps To The Brain
   Light
   Eye
   Optic Chiasm
   LGN
   Occipital Lobe

Light

Electromagnetic energy comes straight at you

Frequency
   wave length
   peak to peak
   400-700 nm
   longer is slower
   color
   spectrum
   cosmic rays = very very very fast
   gamma rays = very very fast
   X rays = very fast
ultra violet rays = fast
visible light = medium
infrared = very very slow
Tv & radio = very very very slow
electricity = very very very very slow

Amplitude
height
intensity
brightness

Absorption
Light source (sun, moon, candle)
object
absorption
reflection (shiny, smooth)
perception (see color not absorbed)

Choice 1: Eye
Human eye

Sclera
Greek for hard
1 mm thick
Fibrous strands in parallel
like fiber strapping tape
White of the eye
Covers entire ball
Not cornea & optic nerve exit
Fibers resist internal pressure
twice the atmosphere

Muscles
Held-moved by 6 tiny muscles
Nystagmus = can’t hold eyes still
Strabismus (strabismic amblyopia)
Lazy Eye or Amblyopia
Eyes don’t point same direction
Two don’t help perceive depth
Treatment
Patch over active eye
Play action video games

Cornea
Bulges out from sclera
Smooth, neatly organized
Transparent (no blood vessels)
Very sensitive to touch (close lid)
Nourished by tears (on outside)
aqueous humor (on inside)
2/3 of focus of eye  
Dome-shaped  
Irregularity of surface  

**Astigmatism**  
Inherited  
Cornea warping  
Blurred vision for lines in one direction  
Symptoms  
- squinting & blurred vision  
- headaches, eye strain  
Treatment  
Glasses before age 3-4 years  

**Aqueous Humor**  
Spongy tissue  
Keeps eye inflated  
Removes waste  
Mostly water  
- Also an antioxidant  
- Protects from UV rays  
Provides oxygen, nourishment to cornea & lens  
Continuously refreshed  
- In from ciliary body  
- Drained into Schlemm’s canal  

**Glaucoma**  
Blockage of aqueous humor  
Damage to iris  
Blindness  

**Iris**  
2 layers  
- Outer layer of pigment  
  - Color part of eye  
  - Can be translucent (albinos)  
- Inner layer of blood vessels  

**Pupil of the Iris**  
Hole in middle of iris  
2 sets of muscles  
- circular = close pupil  
- radial = open pupil  
Varies in size (4:1 ratio)  
Allows 16: 1 ratio of light  
- actual ratio changes with age  
  - in dim light, 80 yr old has half as wide opening as 20 yr old
Advantages of small opening = depth of field

Lens
- held in place by strings (zonules)
- suspended
- crystalline (clear proteins)
- bean shaped
  - diameter & thick of large aspirin
- Has no blood vessels
- Mostly water & protein

3 parts
- elastic covering
  - changes shape of lens
  - controls flow of aqueous humor
- epithelial
  - toward edge of lens
  - synthesizes proteins
- lens
  - Can be irregularly shaped (astigmatism, but not common)

Never stops growing
- Adds fibers to edge
  - center becomes thin
  - some center fibers there at birth
- As ages
  - more dense & hard (sclerosis)
  - less transparent (cataract)

Cataracts
- Born with cloudy lens
  - If surgically repaired at 2-6 months old
    - eventually nearly normal vision
- Early cataract in left eye
  - limits visual info to right hem.
    - face recognition

Vitreous Humor
- Jelly-like, like raw egg whites
- Not continuously renewed
- Floaters
- More liquid with age
- Can become detached
  - posterior vitreous detachment or (PVD)

Retinal Circulatory System
- 1 of 2 blood supplies
- In front of the retina
leaves shadows on retina; brain ignores
Supplies nourishment to non-receptor structures (ganglion, horizontal cells, etc.)

Choice 2: Retina

etina = net

Inner limiting membrane
Separates vitreous humor & retina
Formed by astrocytes
Feet of Müller cells (glial) support cells for retina
act as light collectors
like a fiber optic plate
funnels light to rods & cones

Macula
Off to side
Optic nerve
Blood vessels

Macula Degeneration
Older adults (major cause of blindness)
Loss of vision in center
Can’t read or recognize faces
Lose most detail of images
Dry (nonexudative)
Cellular debris (drusen)
Yellow deposits
Grow between retina & choroid
Retina becomes detached
Severity depends on size and # of drusen
Wet (exudative)
Choroid blood vessels grow
Retina becomes detached
More severe
Treatments
Laser coagulation and meds

Fovea
Fovea centralis
In center of macula
Most cones are here
No S-cones

Fovea regions
Fovea = L & M cones; v. sharp
Parafovea = S & rods; sharpish
Perifovea
   Outer region, Poor acuity
   Mostly rods

Net of Layers
   Ganglion cells = to brain
   Amacrine cells = interneurons
   Bipolar cells = connect receptors to ganglions
   Horizontal cells = sharp edges (lateral inhibition)
   Rods = respond to many wavelengths, shades of gray
   Cones = respond to narrow range of wavelengths, color

Rods

Outside rods
   narrow and cylindrical in shape
   filled with rod disks
   900 free-floating lamellae
   Floating in cytoplasm
   Contain visual purple (rhodopsin)
      Like ink in laser printer
      Can’t process purple light

Inside rods:
   cell nucleus
   fiber ending in a single end-bulb (a rod spherule)

Polarization
   Normal neuron
      -70mV resting potential
      depolarises to +40mV.
   Rods resting potential is -30mV
      Hit by light
      Hyperpolarizes to -60mV

Connect to bipolar cells
   Many rods to one ganglion
   Spatial summation

Summary
   Rods are peripheral
   Night vision (10k more sensitive)
   Target detection
   Fast processing
   Low quality images
   Intensity & shades of gray
   Sensitive to lots of wavelengths

Cones

Summary
   Cones are centralized
Day vision
Target identification
Slow processing
High quality images
Color
Sensitive to specific wavelengths

**Structure**
- Shorter, broader, and more tapered than rods
- Have no visual purple
- 1 to 1
  - 1 cone to 1 bipolar cell
  - 1 bipolar to 1 ganglion cell, chain to brain
- Each cone has corresponding spot in visual cortex

**Midget Ganglion Cells**
- Small
- Each cone has one
- 1 to 1
- Each fovea cone
- Direct line to brain
- Exact location of point of light

**Wiring**
- 1st route is direct to bipolar cell
- 2nd route is to horizontal cell
- Horizontal then goes to bipolar

**Retina**
- 120 million rods (20:1)
- 6 million cones

**Lateral inhibition**
- Horizontal cells inhibit neighbor
- Inhibit bipolar cells
- Activate 1 cone, tells next to stop
- Give very sharp lines & edges

**Bipolar cells**
- Separate ones for rods & cones
- 10+ types of cone bipolar cells
- 1 type of rod bipolar cells

**Output channels**
- 3 Color receptors (plus B-W)
- 3 Channels of information
- Retina info is sorted into three “channels”

**Choice 3: Color**
- Molecules absorb light
- Even molecules come in colors
If hit by light, molecule changes

**Chromophore**
- Form of Vitamin A
- Photons changes it shape
- Causes activation of large protein called an opsin

**Opsin**
- Several types, similar process
- **Rods**
  - Thermally stable
  - Rhodopsin
- **Cones**
  - Less stable
  - Photopsins
  - Long = Red region
  - Medium = Green region
  - Short = Blue region
  - Respond to range of wavelengths
    - Not just one color
    - Varies with light intensity

**Photo Receptors**
- Different combos of 3 pigments
- Each cone detect all colors
- Level of energy need varies

**Color**
- 3 Color receptors (plus B-W)
  - Long = slow red light
  - Medium = medium green light
  - Short = fast blue light
- Rods = intensity

**Retina output**
- Spatially encodes images
- Filters & compresses data
- 100 times more receptors than ganglion cells
- Spontaneously firing base rate
  - Increase rate = excitation
  - Decrease rate = inhibition

**Theories of Color**
1. **Trichromatic**
   - Young-Helmholtz Theory
   - 3 types of cones
   - Doesn’t explain red-green color blindness
2. Opponent-Process Theory
Paired opposites:
  white-black
  red-green
  yellow-blue
Afterimages from fatiguing
Prolonged stimulation
 Doesn’t explain color constancy
3. Retinex Theory
Recognize color as light changes
Cortex compares inputs
Determines appropriate bright

NOT COVERED IN LECTURE

Types of ganglion cells

Midget
  80% of ganglion cells
  Small dendritic trees
  Small center-surround fields
  Small bodies; slow
  Mostly from midget bipolar (1:1)
  Color but weakly to contrast
  Parvocellular; P pathway
  B cells
  Synapse only to LGN

Parasol
  Respond well to low-contrast $
  Center-surround large fields
  Magnocellular
  M pathway
  A cells
  Respond best to moving stimuli
  Most synapse to LGN
  Few to other areas of thalamus

Bistratified
  Small as dust cells
  10% of ganglions
  Koniocellular
  K pathway
  Moderate # of inputs
  Moderate resolution
  Moderate contrast
  Moderate speed
  Center but no surrounds
    Always on to blue
    Always off to red and green
Misc
Photosensitive Ganglion Cells
Giant retinal ganglion cells
   Melanopsin
   Light responsive
   Circadian rhythm
Other cells too (more than you need to know now)

Ganglion cells
Retina output
   Form the optic nerve (optic tract)
   Leave eye through blind spot
Function
   abstract & enhance cone signals
   recognize diff in color
   despite variations in light level = color constancy

LGN
Lateral Geniculate Nucleus
   Part of thalamus collection
LGN input from
   Eye
      90% of fibers go to LGN
      10% go to Superior Colliculus
         controlling eye movements
   Other parts of thalamus
   Other parts of LGN
   Brain stem
   Cerebral cortex
      More input from cortex than to it
   Small signal back to cortex
      10 in from retina
      Sends 4 to cortex

Development of Visual Cortex
LGN and V1 develop early
   Needs real life to fine-tune them

