McMaster University

Inspiring Innovation and Discovery

KIN 3E03/Life Sciences 3K03

Neural control of human movement

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Fall 2012

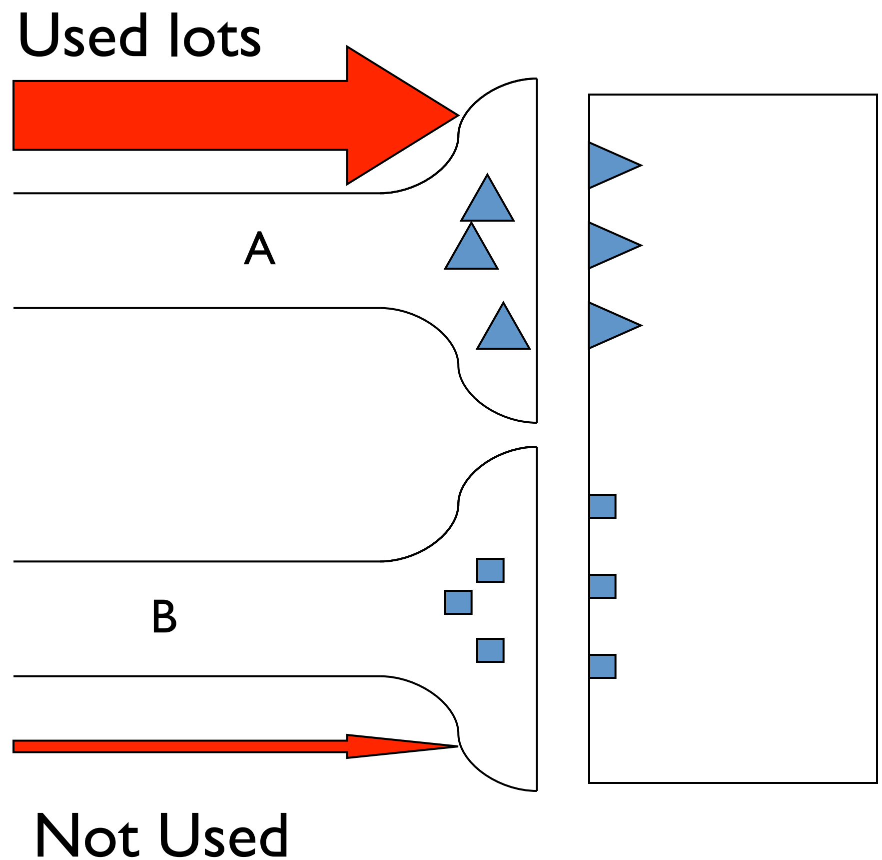
NOTES Unit 3

Neural plasticity

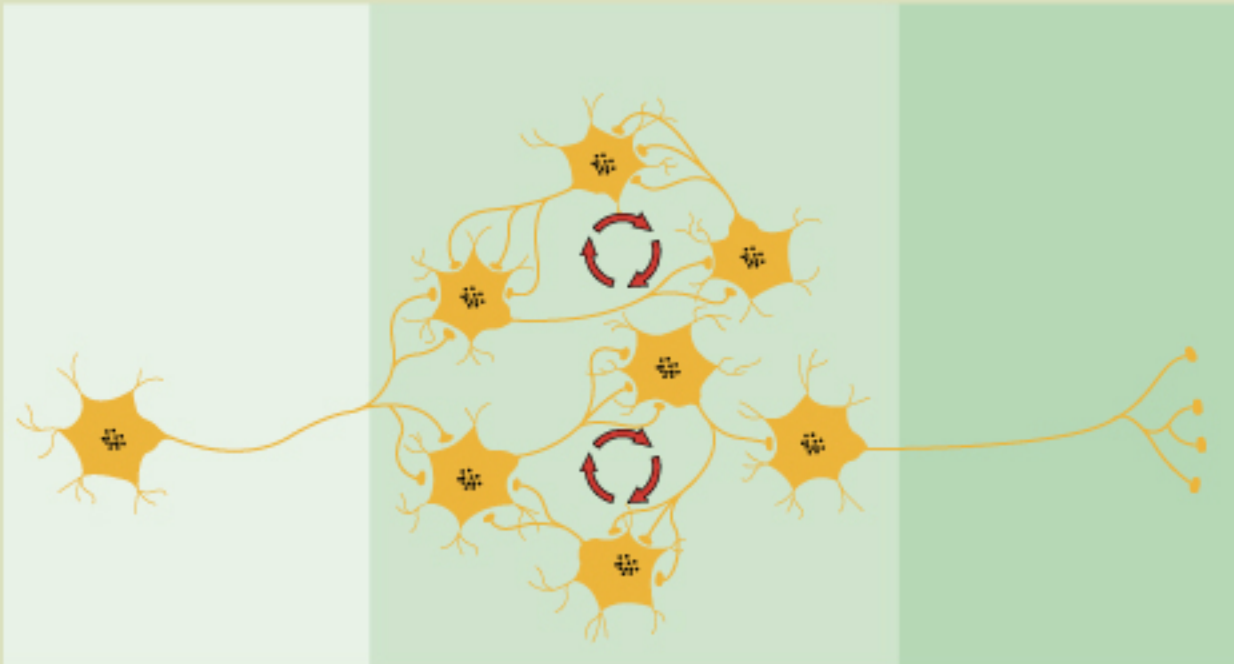
How development, learning and memory change the brain?

Strengthening synapses

- Synapses used often are strengthened (A)
- Those not used are weakened and can become ineffective (B)



Hebb's (1949) Theory of Consolidation



1 Experience activates sensory pathways, which conduct neural impulses to the CNS.

2 **Short-Term Memory**: Hebb hypothesized that the short-term memory of each experience is stored by neural activity reverberating in closed-loop CNS circuits.

Long-Term Memory: Hebb hypothesized that reverberating activity, if maintained for a sufficiently long time, produces structural changes in synapses and that these changes facilitate subsequent transmission over the same pathways.