Visual Paths
Dorsal Path (where)
   To parietal lobe
   3D view of the world
Damage
   Have most normal vision
      can read
      recognize faces
      describe objects in detail
Ventral Path (what)
To temporal lobe
Encyclopedia

Damage
Know what things are but not where
Can’t reach out and grab
Can see and grab
Can’t watch TV
Can’t tell what is what

Face recognition

Fusiform gyrus of inferior temporal cortex
Car model identification
Bird species
Lateral fusiform gyrus
    Left = recognizes "face-like" features in objects
    Right = determines if actual face
where temporal lobe meets occipital lobe
Vital for object & face recognition
    processing color info
    word recognition
    number recognition?
    within-category identification

Infant Vision
Infants strongly prefer: Faces
2 days old, mimic expressions
Not aware of emotional content
At 2 months: want parts in right places
Five-month old: pay same attention to happy and fearful faces
Seven-months: focus more on fearful faces

Face Recognition is a very difficult task
Lots of info to process
Gender, expression, age, pose…
Estimating age from face images is hard

Faces are so similar
Greebles
Complex 3D objects
Organized into two categories: gender & family
Expert greeble identifiers
Activity in right middle fusiform gyrus is similar to when recognizing faces
Novice greeble idenfiers
Not similar
Right hemisphere
holistic strategy

Left hemisphere
analytic strategy

Right lateral fusiform gyrus
hallucinations of faces
Charles Bonnet syndrome
Hypnagogic hallucinations
Peduncular hallucinations
drug-induced hallucinations
perception of emotions in facial $
may be related to face blindness (prosopagnosia)

Prosopagnosia = Impairment in recognizing faces
usually caused by brain injury
differ in abilities to understand face
Inability to recognize faces
No loss of vision or memory
Can identify young-old
Can indentify male-female
Not know who they are

Lateralization in face identification
Male use right hemisphere
men are right lateralized for object and facial perception
Women use left hemisphere
left lateralized for facial tasks
right or neutral for object perception

Sex differences
Men tend to recognize fewer faces of women than women do
No sex differences with male faces
Several independent sub-processes working in unison?

Best at familiar faces
People we know
People related to
People who look like us
Same ethnicity

Object Recognition
Identifying objects
Figure & background
Respond same way even if change position, size and angle
Important for shape constancy
Changes in orientation
Moderately occluded
Changes in size
Novel examples of objects
Degraded images

Retina image varies
Size of retinal image impacted by
Distance from image
Which retina part $ impacted by
Vantage point viewed
Relative loc. of object-viewer

Rotational Invariance
Different angles & vantage points
Even if never seen before
More local features

Size Invariance
Actual or apparent size variations
But not at extremes

Translational Invariance
Moved to a new position
$ different part of retina
Still recognize it
Not absolute position in environment
Not relative position to objects

Objects with missing parts
Correctly ID if have 2 or 3 parts
Missing 1 sail is easy
Not when 1 part only

Geons Theory
The major idea: visual system extracts geons (basic shapes)
cubes, spheres & wedges…
Stored in brain as structural descriptions?
Which geons
How interrelate (cube on top of triangle)
Parse object into geons
Determine interrelations
Maybe as few as 36 geons
Local features = not enough

Dual Recognition Theory
Primal recognition
fast-acting
not higher-level cognitive processes

Higher-level processing
shading, texture, or color
top-down processing of environmental cues
Use context to ID difficult ones

**Agnosia**

Lose ability to recognize
- Objects and shapes
- Faces
- Sounds
- Smells

**Visual agnosia**

Can’t recognize objects
- Lesion in
  - Left occipital lobe
  - Left temporal lobe

**Form agnosia**

Can’t perceive whole
- Only recognize parts

**Inferior Temporal Cortex**

Underside of temporal lobe
- Input from occipital lobe
- Cells respond to physical stimuli
- Cells also respond to what viewer perceives (visualizes)

**Optic nerve problems**

**Multiple Sclerosis**

One of the places it impacts
- De-myelinization
- Blurred vision, etc.

**Striate Cortex**

Development of Visual Cortex
- LGN and V1 develop early
- Needs real life to fine-tune them

**Primary projection area**

**5 major layers**

- Striped look
- V1 = 1st stage of processing
- V2 = associations (circle, angles)
- V3 = lower visual field
- V4 = color & spatial
- V5 = motion+

**Primary Visual Cortex (V1)**

- Striate cortex in occipital lobe
- 1st stage of visual processing
- Most visual input goes into V1
Geniculo-Striate Pathway

Striate Neurons (Neurons in V1)

1. Simple cells
   Only in V1
   fixed excitatory & inhibitory zones
   Most have bar-shaped or edge-shaped receptive fields

2. Complex cells
   In V1 or V2
   Orientations of light
   No fixed excitat-inhib zones
   Input from combos of simple cells

3. Hyper-Complex cells
   End-stopped
   Bar-shaped recpt. field at 1 end
   Like complex cells
   But with strong inhibitory area

Columns of Cortex

Grouped in columns
Perpendicular to the surface
Arrange by specific function
   Left eye only
   Both eyes equally
   One orientation only

Feature Detectors?
   Prolonged exposure decreases sensitivity
   Stare at waterfall illusion
   Looks like flowing upwards

Damage to V1
   No conscious vision or visual imagery, even in dreams
   Blind sight

Temporal

Overview

   Ventral = high level vis. process
   Medial = memory
   Superior = cochlea
   Posterior = audio-motor proces
   Temporal-parietal = Wernicke

Inferior Temporal Region

   Ventral stream for vision
      Occipital to temporal
      Under part of temporal lobe
Main input from
LGN
Parvocellular cells of V4
As info moves thru temporal
Processes larger receptive fields
Takes longer to process
Analyses more complex
Rep. of entire visual field
Uses cues to judge significance
  Attention
  Stimulus salience
  Working memory
High-level visual processing
Complex stimuli
Faces (fusiform gyrus)
Scenes (parahippocampal)
Surrounds hippocampus
Inferior temporal gyrus
Complex object features
  global shape
  face perception?
Medial Temporal Lobe
  Declarative memory
    Facts you know – L hemisphere
    Events you’ve experienced – R
    Interacts with frontal lobes
    Create long-term memories
    Maintain long-term memories
  Long-term memory
    Becomes independent of encoding process
Hippocampus & adjacent areas
  No simple dichotomies
    associative vs. nonassociative
    episodic vs. semantic memory
    recollection vs. familiarity
  Work together
  Transfer from STM to LTM
  Control spatial memory
Damage causes
  anterograde amnesia
  Medial Temporal Lobe
Declarative (explicit) memory
  Semantic memory
  Left hemisphere: Facts
  Right hemisphere: Episodic memory
What I did on my vacation

Choice 1: Ear

Anatomy of the Ear

1. Outer Ear = pinna
   Pinna (pinnae) - visible ear
   funnels sound to ear drum
   helps in sound localization
   Anatomy of the Ear
   Tympanic membrane
   Connects pinna to ear drum
   Vibrates to sound wave

2. Middle Ear
   Ossicular Chain
   Pre-amplifier
   amplifies vibrations 20x
   3 small bones
   Attenuation reflex
   brain senses loud sound, tenses up muscles
   To prevent damage, bones don’t move
   Greater for low frequencies (higher freq. easier to discern)

3. Inner Ear
   A fluid-filled structure
   fluid is called endolymph
   similar to intracellular fluid
   high in potassium
   low in sodium
   Composed of
   bony labyrinth
   membranous labyrinth
   suspended within bony labyrinth
   delicate continuous membrane
   Space between membranous & bony labyrinths
   filled with perilymph
   similar to cerebral spinal fluid
   2 outlets to air-filled middle ear
   Oval window
   filled by plate of stapes
   Fluid pressure
   Round window
   pressure valve

Cochlea
   Spiral-shaped tube
Has 2 connected canals
  Upper vestibular canal
  Lower tympanic canal
Separate at large end
continuous at the apex
Fluid filled (perilymph)
Has a middle canal
  Cochlear duct
  Filled with endolymph

Organ of Corti
  “spiral organ”
  hair cells for hearing (cilia)
  Basilar membrane with hair cells rest on it
  The basilar membrane separates the cochlear duct from the tympanic canal
  The tectorial membrane lies above the hair cells

Stereocilia
  Connected by extracellular links
  Graded in height
  Arranged in bundles
  Pseudo-hexagonal symmetry
  Moving fluid $ hair cells
  Signals to brain
  Perceived as sound

Hearing Loss
  Bad bone conduction
  Hearing aids
  Bad cochlea
  Implant
  Dead cilia

Most Common Causes
  Age (presbycusis)
    Gradual, steady loss
  Noise
    Motorcycles, lawn mower
    Music in headphones
    Gun shots
db
  0 barely audible
  20 leaves ruffling
  40 quiet suburbia
  60 speaking voice
  100 subway train
  140 jet taking off

Obstructions
Earwax
Objects
Chemicals
  Some antibiotics
  Arsenic, mercury, tin, lead
Head injury
  Structural damage
Infections
  Middle ear (otitis media)
  Swimmer’s ear (otitis externa
  Fluid (cold or flu)

C. Prevention
  Good genes
  Cover your ears
    Lawn movers
    Guns
  Don’t smoke
    Correlation, cause unknown
    Oxygen
    Neurotransmitters
    Developing brain
  No loud music

Choice 2: Processing Sound
  Vestibulocochlear nerve
  Cochlea but stops at
  Cochlear nuclei
  Superior olivary complex
  Vestibulocochlear nerve
  Inferior colliculus
  Thalamus (medial geniculate)
  Primary auditory cortex
  Dorsal cochlear nucleus
  Ventral cochlear nucleus
  Superior olivary complex
    In the pons
      Input: ventral cochlear nucleus
  Lateral superior olive (LSO)
    Detecting ineraural level
  Medial superior olive (MSO)
    Interaural time difference
  Inferior colliculi
    just below superior colliculi
    visual processing centers
    Integrates sound source info
  Medial geniculate nucleus
Auditory Cortex

Highly organized

3 Parts (concentrically)

Primary auditory cortex
Direct input from MGN
Tonotopically organized
Frequencies respond best to
Low at one end, high at other
Complete frequency map
Identifies loudness, pitch, rhythm

Secondary auditory cortex
Interconnected
Process patterns of
Harmony
Melody
Rhythm

Tertiary auditory cortex
integrates musical experience

What audio cortex does

Analyses
Identifying auditory objects
Identifying location of a sound
Segmenting streams

How it all works

Unclear

Input
Multiple sounds
Occur simultaneously

Task
which components go together
location of sounds
groupings based on
Harmony
Timing
Pitch

Frontal & Parietal lobes too
Why each note played by different instrument in orchestra sounds different
Same pitch

Gamma waves
Ss exposed to three or four cycles of 40 hertz click
Spike in EEG
Hallucination 12-30 Hz
Left auditory cortex of schiz.
When remember song in mind
  Don’t perceive sound
  Experience melody, rhythm & overall experience

Choice 3: Wernicke’s area
  Where temporal & parietal lobes meet
  Understanding of written words
  Understanding speech
    auditory word recognition
    mimicking words

Dominant Side
  Usually left hemisphere
  Resolve associative meanings
  Bank---------teller

Non-Dominant Side
  Usually right hemisphere
  Resolve subordinate meanings
  ambiguous word meaning
  River bank
  Money bank

Damage to this area
  Receptive aphasia
  Impairs language comprehend
  Natural-sounding rhythm
  Normal syntax
  Gibberish
  Also called
    Fluent aphasia
    Jargon aphasia
  Nonverbal sound problems
    Animal noises
    Machine sounds

NOT COVERED IN LECTURE

Parietal Lobe

Overview
  Named for overlying bone (parietal bone)
  Above occipital lobe
  Behind frontal lobe
  Integrates sensory information
  Spatial sense
  Navigation
1. Somatosensory Cortex
   - Visual
   - Auditory
   - Olfactory
   - Gustatory
   - Parietal lobes

2. Posterior Parietal Cortex
   - Also called the Somatosensory Assoc. Cortex
   - Multimedia
     - Dorsal stream of vision
       - Where stream of spatial vision
       - How stream of visual action
     - Used by oculomotor system for targeting eye movements
     - Spatial location
     - Organized in gaze-centered coordinates
       - 'remapped' when eyes move
     - Input from multiple senses
     - Encode location of a reach target
     - Manipulation of hands
     - Shape, size & orientation of objects to be grasped

Damage to right hemisphere
   - Problems with visualization
   - Imagery
   - Neglect of left-side space
   - Neglect left side of the body

Damage to left hemisphere
   - Problems in mathematics
   - Understanding symbols
   - Reading
   - Writing

Mechanical Senses
   - Vestibular sensations (inner ear)
   - Tactile Sensations
     - Itch, touch, pressure, pain

Vestibular System
   - Measures
     - Position-movement of head
     - Pressure, bending
     - Spatial orientation
     - Balance
     - Motion
       - objective = world moving
       - subjective = you’re moving
       - pseudovertigo = rotation
       - Vertigo = whirling, spinning
Common
20-30% of people
all ages (more common as age)

Causes
- cold
- head trauma
- chemicals
- motion sickness
- central nervous system (slow or no improvement)
  - spinal injury
  - Parkinson’s
  - migraine
  - MS

Projects to cerebellum, thalamus, eyes & spine

Signals come from:
- Semicircular canals
  - push-pull
  - rotational movements
    - lateral = horizontal (pirouette)
    - superior = anterior (head over heels)
    - inferior = posterior (cartwheels)
- Neck muscle "stretch" receptors
- Utricle (gravity)

Sends signals to
- control eye movement
- keep you upright

Labyrinth of inner ear
- Two major components
  - Semicircular system
    - rotational movements
  - Otoliths
    - linear acceleration
      - Utricle = eye movement
      - Saccule = posture
    - otoconial crystals
      - heavier than gel
      - displaced during linear acceleration

Choice 1: Itch
- Exists in two forms
  - tissue damage
    - release of histamine
- plants

Single spinal pathway
- slower than other tactile senses
- activates neurons in spinal cord
produce a chemical called gastin-releasing peptide

**Why Itch**

- Alert to remove irritation
- Scratch irritant off skin
- Not type of pain
  - Opiates less pain & increase itch
- Correlated
  - Vigorous scratching causes pain
  - Reduce pain, reduce itch
- Similar to pain but not
  - Both use unmyelinated neurons
  - Same nerve bundle
  - Both originate in skin
  - Two distinct systems
- Itch receptors
  - Only on top two skin layers
    - Epidermis
    - Epidermal
  - Itch on top, pain under skin?
  - No itch in muscles or joints
- Sensitivity
  - Evenly distributed across skin
  - Similar density to that of pain
- Neuropathic
  - Itch can originate at any point along afferent pathway
- Damaged nervous system
- Diseases or disorders
  - CNS or PNS
- Causes
  - Multiple sclerosis
  - Opioid use
  - Psychiatric Itch
    - Hallucinations & delusions
    - Obsessive-compulsive
    - Neurotic scratching
- Pain can reduce itch
  - Rubbing, scratching
  - Electric shock
  - Noxious heat
  - Chemicals
- More sensitive to pain
- Less sensitive to itch
- Central sensitization
  - Spinal cord input (noxious $)$
  - Allodynia = exaggerated pain
  - Hyperalgesia = extra sensitivity
Contagious Itch
  Want to scratch
  Talking about it
  See someone scratch
  Mirror neurons?
Treating Itch
  Itch-scratch-itch cycle
  Self-contagious

Choice 2: Touch
Skin Mapping
  4 findings:
    1. Sensations not continuous across skin; localized in discrete points
    2. Localization shifts over time
    3. Number of pain spots > number of pressure > number for temperature
    4. Specific sensations do not always directly correspond with the type of receptor found at that location in the skin

Somato-sensation
3 types of tactile sensations
  1. Temperature
  2. Pressure
  3. Pain

1. Temperature
  Two independent systems
  Cold
  Warm
  Not Hot
    Hot is not the extreme of warm
    Both warm and cold spots respond to “hot” stimuli
  Physiological zero
    Current skin temperature
    things you touch are compared to your current skin temperature
Structure
  Free endings of touch neurons
  Non-specialized endings
  Not so much separate neuron
  warmth receptors are slow
  Unmyelinated C-fibers
cold uses both
  C-fibers (unmyelinated)
  A delta fibers (thin myelinated)
How it works
  Warm = increase firing rate
Cooling = decrease warm rate
Cold = both
  = increase cool firing rate
  = decrease warm firing rate
Some cold receptors
  Brief pulse at high temp
  paradoxical response
Paradoxical cold
  Can’t distinguish extreme hot from extreme cold

**Temperature receptor location**

- Skin
- Bladder
- Cornea
  - Pre-optic & hypothalamic regions
- Core temp

**Up spinal cord**

- To thalamus

2. Touch

**Pressure**

- Light & Deep
- Use internal organ feedback

**Ttouch receptors**

- Meissner’s corpuscles
  - Unmyelinated nerve endings
  - Slow vibrations; texture changes
  - Lips, finger tips, palm, foreskin
  - Close to surface
  - Onset & offset
  - Touched a coin
- Merkel’s discs
  - Sustained touch and pressure
  - Close to surface
  - Fingertips
  - Slow adapting
  - Still holding coin
- Ruffini’s end organs
  - Sustained pressure
  - Slow adapting
  - Deep in skin
  - Skin stretch
  - Where coin is
- Pacinian corpuscles
  - Fast vibrations; deep pressure
  - Fast adapting (joint position)
Sudden displacements
Onset & onset
Coin leaves hand

Pressure on receptor
- opens sodium channels in axon
- action potential if enough NT
- Body to CNS
- Touch perception

Cutaneous rabbit illusion
- Tapped very rapidly 6x on wrist and then 3x near elbow
- Sensation of rabbit hopping from the wrist to elbow with extra illusory stop in between

Damage to somatosensory cortex (Alzheimer’s)
- impaired body perception
- trouble putting clothes on

Choice 3: Pain
- All tactile senses except pain adapt quickly
- Survival function of pain

Independent systems
- Sharp and dull
- Treatment for one not usually effective for the other

A. Sensing Pain
- Nociceptors
- Bipolar neurons
- Cells in dorsal root of spinal cord
- Send signals on to brain
- Signal skin damage
- Muscles, joints and organs
- Degree of pain depends on:
  - Sensitivity of receptors
  - Level of stimulation

Several types of nociceptors
1. Thermal nociceptors (extreme)
2. Mechanical nociceptors
   - Respond to intense pressure
   - Not Pacinian corpuscles (touch only)
3. Silent nociceptors
   - Respond to inflammation chemical
   - Once activated sensitive to thermal and mechanical stresses too
4. Polymodal nociceptors
   - Respond to everything
   - Thermal
   - Mechanical
Chemical stresses

Axons that carry pain info

- Vary in diameter
- Myelinated faster than unmyel.
- Thicker the faster
  - A-alpha
    - Largest
    - Insulated
    - Muscles sensations
    - Proprioception
  - A-beta
    - 2nd largest
    - Insulated
    - Touch
  - A-delta
    - Smallest of alphas; nearly as small as Cs
    - Thinly insulated
    - Pain, heat, touch
    - “Good pain”
      - Do something and it will go away
      - Put down hot frying pan
  - C fibers
    - Smallest
    - Unmyelinated
    - Slowest
    - Heat & itch
    - Diffuse, dull, chronic pain
    - “Bad pain”
      - Removing $ doesn’t remove pain
      - Signals damaged tissue

Example

- Stub (hurt) your toe
  1. moving your foot
    - A-alpha proprioceptive info
  2. sensation of hitting object
    - A-beta nerve fibers
  3. pain of tissue damage
    - A-delta and C-nerve fibers

Primary afferent axons

- Vary in diameter
  - A-alpha: largest
  - A-beta: 2nd largest
  - A-delta: 3rd largest
  - C fibers: smallest