3 The changed pathways of transmission produced by synaptic facilitation can influence motor output and thus behavior.

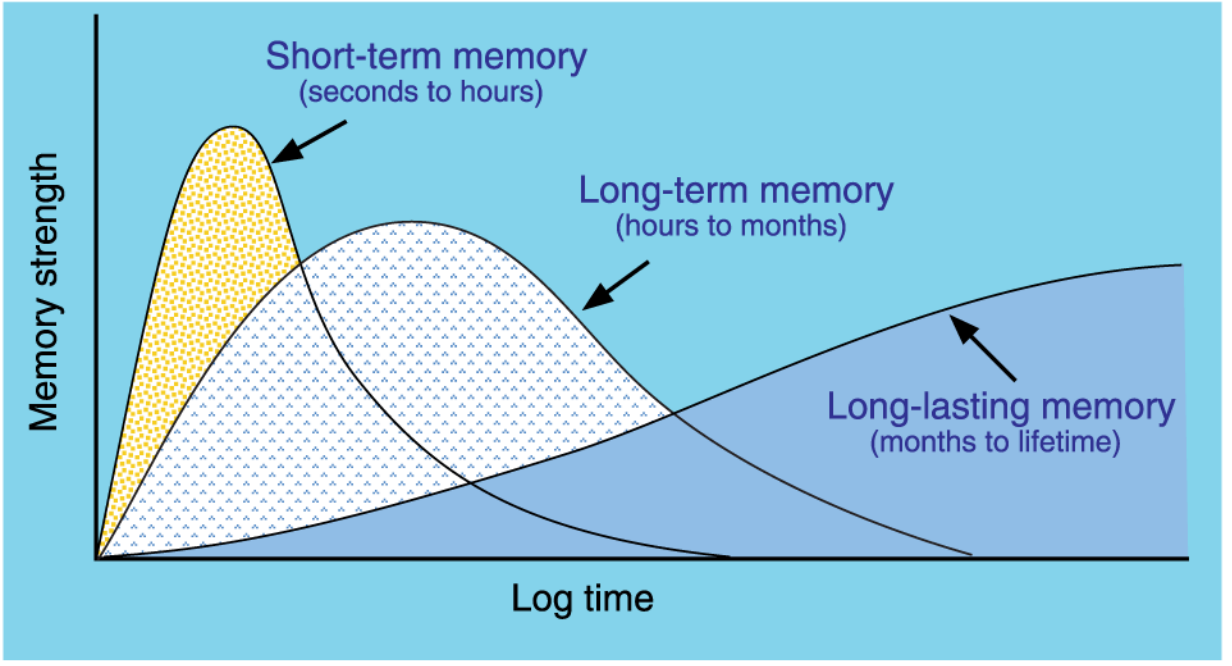


Fig. 1. Memory consolidation phases. Studies of memory and neuroplasticity support Müller and Pilzecker's hypothesis proposing that the consolidation of new memory into long-term memory is time dependent (*1*), but strongly suggest that short-term and different stages of long-term memory are not sequentially linked, as proposed by the dual-trace hypothesis (*9*). Evidence that drugs can selectively block either short-term (seconds to hours) or long-term memory (hours to months) suggests that time-dependent stages of memory are based on independent processes acting in parallel. Later stages of consolidation resulting in memory lasting a lifetime likely involve interaction of brain systems in reorganizing and stabilizing distributed connections.

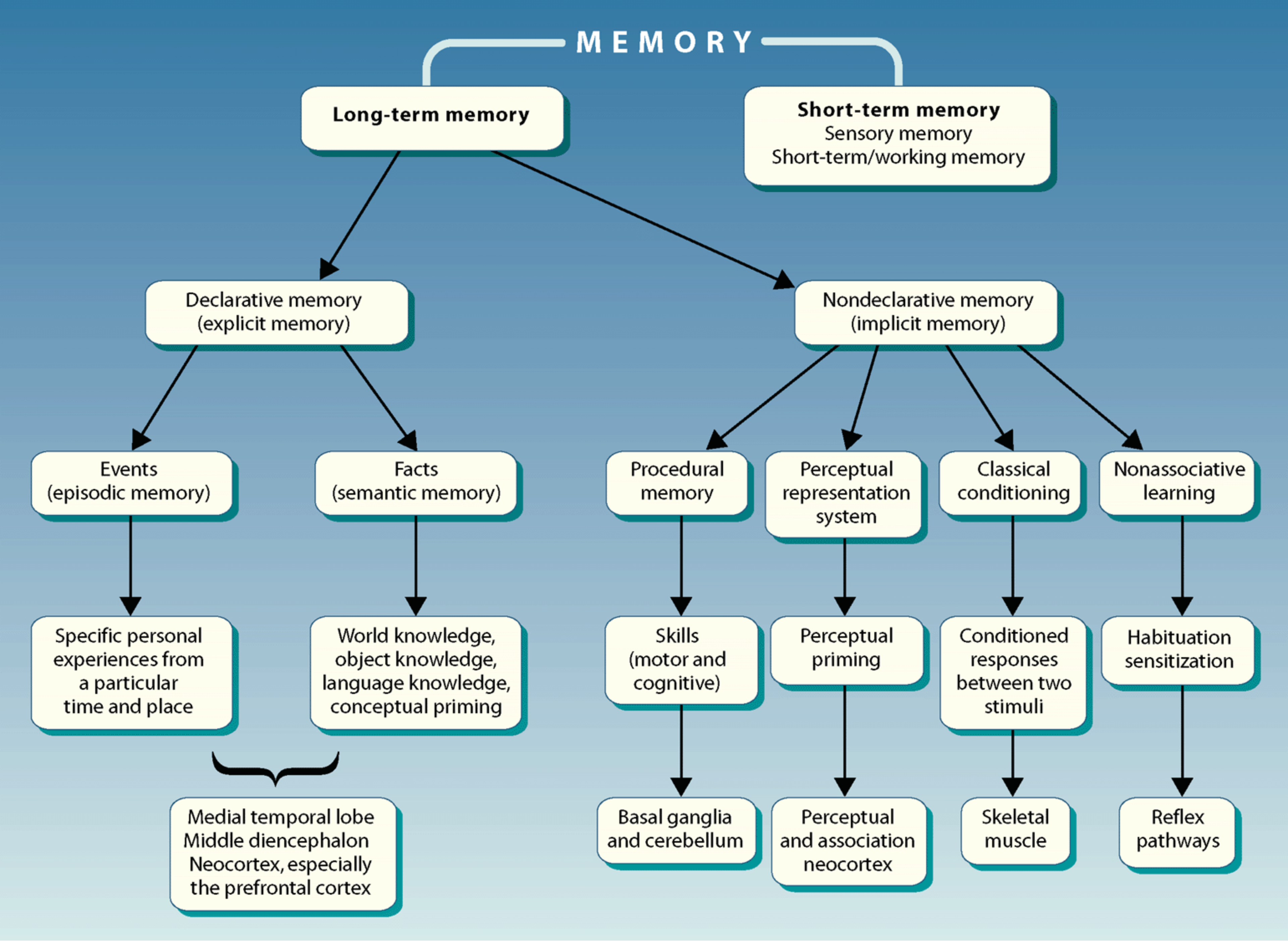
Synaptic changes over in Organism Development

- Over the first few years of life, the brain grows rapidly. As each neuron matures, it sends out multiple branches (axons, which send information out, and dendrites, which take in information), increasing the number of synaptic contacts

- At birth, each neuron in the cerebral cortex has approximately 2,500 synapses.

- By the time an infant is two or three years old, the number of synapses is approximately 15,000 synapses per neuron (Gopnick, et al., 1999).

- As we age, old connections are deleted through a process called *synaptic pruning*.


MEMORY
Long-term memory
Short-term memory
Sensory memory
Short-term/working memory
Declarative memory (explicit memory)
Nondeclarative memory (implicit memory)
Events (episodic memory)
Facts (semantic memory)
Procedural memory
Perceptual representation system
Classical conditioning
Nonassociative learning
Specific personal experiences from a particular time and place
World knowledge, object knowledge, language knowledge, conceptual priming
Skills (motor and cognitive)
Perceptual priming
Conditioned responses between two stimuli
Habituation sensitization
Medial temporal lobe
Middle diencephalon
Neocortex, especially the prefrontal cortex
Basal ganglia and cerebellum
Perceptual and association neocortex
Skeletal muscle
Reflex pathways

Amnesia

Amnesia Partial or total loss of memory, usually resulting from shock, psychological disturbance, brain injury, or illness.

Organic caused by shock, brain injury, illness

- hypoxic episode, herpes encephalitis
- epilepsy, brain injury, Alzheimer's disease

Psychogenic caused by psychological trauma

- dissociative disorders
- psychogenic fugue
- multiple personality or bipolar disorder

Amnesia

Amnesia can be **global** or **material-specific**

Global any kind of information is affected
Material-specific certain kinds of material
(e.g., faces)

Amnesia can be **anterograde** or **retrograde**

Anterograde amnesia inability to learn anything new since the time of the trauma (usually organic)

Retrograde amnesia loss of memory for events prior to the time of the trauma (psychogenic or organic)

HM

Most famous case reported by **Scoville & Milner (1957)**

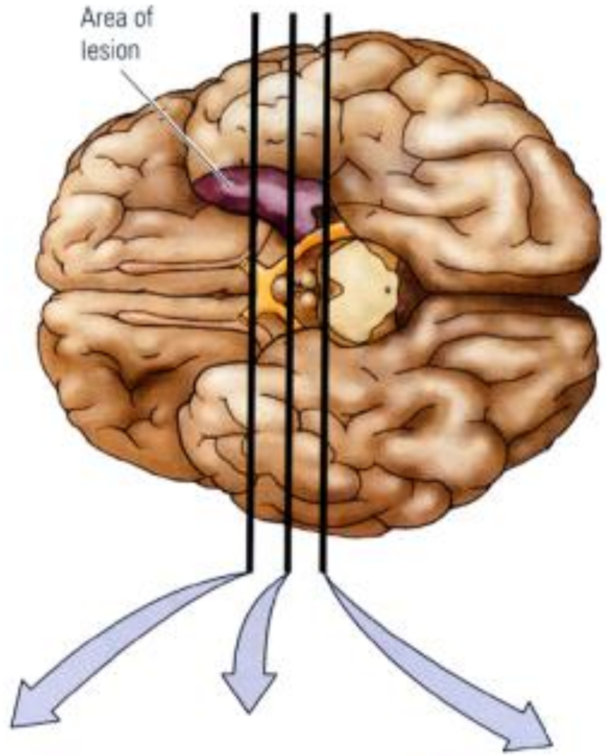
Scoville did the surgeries for psychosis but didn' t work, so tried it for epilepsy on about 30 patients. Patients studied by Brenda Milner

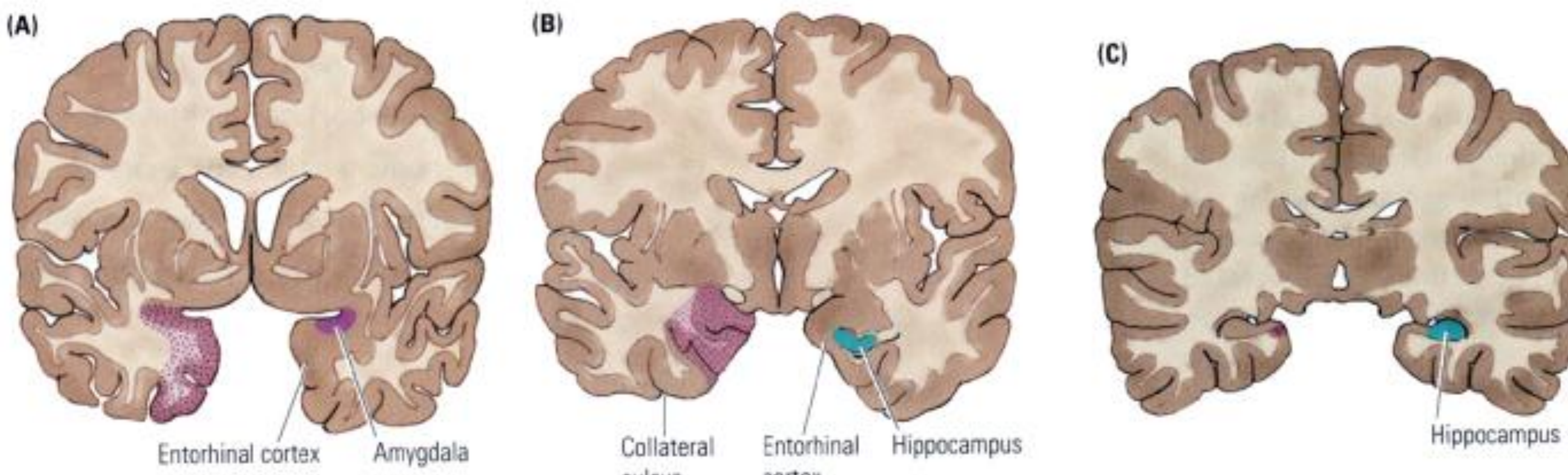
HM: bilateral medial temporal lobe lesion for status epilepticus in 1953