- Vary in speed
  - A-alpha: 265 mph
Cognitive Factors influence pain

Socialization
Cultural differences
Attention (Lamaze child birth)
Pain is usually transitory
  Only lasts until $ or damage removed
Chronic Pain
  Some condition last for years
    Rheumatoid arthritis
    Peripheral neuropathy
    Cancer
Phantom Pain
  Upper limb, nearly 82%
  Lower limb, 54%
  Some have continuous pain that varies in intensity or quality
Phantom Pain Treatment
  Anesthetic injections into stump; one dose can relieve for days, weeks or permanently
  Injections of saline into soft tissue between vertebrae; pain, then relief
  Vibration or massage

Congenital Insensitivity To Pain
  Born without sense of pain
  Rare
  Continue activity after injury
  Not detect broken bones-gash
  Often get pressure sores & damaged joints

B. Relieving pain
Capsaicin
  disrupts steady $ of pain cells
Steroids
  Cortisone injections
    Relieve pain & joint inflame
    Released by adrenal gland
    Steroid hormone
    Suppresses immune system
    Which reduces inflammation
    Stops trying to heal you
Non-steroidal anti-inflamm. drugs
  3 enzymes
    Proteins
      Synthesized by 3 major enzymes
        cyclooxygenase 1 (Cox-1)
        cyclooxygenase 2 (Cox-2)
cyclooxygenase 3 (Cox-3)
All three are blocked by:
aspirin
ibuprofen (Advil, Motrin)
aproxen (Aleve)
opioids (opiates)
social pain
emotional pain

Summary
Meissner’s corpuscles
  nnmyelinated nerve endings
  slow vibrations
  fast vibrations
  texture changes
  onset-offset
Merkel’s discs
  sustained touch
Ruffini’s end organs
  sustained pressure
  slow adaptation
  fast adaptation
  skin stretch
Pacinian corpuscles
  vibrations
  deep pressure
  joint position
emotional pain
histamine
hyperalgesia = extra sensitivity

Frontal Lobe
Overview
  Lesions: wide variety symptoms
  More than any part of brain
Involved in:
  motor function
  problem solving
  spontaneity
  memory
  language
  initiation
Extremely vulnerable to injury
  Large in size
  Located up front
Most common brain injury
Mild to moderate trauma

3 Sections
1. Primary Motor Cortex
2. Pre-motor Cortex
3. Pre-frontal Cortex

Primary Motor Cortex
M1
Pre-central gyrus
Directs motor coordination
Voluntary movement

Input
Pre-Motor area
Plan and execute movements
Posterior Parietal Cortex
Visual information
Supplementary Motor Area
Planning & coordinating complex movements
(requiring two hands)
Cerebellum
Balance

Output
contains Betz cells
large neurons
long axons down spinal cord
synapse directly to motor neurons of muscles
Send info to
Cranial nerves
Lower motor neurons

Functions
Elicits movements
Not directly connected to muscles
Axons go to brainstem & spine
Central pattern generators control actual muscle move

Homunculus
Organized by body region
Top-down
Toes
Knee
Hip
Trunk
Stomach
Arm
Elbow
Wrist
Hand
Fingers
Thumb

**Important for complex actions**
Writing
Less important for coughing, sneezing, laughing, or crying

**2 major actions**
Elicit complex movement patterns
Also $ when imagine movement

**Note:**
Causes movements
Doesn’t plan them
Movement needs Muscles

**Other Areas**

**Posterior Parietal Cortex**
Planning a movement
Keeps track of body position
Intention to move

**Damage causes**
trouble converting visual perceptions into actions
trouble finding objects in space

**Supplemental motor**
Plan-organize rapid sequences
pushing, pulling, and then…

**Function**
Preparing to do movement
Watching someone else do it

**Mirror Neurons**
Type of cell or function?
Some innate
Some acquired by experience
Imitating & understanding others
Modeling

**Pre-Motor Cortex**
Preparing for movement
Somewhat active during move
接收s info about
Where target is in space
Current position of your body

**Damage**
Poorly planned movements

**Cerebellum**
Balance and coordination
Coordinates movements, not cause them
Regulator or timing mechanism
Timing of skilled movements
Posture and balance
More neurons than rest of brain combined
Anything require aim & timing
Point at moving object
Clapping hands
Writing, typing
Not good at discrete tasks
Good at continuous tasks; drawing continuous circle
Feel things with both hands
Decide if two objects same
Habit formation
Timing
Attention

Damage
Problems making rapid move
Clumsiness
Slurred speech
Inaccurate eye move

Alcohol
1st brain area impacted
Highly impacted by alcohol
Speaking rhythm (slur)
Can’t walk straight
Finger to nose test

Cellular Organization
Input from
spinal cord
sensory systems (eyes, ears…)
cerebral cortex

Neurons characteristics:
Cells highly organized
Repeating geometrical patterns
Precisely arranged
Multiple copies of same unit

Parallel fibers
Parallel to each other
Perpendicular to Purkinje cells

Purkinje cells
Flat cells in sequential planes
Inhibit cells in cerebellum nuclei
Inhibit vestibular nuclei too
Controls timing of movement
Muscles

1. Smooth muscles
   - Digestive sys. & internal organs
   - Long, thin cells

2. Striated
   - Skeletal muscles
     - Acetylcholine causes contraction
     - With no acetylcholine, relaxes

3. Cardiac muscles
   - Smooth & skeletal combo
   - Looks striated
   - Acts like smooth
   - Many individual fibers
   - Fibers fuse together at points
   - One axon may innervate more than one muscle fiber
   - Distinctive Firing Rhythm
   - They just beat

Principles

- One movement per muscle
- Contraction; relaxation
- Antagonistic muscles
- Two in opposite directions
- Flexor muscles
- Limbs flexed or raised
- Extensor muscles
- Extend or straighten limbs

Fast and Slow Muscles

- Contraction are chemical
- Affected by temperature
- Fish use more muscles in cold
- Fewer muscles in warmer water

Fish muscles:

- Red: slow move, no fatigue
- White: fast move, quick fatigue
- Pink: intermediate on both

Human Muscles

- Muscle fibers are mixed
- Fast-twitch fibers
- Fast contractions, fast fatigue
- Slow-twitch fibers
slower contraction, less fatigue

**Aerobic**: use oxygen during movement
- Walking, swimming, running

**Anaerobic**: don’t use oxygen
- Short burst
- Less than 2 min.
- Fast-twitch fibers fatigue
- Anaerobic = don’t require oxygen
- Oxygen is needed for recovery
- Produce lactate and phosphate
- Give sensation of fatigue

People vary
- Amounts of fast- and slow-twitch can increase one or the other depending on which use

**Proprioceptors**
- Receptor that detects position
- Indicates movement of body part
- Detect stretch & tension of muscle

**Stretch reflex**
- Also called myotatic reflex
- Monosynaptic reflex
- Muscle lengthens, spindle stretches
- Increases nerve activity
- Contracts muscle, resist stretch

**Muscle spindle**
- Kind of proprioceptor
- Inside muscle
- Detect changes in length
- Also activates stretch reflex
- Resist muscle stretch

**Human Muscles**
- Golgi tendon organ
- Located in tendons
- At ends of muscles
- Inhibit contraction when too intense

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**NOT COVERED IN LECTURE**

**Movements**
- Involuntary Movements
  - Consistent
  - Automatic responses
  - Not affected by reinforcements, punishments, and motivations
Pupil constricting to bright light

**Reflexes**

**Infantile Reflexes**
- Infants have more
- Fade with time
- Suppressed by maturing brain
- Sometimes reappear when brain damaged

**Grasp reflex**
- Put object in hand, grasp tightly

**Babinski reflex**
- Stroke sole of foot
- Extend big toe, fan others

**Rooting Reflex**
- Touch cheek, turn head & suck
- Not pure reflex
- Intensity increase when hungry

**Life-long Reflexes**
- Knee jerk reflex
- Lengthen muscle fibers
- Monosynaptic reflex
- Not involve brain
- Knee jerk reflex
- When muscles lengthen
- Reflexively contract
- Helps in posture
- Sneezing
- Coughing

**Most behaviors are not purely voluntary or involuntary**
- Most are sensitive to feedback
- Ballistic movements
- Once initiated, can’t be altered
- Completely ballistic are rare

**Central pattern generators**
- Generate rhythmic patterns of motor output
- Wings flapping
- Fin movements

**Motor programs**
- Fixed sequence of movements
- Can be learned or built in

**Motor coordination**
- Integration of auditory-visual info
- Skilled movements
- Dancing
Throwing

Conscious Decisions
Readiness Potential?
Motor cortex activity may proceed decision to move
Decisions to make movement unconscious?
Connects To Spinal Cord

Learning movements
Movement patterns more consistent from trial to trial
Inhibition of Movement

Antisaccade task
Task: Look in opposite direction of moving object on periphery of visual field
Very hard to do
Very strong tendency to watch moving object
Inhibition of Movement
Almost impossible for kids 5-7
Ability develops slowly
prefrontal cortex is one of slowest brain areas to reach maturity

Corticospinal tracts
Two tracts

Lateral corticospinal tract
Axons from
primary motor cortex
red nucleus of midbrain
Path downward
Get closer together as they go
Down thru white matter
Includes Betz cells
Lateral corticospinal tract
Path downward
Go to medulla oblongata
Cross contralateral
Medulla Pyramids
Down spine to control
Hands, fingers, feet & toes
Many cranial nerves
(called corticobulbar tract)
Facial muscles

Medial corticospinal tract
Also called
Anterior corticospinal
Ventral corticopinal
Direct pyramidal tract
Much smaller than lateral tract
Inversely related
Ipsalateral motor control

**Route**
- Axons from primary motor cortex
- Supplementary motor cortex
- Midbrain tectum
- Reticular formation
- Vestibular nucleus

Go to both sides of spinal cord largely responsible for:
- Neck, shoulder & trunk move
- Medial corticospinal tract
- Control posture
- Vestibulospinal: vestibular information
- Tectospinal: visual information
- Reticularspinal: controls muscles