HM's lesion: bilateral medial temporal lobe removal

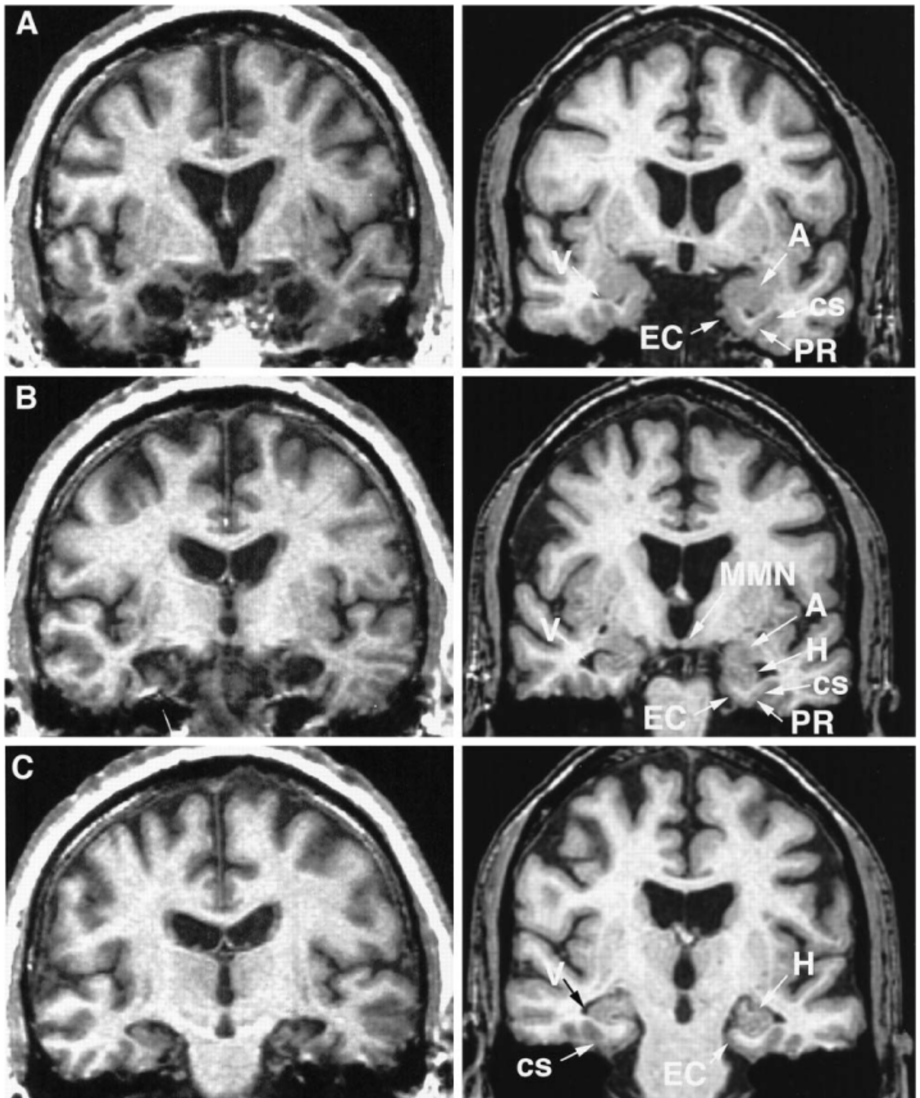
H.M.

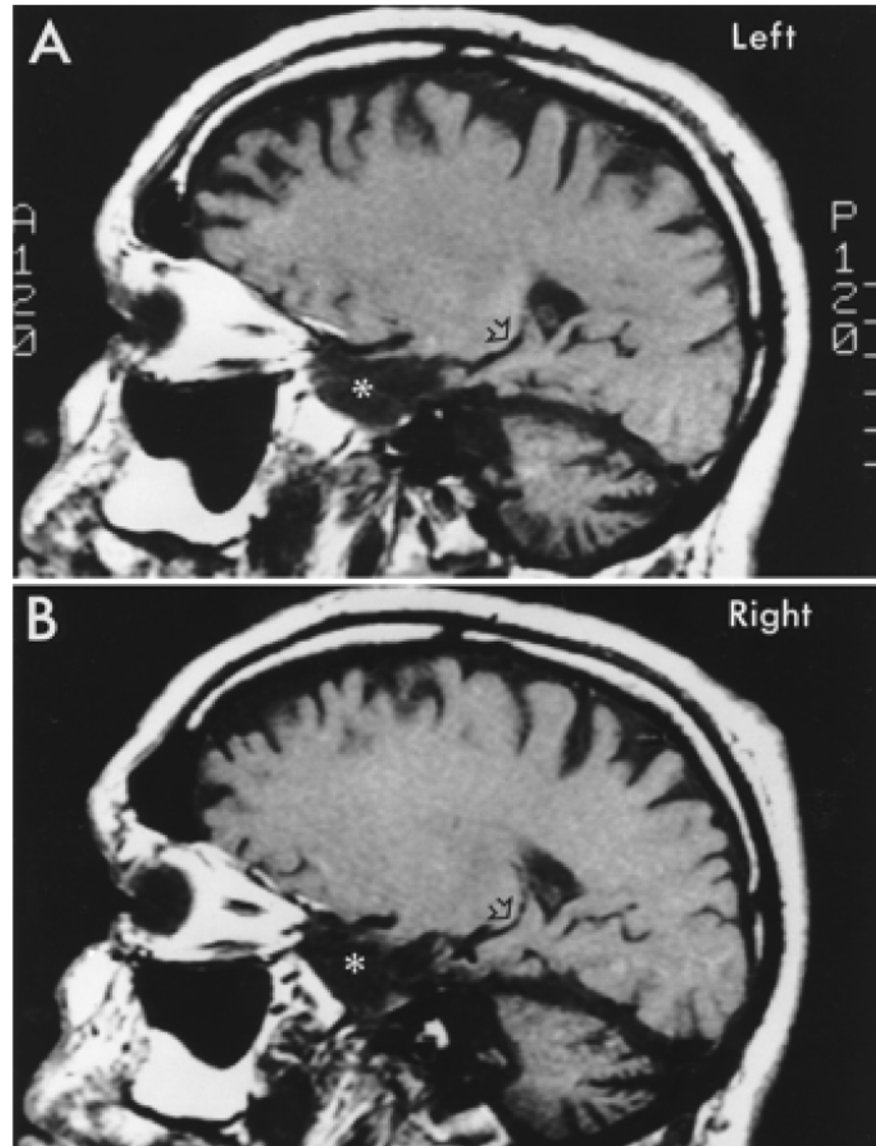
Lesion is bilateral, right side left intact for comparison





HM = no hippocampus, amygdala, overlying (rhinal) cortex





HM's amnesic syndrome

Severe deficit (**global anterograde amnesia**)
- show word or face, ask later, doesn't know
- reads newspapers repeatedly
- doesn't remember own physician
- see on formal tests or everyday life
    - word lists
    - faces and objects
    - recall or recognition

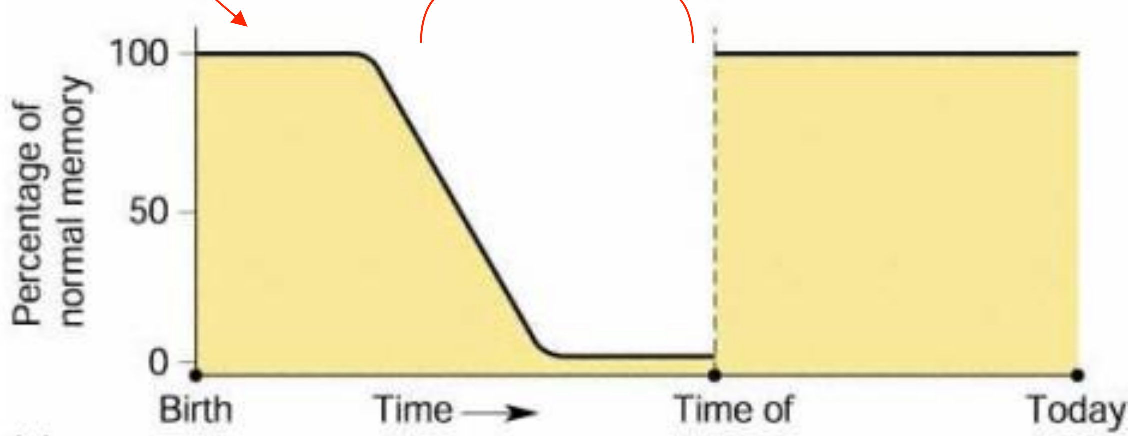
Only mild **retrograde amnesia**
- loss of memories that are a up to 2-3 years old at the time of the lesion, but childhood memories in tact
- known as a **time-limited** or **temporally-graded retrograde amnesia**

Retrograde vs. anterograde amnesia

normal memory for remote events (childhood, etc)

for HM, retrograde amnesia is approx. 2 yrs

- Retrograde amnesia

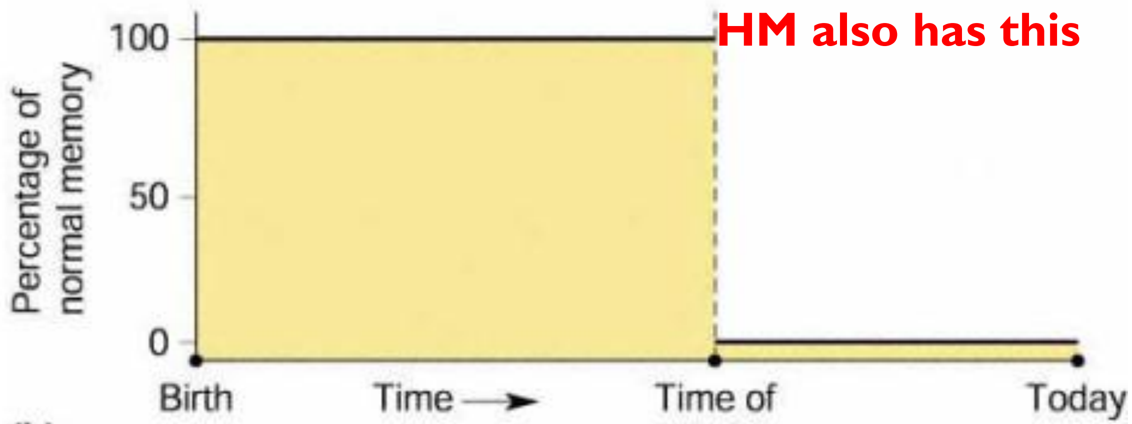


(a)

Retrograde and anterograde amnesia can occur together or separately (HM has both)