**Cerebral Palsy**
- Clumsiness is from competition of contralateral and ipsilateral paths

**Parkinson’s Disease**

**Symptoms**
- First symptom is loss of smell
- Slow movements
- Resting tremor
- Rigidity
- Difficulty initiating movement
- Cognitive deficits
- Depression (no outbursts)
- But can follow visual cues
- Follow parade
- Climb stairs
- “Step on the cracks” (sidewalk)

**Incidence**
- 1–2% of those over 65
- 50% more men than women
- Parkinson’s Disease
- Progression
- Gradual progressive death of neurons
- Especially in substantia nigra

**Substantia nigra**
- Less $ of motor cortex
- Slower onset of movements
- When over 45
- Neuron loss of 1% per year
- Most have enough to spare
- When reach 20-30% of normal
Parkinsonian symptoms begin
Early onset
Probably genetic
Late onset
More common
Not genetic
Chances decrease if
Drink coffee
Smoke
Decaffeinated coffee and nicotine free cigarettes work just as well
Reduce damage to mitochondria
Progressive death of neurons
Gradual
Decrease in dopamine
Decreased neural activity
Atrophy
Cell death

L-Dopa Treatment
Precursor to dopamine
Crosses BBB
Hope it converts to dopamine
Not prevent continued loss
may contribute to neuron death
Harmful side effects
Effective in early stages?
Could do harm?
Doesn’t stop the disease

Other Therapies
Antioxidant drugs
Dopamine agonists
Glutamate antagonists

Dorsolateral
Overview
Frontal lobes
3. Prefrontal cortex
Most anterior
Not short term storage
But if damaged, poor executive processes
10+ microscopically different cells
working memory for objects
working memory for spatial locations
Executive Functions
- Working memory
- Cognitive flexibility
- Planning
- Inhibition
- Abstract reasoning

Highest cortical area involved in motor planning, organization and regulation
not exclusively responsible for executive functions
requires additional cortical and subcortical circuits

Spatial selective neurons
- integrated response
- sensory input
- STM retention
- motor signaling
- Spatial selective neurons
- connected to:
  - superior temporal lobe
  - posterior parietal lobe
  - cingulate premotor cortex

Dorsolateral
- Damage causes problems with
- Social judgment
- Executive memory
- Abstract thinking
- Intentionality
- Tumors produce symptoms similar to schizophrenia
- Sleep deprivation inhibits activity here

Truth Telling
- Involved in lying?
- Inhibit of normal process
- People usually tell the truth
- Need it for stability & function
- Lucid dream states?
- Hallucinations?

Schizophrenia
- Psychotic disorders
- “The Schizophrenias”
- 1% incidence
- More likely in US & Europe
- 10 to 100 times
- Chronic patients
  - under-active in dorsolateral
  - lack of dopamine neurotransmitter
  - abnormal activation during working memory
Demographic Data
- Slightly more common in men
- Earlier onset
- More severe

Originally: dementia praecox
- Eugen Bleuler called it schiz
- 1911

Starts as teens or early adult
- Typical onset 16 to 30
- Uncommon onset over 45

Symptoms vary
- Seem OK until share thoughts
- Sit without moving...for hours

Episodes
- Typical: not more than 6 weeks
- Symptoms come & go
- Lasts a few days
- Feel agitated

Hallucinations
- Lasts a few months

Delusions
- Last months or years

Range of severity
- Hospitalized
- Meaningful lives in communities

3-Factor Model
- Disorganized thinking
- Distorted thinking
- Delusions & hallucinations

Disconnected mind-motor
- Spontaneous movement
- Fluid speech
- Self control

Positive symptoms
- Happy symptoms?
- Unique to schizophrenia
- Not schiz without them

Delusions
- Unusual false beliefs
- Martians are controlling me
- Reading my mind
- Thought insertion
- “I killed someone”
- Behavior controlled by
- People on TV or movies
− Special messages
− Behavior controlled by
− Magnetic waves
− Aliens
− Believe you are someone else
− Often historical person
Hallucinations
− False sensory experiences
− Hear voices not there
− See things not there
− Voices are most common
− Hear voices
− Talk to invisible person
− Voices talk to each other
− See invisible objects or people
− Feel invisible fingers touching
− Smells
Thought disorders
1. Disorganized thinking
− organizing thoughts
− connecting thoughts
− garbled talk
2. Thought blocking
− Stop in middle of thought
− Feel thought taken out head
3. Nonsense words
− Neologisms = new words
− Disorganized speech

− Rambling sentences
− Incoherent patterns
Movement disorders
− Agitated movements
− Repeat motions over and over
− Catatonic = immobility
− Rare—treated with drugs

Negative symptoms
− Occur in other disorders
− Flat affect
− face immobile
− monotonous voice
Similar to brain damage
− poor control of eye movements
− unusual facial expressions
Negative = lack of
− Lack of pleasure
- Lack of persistence
- Social withdrawal
- Poverty of speech
- Lacks fluidity of speech; words don’t flow
- Don’t talk much, even when forced

Cognitive symptoms
- Difficult to notice
- Executive functioning
- Trouble switching tasks
- Trouble paying attention
- Trouble with working memory

Disturbed emotions
- Hyperemotional
- Depressed
- Flat affect (no emotion)

Abnormalities of perception
- Schizophrenic Art
- No difference in foreground-background
- Obsessed with objects (skulls)
- Emotionally distant
- Dark silhouettes
- Watchful eyes
- Fragments

Types
1. Disorganized
   - “Hebephrenic schizophrenia”
   - Inappropriate thoughts & behav.
   - Don’t make sense
   - Severe
   - Can’t do routine daily activities
   - bathing & meal prep
   - Hard to understand what say
   - Frustration, agitation, anger
2. Catatonic
   - Coma-like daze or Talk in bizarre-hyperactive
   - May last month+
   - Easily treated with drugs
   - Can be caused by non-schiz
3. Paranoid
   - Delusions
   - Someone trying to harm you
   - Hear voices
   - Not as many memory problems
   - Okay concentration
   - Handle daily life okay
   - Suicide risk
4. Undifferentiated
   - Not meet all criteria
   - Miscellaneous
   - Junk term

**Causes**

**Genetics**
- Runs in families
- Environmental trigger?

Old egg-sperm theory
- Older parents more schiz children

Children of schiz patients
- Less than ½ become schiz
- Inherit susceptibility to environmental factors?

**Why likely genetic component**
- Men & women about equal
- Men slightly more
- Men have earlier onset
- Men have more severity
- About 1% worldwide

**Runs in families**
- 1% in general population
- 10% when parent or sibling
- 15% in fraternal twin
- 50% when identical twin

Pure genetic effect = 100%
- greatest environmental similarity
- monozygote

**Adopted Children**
- One study
- 12.5% siblings in same environ.
- None adopted had schiz

**Correlated factors**
- Women with schizophrenia
- drink & smoke during preg?

Not one single gene
- 10+ genes are more common in schizophrenics
- DISC1 gene (disrupted in schizophrenia 1)
  - Controls production of dendritic spines
  - Controls generation of new neurons in hippocampus

**Other genes linked to**
- brain development
- glutamate synapses
- hippocampus & prefrontal cortex connections

**Combo of Genetics & Environment**

**Dopamine hypothesis**
- Over-activity of DA synapses
- In mesolimbic pathway?
DA agonists-antagonist effects
- All treatment drugs block DA receptors
- Chlorpromazine
- Originally used to prevent surgical shock
- Dramatically effective
- Reduces symptoms of schizophrenia
- DA agonists cause schiz sympts
- Cocaine
- Amphetamine
- L-DOPA
DA agonists cause schiz sympts
- Elation, euphoria
- Similar to start schiz. episode
Paranoid delusions
- Maybe caused by increased DA input to amygdala
- in emotional responses for aversive events
DA neurons release more DA?
- Clozapine
- atypical antipsychotic drug
- blocks D4 receptors
- in nucleus accumbens
- Part of the reward circuit
Caused by excess activity at some dopamine synapses
- Evidenced by
- Drugs that help
- Drugs that aggravate
Aggravaters
- Cocaine
- Amphetamine
- LSD
Dopamine not cleaned up?
- Schiz have twice as many D2 receptors occupied by dopamine as normal
Dopamine not sole cause
- Drugs that block dopamine receptors
- do so immediately
- but effects on behavior build up
- gradually over 2 or 3 weeks
Glutamate Hypothesis
Caused by poor glutamate functioning
- dopamine inhibits glutamate
- Mixed evidence
Schiz
- release less glutamate
- in prefrontal cortex & hippocampus
- release less glutamate
- have fewer glutamate receptors
  Phencyclidine (PCP)
- blocks NMDA glutamate receptors
- produces symptoms similar to schiz
- induces both negative and positive symptoms
- Doesn’t produce psychosis in preadolescents
- produces more severe symptoms than schiz
Risk to increase glutamate
- Too widely used
Don’t stimulate directly
Working on glycine (amnio acid)
- enhances NMDA effects
- not effective antipsychotic
- increases antipsychotics effects

**Brain abnormalities**

**MRI & CT studies**
- Found loss of brain tissue in patients with schizophrenia
Ventricles
- Relative size of lateral ventricles
- 2+ size of control subjects

**Mild Brain Abnormalities**
- Less than average gray matter
- Larger than average ventricles
- Smaller thalamus
- left hemisphere slightly larger
Worst in
- left temporal lobe
- frontal lobe
Immature or poorly developed
- dorsolateral prefrontal cortex
- deficits in memory & attention
Smaller cell bodies
- in frontal cortex & hippocampus

**Environmental Causes**

**Famine during pregnancy**
- (especially thiamine deficiency)

**Predictors**
- More likely if mother underweight
- More likely if low birth-weight
- More likely if Rh incompatible

**Neurodevelopmental hypothesis**
- Schiz caused by abnormalities to nervous system during prenatal or neonatal periods

**Prenatal and Neonatal**
- Mother’s nutrition
- Premature birth
- Low birth weight
- Complications during delivery

**Rh-negative & baby Rh-positive**
- may trigger immunological rejection by mother
- hearing deficits
- mental retardation
- twice usual probability of schiz
- 2%

**Season-of-birth effect**
- Winter, slightly greater
- Nutrition
- viral infections
- fever and influenza

**Flu (or other viral illness)**
- More likely if born during late winter and early spring
- More likely in cities than countryside
- More likely far from equator
- Decreased winter temp?