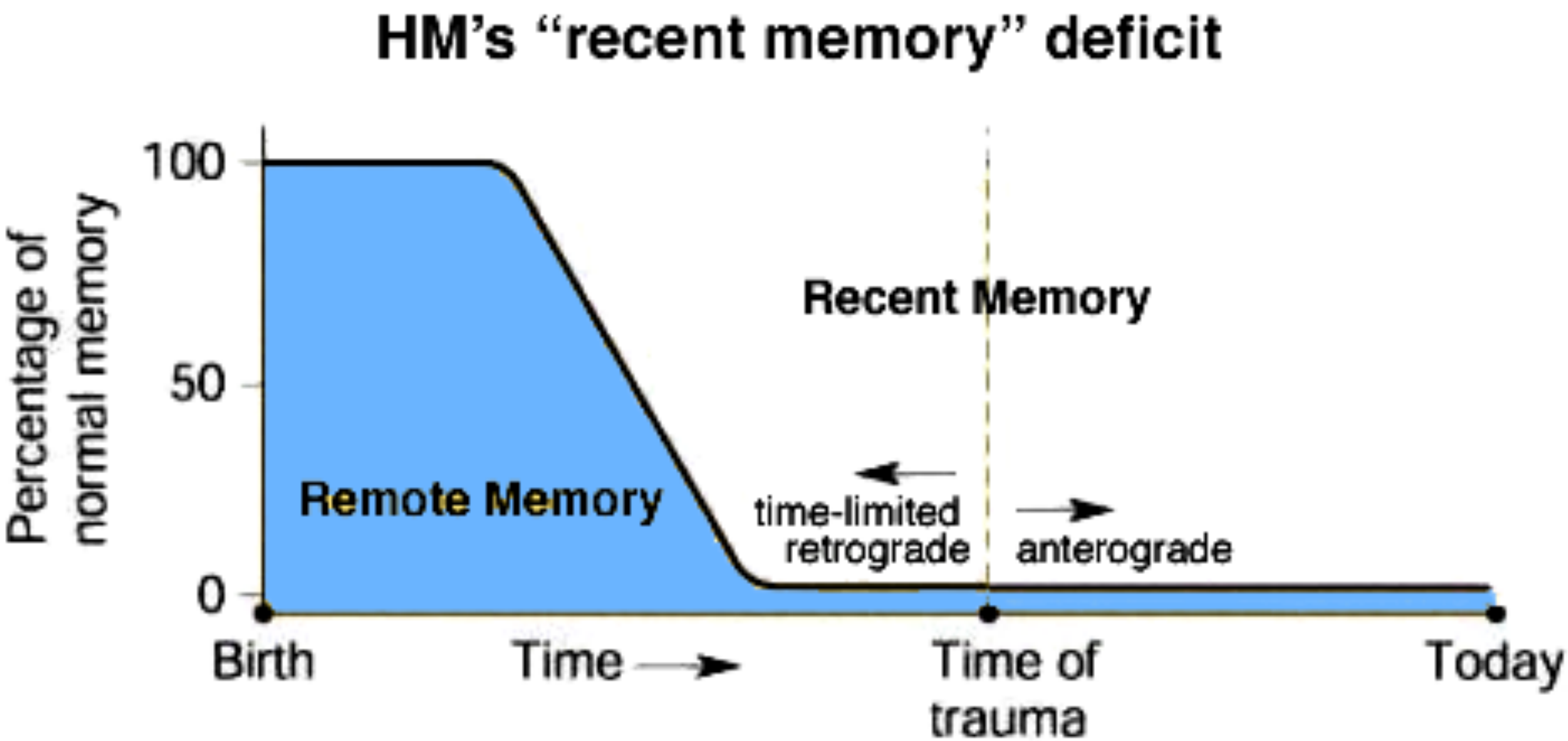
HM also has this

- Anterograde amnesia



(b)

Remote versus Recent Memory:
HM, Loss of Recent memory



Rey-Osterrieth Figure

3946 J. Neurosci., May 15, 1998, *18*(10):3943–3954 Reed and Squire • Retrograde Amnesia

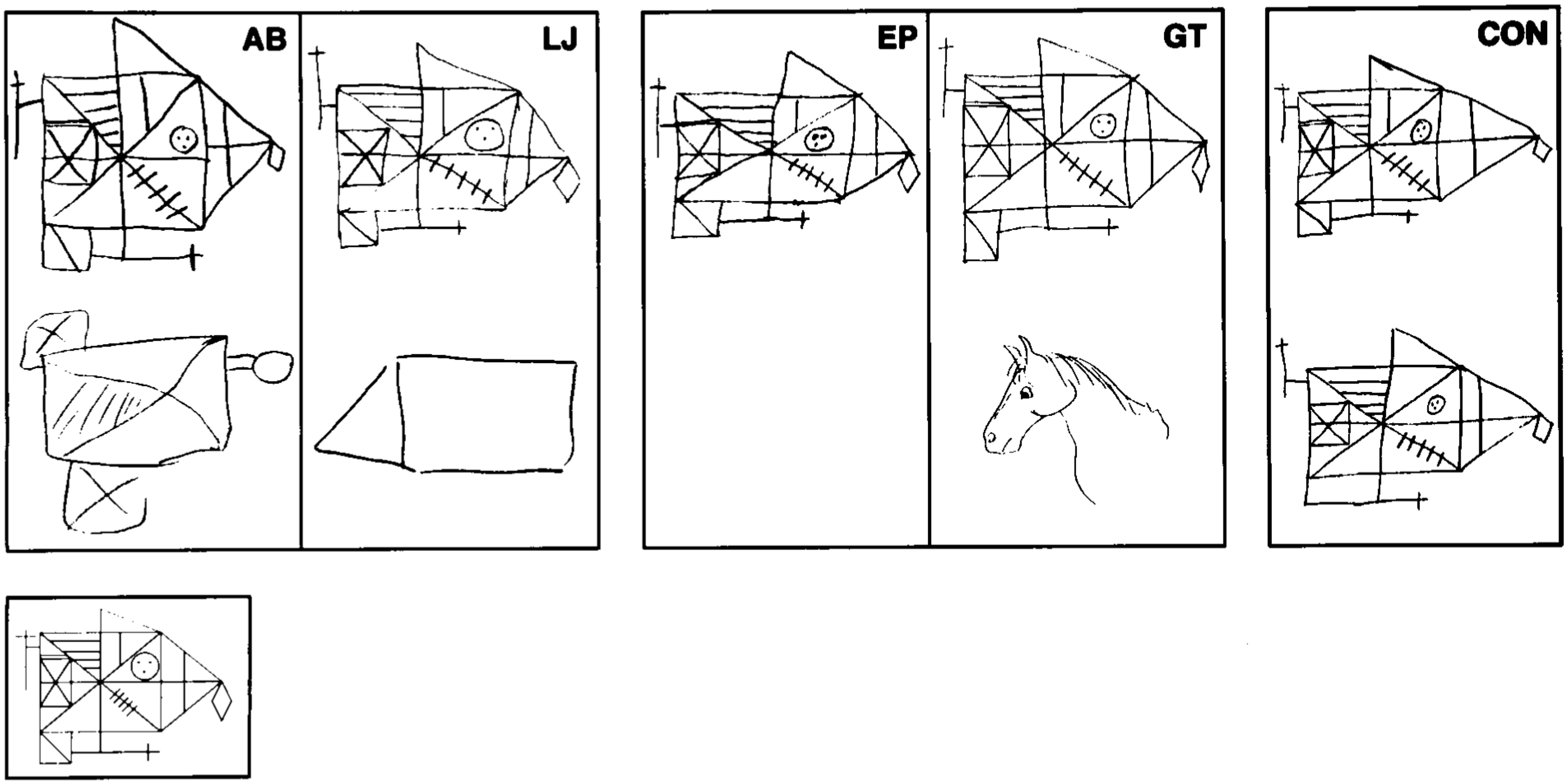


Figure 2. The Rey–Osterrieth figure. Patients were asked to copy the figure illustrated in the *small box* to the *bottom left* and, 5–10 min later, to reproduce it from memory. The copy (*top*) and the reproduction (*bottom*) are shown from *left* to *right* for the four patients and for a representative control subject (*CON*). Neither E.P. nor G.T. recalled copying the figure. Encouraged to draw whatever came to mind, patient E.P. declined to try and G.T. produced a drawing of a horse's head.

Other forms of amnesia

Unilateral hippocampal damage results in material-specific deficits

left = words, names, etc.

right = faces, objects, etc.

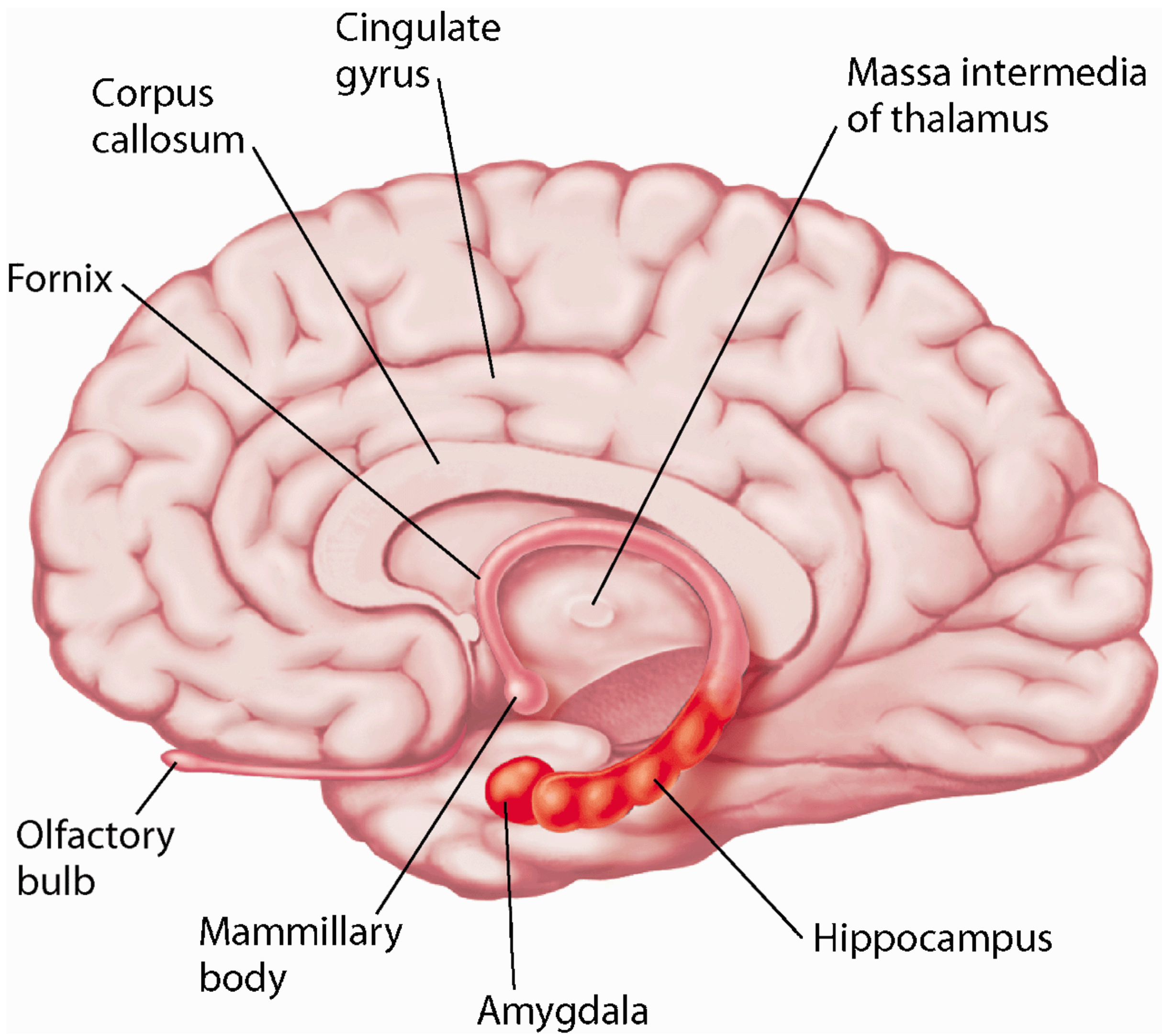
Korsakoff' s psychosis results in similar amnesia plus **confabulation (fill in memory gaps with fabricated stories)**

caused by alcohol poisoning

Frontal lobes STM, working memory, temporal order, confabulation

Electroconvulsive Therapy (ECT,ECS)

Location of the hippocampus



Patient with damage to the hippocampus.