**Infections**
**Childhood infections**
- Such as toxoplasma gondii
- memory disorders, hallucinations, and delusions
- bacteria only reproduces in cats
- more likely to have a pet cat

**Diagnosis**
**Confused with drug abuse**
Can’t show abuse causes schiz
- Self medication
- Makes treatment less effective

**Prodromal** = pre-symptoms
- Self-isolation
- Increased unusual thoughts
- Increased suspicions
- Family history of schiz

**Self-diagnosis as bipolar**
- Or something “less sever”

**Drugs can help-hurt**
Some drugs make it worse
- Marijuana
- Amphetamines
- Cocaine

**Smoking**
- 3x likely addicted to nicotine
- 90% in schiz
- Schiz worse during withdrawal
  Chlorpromazine (Thorazine)
- 1st drug successful
Antipsychotic drugs
- Primarily work by blocking dopamine receptors
Phenothiazines
- class of neuroleptic drugs
- includes chlorpromazine
Try several medications
- Not all work the same for all
- Best combination, right dose
Relapse
- Stop taking meds
- Feel better, think don’t need
- Interact with other drugs
- Interact with alcohol
Antipsychotic medications
- available since mid-1950's
- Chlorpromazine (Thorazine)
- Haloperidol (Haldol)
- available since mid-1950's
- Perphenazine (Etrafon)
- Fluphenazine (Prolixin)
- "atypical" antipsychotics
- Clozapine (Clozaril)
- psychotic symptoms
- Hallucinations
- breaks with reality
- Clozapine (Clozaril)
- Side effect for clozapine
- Agranulocytosis = loss of white blood cells
- Risperidone (Risperdal)
- Olanzapine (Zyprexa)
- Quetiapine (Seroquel)
- Ziprasidone (Geodon)
- Aripiprazole (Abilify)
- Paliperidone (Invega)
- Old & new ones about equally effective

**Side effects**
- Worse when start
- Last few days for most
- Dizzy when changing positions
- Blurred vision
- Drowsiness
- Rapid heartbeat
- Sensitivity to the sun
- Skin rashes
- Major weight gain
- Rigidity of joints
- Muscle spasms
- Restlessness
- Tremors

Tardive dyskinesia
- Caused by long term use
- Can’t control mouth muscles
- Tremors & involuntary move
- Caused by prolonged blocking
- Of dopamine receptors in basal ganglia
- Usually in pill or liquid form
- Some are shots given monthly

New Drugs
- Mesolimbocortical system
- Where antipsychotics impact?
- Set of neurons
- Project from midbrain tegmentum to limbic system

New drugs (atypicals)
- Don’t cause movement problems
- Less intense effects on dopamine type D2 receptors
- Stronger effects at D4 and serotonin 5-HT2 receptors

Atypical antipsychotics
- More effective?
- Better with positive symptom
- Not so much with negative
- Don’t improve overall quality of life any better

Long-term drug treatment
- Antipsychotic drugs not cure
- Don’t fully treat condition
- Don’t work for 1/3 of patients
- Serious side effects
- Similar symptoms to Parkinson’s disease
- Slow movement, lack of facial expression, general weakness

NOT IN LECTURE

Phineas Gage
First indication can survive major brain trauma
- Lost 1+ frontal lobe
Working on a railroad
- Gage (then 25)
- Foreman on work gang
- Blasted a path through rock
− R&B Railroad, Vermont
  Process
  − Bore a hole, add blasting powder
  − Put in a fuse, add sand
  − Pack it in with tamping iron
  − 3’ 7” long and 1¼ inch diam.
  − 13 pounds
  − Tapered
  September 13, 1848   4:30pm
  − No sand added
  − Rod entered on left side of face
  − Tapered part first
  − Passed thru back of left eye
  − Out the top of his head
  − Landed 80 feet away
  Don’t know much about his life
  − Before or after accident
  − Can’t gauge the Gage
  Retained
  − Normal memory
  − Speech & motor skills
  Changed?
  − Mood, irritability, impatient
  − Personality
  − Exaggerated after his death
  − No longer Gage

“American Crowbar Case”
− Localization of functions
− Both sides
Damage to frontal lobe
− Describe best course of action
− But seek immediate gratification

**Orbitofrontal**

**Overview**
− Basic functions

**Dorsolateral**
− Last to myelinate
  Sleep deprivation
  Executive Functions
  Working memory
  Cognitive flexibility
Planning

Orbitofrontal
named by location
above eye orbits
least explored
least understood
sometimes considered part of limbic system
anatomically the same as ventromedial
Vary by person
Considerable individuality

Research Difficulties
OFC is close to sinuses
air filled
Hard to image (MRI, etc)

Function
Cognitive processing
Decision making
Sensory integration
Affective value of reinforcers

Controls
social adjustment
responsibility
mood
drive
Expectation of rewards-punish
Compare expected with actual
Intuitive judgments

Extensive connections
Reciprocal connections
Ventral & dorsal visual streams
Auditory-spatial processing
Phonetic processing
All sense modalities

Damage
Lesions
feel no regret
Damage causes problems with
decision-making
emotion regulation
reward expectation

ADHD
dysfunction of reward circuitry
controlling motivation
reward
impulsivity

**Obsessive-Compulsive**
- Executive functioning
- Impulse control

**Addictions**
- Dopaminergic activation of reward circuits
- Compulsive behavior
- Increased motivation take drug
- Drug addiction
- Decision making
- Reward system
- Compare expected reward/punishment with actual reward/punishment activated during intuitive coherence judgements

**Auditory Processing**
- Distinct pathways
- phonetic processing
- rostral stream
- auditory-spatial processing
- caudal stream
- extensive overlap

**Visual Processing**
- Both ventral & dorsal streams
- integration of spatial and object processing
- Medial portion of orbitofrontal
- connect with hippocampus
- cingulate
- thalamus
- Lateral portion of orbitofrontal
- connections with amygdala
- association cortex

**Lateral OFC**
- stimulus-outcome associations
- evaluation of behavior
- encode new expectations about punishment and social reprisal
- conflict resolution
- suppressing negative emotions
- approach-avoidance situations
- game of chicken

**Damage**
- inappropriate displays of anger
- inappropriate responses to anger
- left lateral
defensive
- present self in “angelic light”
- Low volume
experiencing “fear of God”
higher volume in left lateral
score higher on Machiavellian personality traits
greater thickness
outgoing and uninhibited
Greater thickness in ventromedial cortex: shy and inhibited

Visual discrimination test
Extinction
DON’T PRESS BUTTON
OFC damage: gotta press!
Disinhibited behavior
Excessive swearing
Hypersexuality
Poor social interaction
Drug, alcohol & tobacco use
Little empathy
Compulsive gambling

Visual discrimination test
reversal learning
presented pictures A and B
Learn rewarded for picking A
When rule set, switch
Damage to OFC, stay with A

Extinction
Punished for either A or B
rules don’t reversing

Iowa Gambling Task
Simulation
decision making
Bechara & Damásio
4 virtual decks of cards
choose card, win money
choose card, lose money
Goal of game
As much money as possible
Reward
Penalty
Choose cards by gut reaction
Start with $2000 (monopoly $)
Don’t know how many cards in deck (it’s virtual)
Original study had 100
Deck A and B
$100 reward
Deck C and D
$50
Deck A and B
$100 reward            Large penalty
Deck C and D
$50                  Small penalty
Same loss
Differ in distribution
number of trials
Bad deck = lose faster
over enough time will make a net loss
Good deck = lose slower
Good deck = win some
other enough time will make a net gain

After 10 cards
healthy show "stress" reaction
GSR if hover over bad deck
Damage to amygdala
never develop GSR
After 40-50 cards
Healthy
stick to the good decks
OFC damage
stick with bad deck
know losing money

Criticism
design

SGT (another task)
100 trials of uncertainty
Healthy
focus on immediate gain-loss
unable to hunch long-term outcome
Probabilistic Learning
must pass up potential large immediate rewards for small longer-term rewards
Warning cues feel like excitement & pleasure?
Healthy
Sample cards from each deck
40-50 cards, stick with good deck
OFC damage
Stick with bad deck
Even if know it's a bad deck
Schiz & OCD
perseverate (persevere)

Faux pas Test
Series of vignettes
Social occasion
Said but should not have said
Awkward occurrence
Faux P as awkward
Identify what awkward
Identify why awkward
Identify how would have felt
Identify factual control fact
OFC dysfunction
Understand the story
Can’t judge social awkward vignettes
said something should not have said
awkward occurrence
1st used with autism

Acquired brain injury
disinhibited behavior
poor social interaction
excessive swearing
hypersexuality
compulsive gambling
drug, alcohol & tobacco use
low empathy

Alzheimer's disease
neurofibrer tangles in this area
Endoplasmic reticulum
Transport system
Axon support
Collapse
Neuro-tangles
Brain proteins fold abnormally

Tau protein
Tangles in cell bodies
Clump together
Interfere with neuronal activity

Amyloid protein
Cause plaque between neurons
Apolipoprotein E
Prevents plague removal
Causes cell loss

Progressive disease
Symptoms get worse with time

Symptoms
Inappropriate emotional R
Decline in intellect
Confused thinking
Memory loss
Repeated questioning
inappropriate emotional R
Violence

Memory
Better procedural vs declarative
Better implicit vs explicit
Acquire new skills but not remember learning them

Age related
Likelihood increases with age
 Strikes 50% of those over 85

Genetic components
Person with Down’s syndrome
(3 copies of chromosome 21)
Always acquire Alzheimer’s in middle age
Early onset
chromosome 1 & 14
Late onset
chromosome 10 & 19

Environmental component
50% no relatives with disease
Yoruba people of Nigeria
high-risk genes
low incidence
Maybe due to diet?
low-calorie, low fat, low salt diet
Treatment to improve memory
Increase glucose & insulin
Acetylcholine activator drugs
Diet rich in antioxidants?
Block Aβ42 production, inoculate with small amounts of Aβ42
Inter-neuron plaque
Addiction
OFC, nucleus accumbens & amygdala
striato-thalamo-orbitofrontal circuit

Addiction
involved in development of addictive behavior
dopaminergic activation
reward circuits
Addicts show deficits in orbitofrontal, striatal, and thalamic regions
Cocaine withdrawal
Increased OFC activity
proportional to drug craving
during protracted withdrawal
(3–4 months)
reduced activity
decreased activity
detoxified alcoholics
significantly less benzodiazepine receptors
OFC to thalamus to accumbens
Bidirectional
Orbitofrontal Cortex
mediodorsal nucleus of thalamus (involved in memory)
nucleus accumbens
Reinforce
Drug administration
Drug effects
Addiction associated with
compulsive behavior
repetitive behavior
drive
anticipated conditioned response
impulsivity
loss of control
craving

Ventromedial

Review

1. Dorsolateral
   Last to myelinate
   Sleep deprivation
   Working memory
   Planning

2. Orbitofrontal
   Gambling strategies
   Alzheimer’s tangles
   Drug addiction

Comparison
   Pepsi    blind test
   Coke     sighted test

Dorsolateral
   Higher order rules
   Brand recognition

Ventromedial
   Strong emotional reactions
   Sugar, salt & carbonation!

Overview
   vmPFC
Anatomically
No difference between orbitofrontal and ventromedial
Only differ in connections

Functions
processes risk & fear
Decision making
Inhibition of emotional responses
rapidly developing during adolescence and young adulthood
connects with amygdala
bilateral lesions severely impair personal and social decision making but retain intelligence
less associated with social functions
more with emotion regulation
bilateral lesions
Difficulty choosing between options with uncertain outcomes
risk
ambiguity
Impairs learning from mistakes
make same decisions again & again
even if have negative consequences
Choose immediate rewards
blind to future consequences

Right hemisphere vmPFC
Detecting irony, sarcasm, and deception
If damaged:
Easily influenced by misleading advertising
"false tagging mechanism"
provides doubt and skepticism
regulates interaction of cognition and affect
empathic responses

Emotion regulation
social emotions
compassion, shame & guilt
moral values
anger & frustration tolerance

Obitofrontal
pleasure responses
ventromedial prefrontal cortex
preference judgement
PTSD

ventromedial prefrontal cortex
reactivating past emotional associations and events
Left vs Right
Right
intellectualization, emotional isolation
Left
projection, splitting, verbal denial, and fantasy

Gender Social Cues
gender stereotypes
categorize gender-specific names, attributes, and attitudes
Damage to vmPFC
consciously make moral judgments without error
in hypothetical situations
not in real life
make decisions inconsistent with professed moral values

Ventromedial
Includes anterior cingulate cortex
Wraps around corpus callosum

Two Computers
Left and Right Hemispheres
Each controls contralateral side
Except taste & smell
Uncrossed; own side of tongue
Work together
Control trunk & facial muscles
Staying Connected

Corpus Callosum
Set of axons interconnect hemisphere
Exchange information
Neural fibers
Wide, flat bundle
Connects L & R hemispheres
Under cortex
Largest white matter structure
200–250 million axons
Fast transmission (myelinated)

Genu = anterior (knee)
Thin axons
Connect prefrontal cortexes
Larger in musicians

Truncus = middle (body)
Thick axons
Connect motor cortexes
M1, premotor & supp. motor
Splenium = posterior portion
  Soatosensory info
  Parietal lobes
  Visual cortices

Sexual Dimorphism
  Different size in men & women?
  No

  R. B. Bean, 1906
  Larger is intelligence
  Men
  Race
  Ultimately refuted

Larger in left-handed?
  11%
  Dyslexic children
  have smaller CC

Childhood
  Gradually thickens as grow
  Slow growth til about age 10
  Eventually develop adult patterns
  Young children behavior similar to split-brain people

Fabric identification task
  Five-year olds
  Equally well w/ one or two hand
  Three-year olds
  90% more errors w/ two hands
  Lateralization of Function

Epilepsy
  Seizures = excessively synched neural activity
  Most treated with drugs (90%)
  More severe, tissue ablation
  Lateralization of Function
  Neural activity rebounds between
  prolongs seizures
  Extreme cases, severe CC
  Called split-brain people

Split-Brain People
  Present input of object to L field
  Info goes to R hem (noses cross)
  Independence
  Draw circles
  One with each hand
One hand going faster
Present input of object to L field
Info goes to R hem (noses cross)
L hand controlled by R hem
Can point to it with L hand
Can’t do it with right hand
Present object input to R field
Info to L hem (noses cross)
Can name or describe what see
Language in L hem (95%; 80%)

Each hem. can process info
Multitask

For a few weeks
Feels like two people in one body
Competition vs Cooperation
Take item off grocery shelf with L
Return them with R
Normal
Cooperation
Flash different word to each visual field at same time
Report combined concept
Flash toad to left & stool to right
Get
Eventually lessens some
Brain uses smaller connection routes to avoid conflicts
CC not the only path
Just the biggest
Other epilepsy surgeries

HM
Henry Molaison (1926-2008)
1 generalized seizure a week
began bilaterally
medial aspects of both temporal lobes
Removed both of H.M.’s medial temporal lobes (in 1953)
included most of
hippocampus
amygdala
adjacent temporal cortex
Post-surgery symptoms
Major seizures almost completely eliminated
Minor seizures down to 1-2 day
IQ increased (104 to 118)
Normal short-term memory
Moderate retrograde amnesia
loss for events shortly before

**Post-surgery symptoms**
- Severe anterograde amnesia
- Memory loss for events after
- Can’t transfer anything to LTM
- Everything is forgotten when attention shifts
- Impaired ability to form LTM
- Newer words (for him)
- Jacuzzi
- Granola
- Regarded as nonsense
- When distracted
- Underestimate his own age by 10+ years
- Can’t form episodic memories
- Memories of a single event
- Could describe previously learned facts
- Not recount personal events
- Retained ability to
- Weakly retain semantic (factual) memories
- Difficult to describe the future

**HM’s Implicit Memory**
- Mirror Drawing
- First to show improvement in HM
- Spatial –motor learning
- Implicit learning
- Rotating Disc
- Keep pen on target
  (rotating disk)
- Improved over 7-day period
- Each time saw task, claimed he had never seen it before

**Hippocampus**
- Temporal lobe & Dorsalmedial
- Semi-circle
- If damaged, amnesia
- Not accident or around it
- Remember before & after accident
- If small damage
- Retrograde amnesia
- Can’t remember past
- Just before accident
- If bilateral damage
- Anterograde amnesia
- Can’t form new memories
- Consolidation memory
- Move from short to long term
- Not necessary to retrieve info
must work to put into long term
Reproduces patterns during sleep
Encodes patterns
Sparse representations (non-overlapping)
Sparse encoding allows quick learning
Componential encoding
9x9 pixel bit map
81 pixels
Componential encoding like cortex
efficient; good for generalization
Sparse encoding
uses 13 lines
Trains cortex
repeats pattern over time
Repeats pattern over time
Find L in field of Ts
Patterns repeated
Ss unaware of pattern
Without damage
No “thinking” required
Improved over time
Priming
Damage
No improvement

Amnesia
Types
retrograde amnesia = before
anterograde amnesia = after
Progression
Normal cognition
Retrograde amnesia
Coma
Confusion
Anterograde amnesia
Normal cognitive function

TERMS
1% incidence
120 million rods (20:1)
140 jet taking off
16:1 ratio of light
18 days after conception
2 days old, mimic expressions
2 fists, crossed arms
2 identical founder cells
2/3 of focus of eye
20 leaves ruffling
3 channels of information
3 color receptors (plus B-W)
3 concentric parts
3 enzymes for pain
3 small bones
3 types of cones
3 types of tactile sensations
3D view of the world
3-factor model of schizophrenia
4 lobes
40 quiet suburbia
400-700 nm
60 speaking voice
-70mV resting potential
9x9 pixel bit map (81 pixels)
A-alpha fibers (largest, myelinated)
A-beta nerve fibers (large, myelinated)
abnormal activation
abnormalities of perception
absorption
abstract reasoning
acetylcholine
acquired brain injury
active transport system
addiction
A-delta fibers (thin, myelinated)
ADHD
adopted children
aerobic: use oxygen during movement
afterimages
agitated movements
agnosia
agranulocytosis = loss of white blood cells
alcohol
allodynia = exaggerated pain
Alzheimer’s disease
Alzheimer’s tangles
amacrine cells = interneurons
American Crowbar Case
amnesia
amphetamines
amplitude
amygdala
amyloid protein
anaerobic = don’t require oxygen during movement
anesthetic injections into stump
antagonistic muscles
anterior cingulate cortex
anterior corticospinal
anterograde amnesia = after
anti-convulsive drugs
antidepressant drugs
antioxidant drugs
antipsychotic drugs
antisaccade task
aphasia
Apolipoprotein E
apoptosis = cell death
aqueous humor
aripiprazole (Abilify)
aspirin
astigmatism
astrocytes
asymmetrical division
atrophy
atypical antipsychotics
auditory cortex
auditory-spatial processing
Babinski reflex
bacteria
bad bone conduction
bad cochlea
bad deck = lose faster
bad pain
ballistic movements
Bank--------teller
basal ganglia
BDNF
Betz cells
bilateral lesions
bipolar cells = connect receptors to ganglions
birth defects
bizarre-hyperactive talk
Bleuler, Eugen = coined term schizophrenia
blind spot
blind to future consequences
Blood-Brain Barrier
brand recognition
brightness
C fibers (small, unmyelinated)
capsaicin
cardiac muscles
cataract
catatonic = immobility
central pattern generators
cerebellum
cerebral cortex
cerebral palsy
chemical gradients
chemical guidance
chlorpromazine (Thorazine)
chronic pain
ciliary body
circadian rhythm
circular muscles = close pupil
cloudy lens
clozapine (Clozaril)
cocaine
cochlea
cold
color
coma
coma-like daze
complex cells
compulsive behavior
compulsive gambling
concussion
cones
conflict resolution
congenital varicella syndrome
connecting thoughts
conscious decisions
contagious itch
core temp
cornea
corpus collosum
corticospinal tracts
cortisone injections
cosmic rays = very very very fast
craving
critical periods
cross contralateral
crystalline (clear proteins)
cutaneous rabbit illusion
cyclooxygenase 1 (Cox-1)
cyclooxygenase 2 (Cox-2)
cyclooxygenase 3 (Cox-3)
D2 receptors
D4 receptors
DA agonists
DA receptors
dB
decaffeinated coffee
decision-making
declarative (explicit) memory
deep pressure
delusions
de-myelination
depth of field
differentiation
DISC1 gene (disrupted in schizophrenia 1)
disinhibited behavior
disorganized speech
disorganized thinking
disturbed emotions
dopamine
dopamine hypothesis
dorsal stream of vision (where)
dorsolateral prefrontal cortex
Down’s syndrome
drug addiction
drug administration
drusen
dry macular degeneration
drug administration
drusen
dry macular degeneration
drug administration
drusen
dry macular degeneration
early onset
Ehrlich, Paul
emotional pain
empathic responses
endolymph
epidermal
epidermis
epilepsy
episodic = symptoms come & go
epithelial
extensor muscles
face recognition
false sensory experiences
false tagging mechanism
farsighted
fast adaptation
fast-twitch fibers
Faux pas Test
feet of Müller cells (glial)
fetal alcohol syndrome
Fetal Hydantoin Syndrome
fin movements
firing rate
first in spinal cord
flat affect
flexor muscles
floaters
flu (or other viral illness)
fluent aphasia
fluid speech
fluphenazine (Prolixin)
forebrain
form agnosia
founder cells
fovea
fovea (fovea centralis)
frequency
frequency map
frontal lobe
fusiform gyrus
Gage, Phineas
gambling
gamma rays = very very fast
ganglion cell
garbled talk
gender stereotypes
Geniculo-Striate Pathway
genu = anterior (knee)
Geons Theory
glaucoma
glutamate
glutamate antagonists
glutamate hypothesis
Golgi tendon organ
good deck = lose slower
good pain
grasp reflex
gray matter
Greebles
GSR
hallucinations
hallucinations (auditory)
haloperidol (Haldol)
harmony
head trauma
hear voices
hearing loss
Hebephrenic schizophrenia
hindbrain
hippocampal gyrus
hippocampus
histamine
HM (Henry Molaison, 1926-2008)
homunculus
horizontal cells = sharp edges (lateral inhibition)
hyperalgesia = extra sensitivity
hyper-complex cells
hyperemotional
hyperpolarization
hypersexuality
hypertension (high blood pressure)
hypnagagic hallucinations
imagery
imagine movement
immediate gratification
implantation = common blood supply
implicit memory
impulsivity
inappropriate displays of anger
inappropriate emotional responses
inappropriate thoughts & behavior
incidence
incus
infantile reflexes
inferior = posterior (cartwheels)
inferior collicui = hearing
inferior temporal gyrus
inhibition
initiation
inner ear
intensity
intentionality
interaural time difference
inter-neuron plaque
intuitive judgments
involuntary movements
Iowa Gambling Task
ipsalateral motor control
iris
itch
itch receptors
jargon aphasia
knee jerk reflex
konio cellular
Korsakoff’s syndrome
l cones (long)
lack of dopamine neurotransmitter
lack of fluidity of speech
lack of persistence
lack of pleasure
late onset
lateral = horizontal (pirouette)
lateral corticospinal tract
lateral fusiform gyrus
lateral geniculate nucleus (LGN)
lateral inhibition
lateral superior olive (LSO)
lazy eye (amblyopia)
L-DOPA
L-Dopa treatment
left fusiform: recognizes "face-like" features in objects
left hemisphere: elements, stories & semantic memory
left lateralized for facial tasks
LGN
light
light & deep touch
linear acceleration
lingual gyrus
low birth weight
LSD
M1
macula
macular degeneration
magnocellular
mechanical nociceptors
mechanical senses
medial corticospinal tract
medial geniculate nuclei (MGN)
medial superior olive (MSO)
medial temporal lobe
medulla oblongata
medulla pyramids
Meissner’s corpuscles
melody
memory consolidation
Merkel’s discs
mesolimbic pathway
mesolimbocortical system
MGN
midbrain
middle ear
midget ganglion cells
migraine
migration
monosynaptic reflex
mood
motion sickness
movement disorders
Multiple Sclerosis
muscle spindle
muscles
Musician’s Cramp
myelination
myotatic reflex
naproxen (Aleve)
nearsighted
neologisms = new words
neural Darwinism
neural plasticity
neural tube
neurofibrer tangles
neuroleptic drugs
neurotic scratching
neurotrophic factors
nicotine
night vision
NMBA glutamate receptors
nonsense words
nucleus accumbens
nutrition
nystagmus
object features
object recognition
obsessed with objects (skulls)
obssessive-compulsive
occipital lobe
OFC
Olanzapine (Zyprexa)
onset & offset
open or closed head injuries
opioids (opiates)
opponent-process theory of color
opsin
optic chiasm
optic nerve (optic tract)
orbitofrontal
Organ of Corti
ossicular chain
otoconial crystals
otoliths
oval window
Pacinian corpuscles (touch only)
pain
paliperidone (Invega)
parafovea = S cones & rods; sharpish
parallel fibers
paranoid
paranoid delusions
parasol cells
parietal lobe
Parkinson’s disease
parvocellular cells
path-finding
peduncular hallucinations
perceived color
perifovea
perilymph (similar to cerebral spinal fluid)
peripheral neuropathy
perphenazine (Etrafon)
phantom pain
phencyclidine (PCP)
phenytoin (Dilantin)
phonetic processing
photopic system
photopigments
photopsins
photosensitive ganglion cells
physiological zero
pinna = outer ear
pitch
polarization
polymodal nociceptors
poor control of eye movements
posterior parietal cortex
posterior parietal lobe
posterior vitreous detachment or (PVD)
poverty of speech
pre-amplifier
prefrontal cortex
premature birth
pre-motor cortex
pressure
primary auditory cortex
primary motor cortex
priming
primitive streak
problem solving
prodromal = pre-symptoms
progressive disease
proliferation
propriceptors
prosopagnosia = impairment in recognizing faces
pseudo-hexagonal symmetry
pseudovertigo = rotation
psychiatric itch
psychotic disorders
psychotic symptoms
PTSD
pupil of the iris
Purkinje cells
quetiapine (Seroquel)
radial glial cells
radial muscles = open pupil
raw egg whites
receptive aphasia
red nucleus of midbrain
red-green color blindness
reflexes
remapped when eyes move
repetitive behavior
resting potential
resting tremor
restlessness
reticular formation
retina = net
retinal circulatory system
retinex theory of color
retrograde amnesia = before
reversal learning
reward circuit
rheumatoid arthritis
rhodopsin
rhodopsin (visual purple)
rhythm
right fusiform: determines if actual face
right hemisphere: whole picture, face recognition & episodic memory
risperidone (Risperdal)
rod
rod disks
rods
rooting reflex
rostral stream
rotating disk
round window
Ruffini’s end organs
runs in families
s cones (short)
schizophrenia
schizophrenic art
Schlemm’s canal
sclera
S-cones
scotopic system
season-of-birth effect
secondary auditory cortex
seizures = excessively synched neural activity
self control
self medication
self-isolation
semantic memory
semicircular canals
sex differences
sexual dimorphism
SGT (another task)
sharp & dull pain
side effects
silent nociceptors
simple cells
skin mapping
skin stretch
sleep deprivation
slow adaptation
slow vibrations
slow-twitch fibers
smells
smooth muscles
sneezing
social judgment
social pain
social withdrawal
somatosensory associative cortex
somatosensory cortex
sound localization
sparse encoding
spatial summation
split-brain people
stapes
stereocilia
stimulus salience
stretch reflex
striate cortex in occipital lobe
striated
subjective = you’re moving
substantia nigra
suicide risk
superior = anterior (head over heels)
superior colliculi = vision
superior olivary complex
superior temporal lobe
supplementary motor cortex
sustained pressure
symmetrical division
synapse elimination
synapse formation
synaptogenesis
tactile sensations
tardive dyskinesia
target detection
target identification
Tau protein
temperature
temporal lobe
teratogens
tertiary auditory cortex
The Schizophrenias
thermal nociceptors (extreme)
thiamine deficiency
thought disorders
thought insertion
tonotopic map
TOPDV protein is 30x more concentrated in dorsal retina
touch
tremors
trichromatic theory of color
truncus = middle (body)
Truth Telling
ultra violet rays = fast
under-active dorsolateral
undifferentiated
unequal pupil size
unmyelinated
unusual facial expressions
unusual false beliefs
unusual thoughts
utricle (gravity)
V1 = 1st stage of processing
V2 = associations (circle, angles)
V3 = lower visual field
V4 = color & spatial
V5 = motion+
varicella (chickenpox)
ventral path (what)
ventromedial prefrontal cortex
vertigo = whirling, spinning
vestibular sensations (inner ear)
vibrations
viral infections
visual agnosia
visual discrimination test
vitreous humor
vmPFC
voluntary movement
warm = increase firing rate
wave length
Weiss (1924)
Wernicke’s area
word recognition
working memory
working memory for objects
working memory for spatial locations
Writer’s Cramp
X rays = very fast
yellow-blue color blindness
Yoruba people of Nigeria
Young-Helmholtz Theory of color
ziprasidone (Geodon)