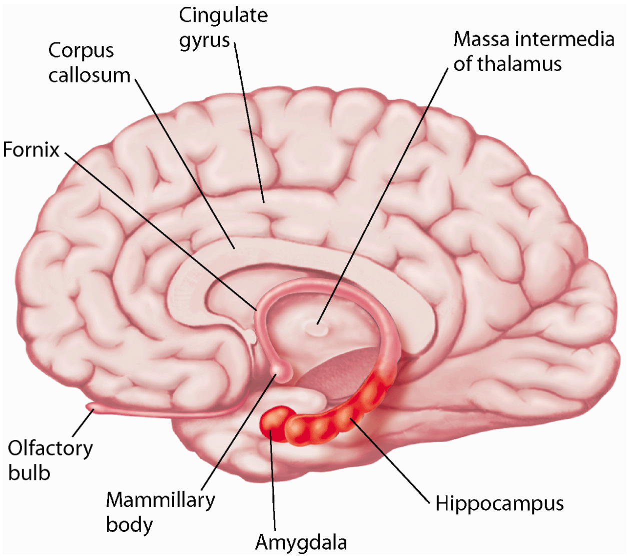
Capgras Delusion

• 'Mme M.' believed that her husband, children, neighbours and others had been replaced by doubles (Capgras 19thC).

•Ramachandran's patient in San Diego called PK (2001).
•He believed that his mother has been replaced by an impostor.

•Cases have been described where the delusion that pets or even inanimate objects have been replaced by replicas.

Face recognition



Analysis of visual form

Capgras?

Capgras strikes in the area between the Amygdala and Hippocampus

Identification

Somatic / Emotional response

MEMORY STRATEGIES

MI K TPUXC GYI OSFP SH DFKUXC RWJF

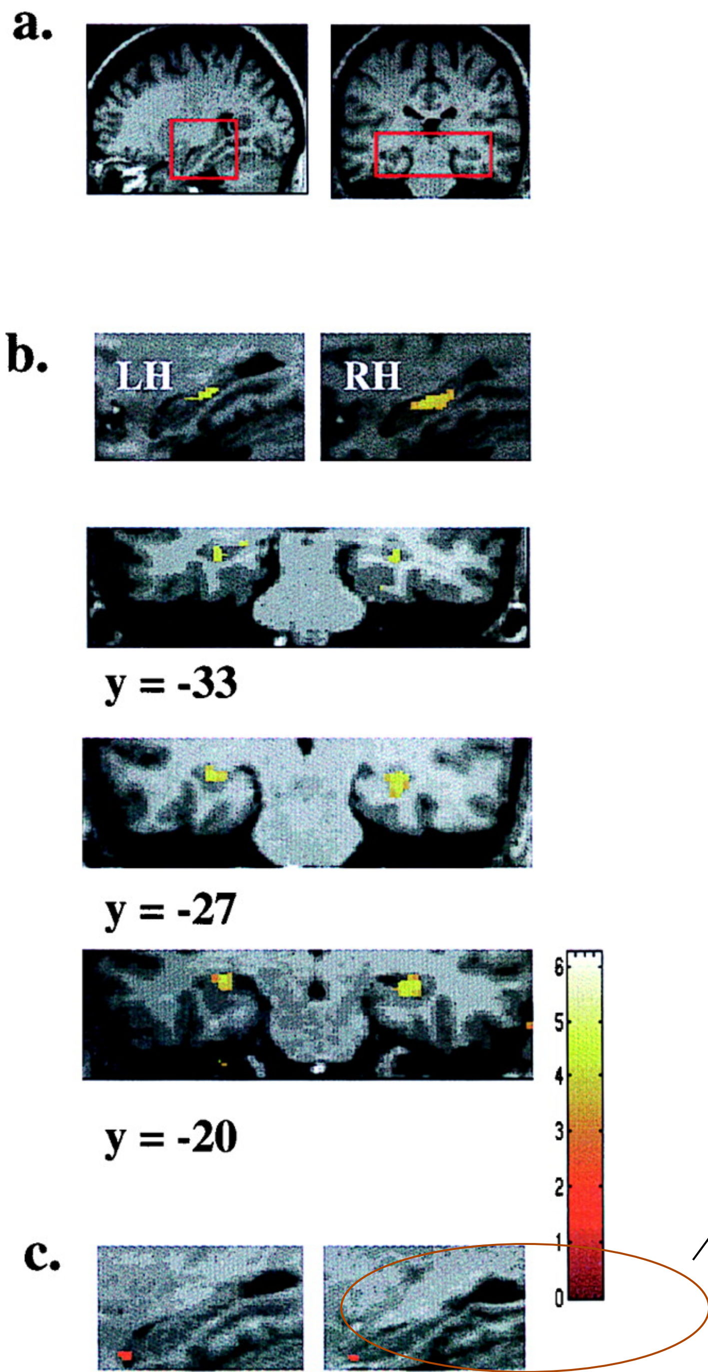
MY MEMORY FOR WORDS IS RATHER GOOD

↓

Better encoding hence better retrieval

Memory is also quite sequence specific

ALPHABET EXAMPLE..



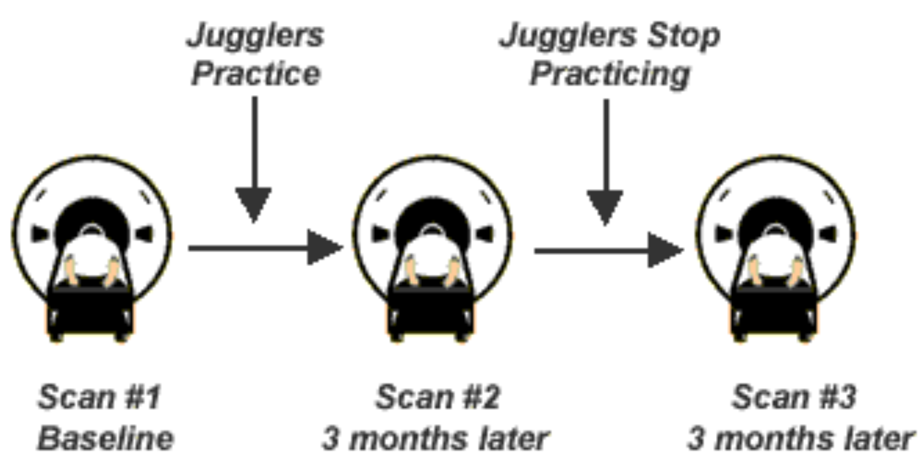
Structural changes in the brain are seen as a consequence of long-term activity.

What about skills like sport? Do they cause changes in the brain?

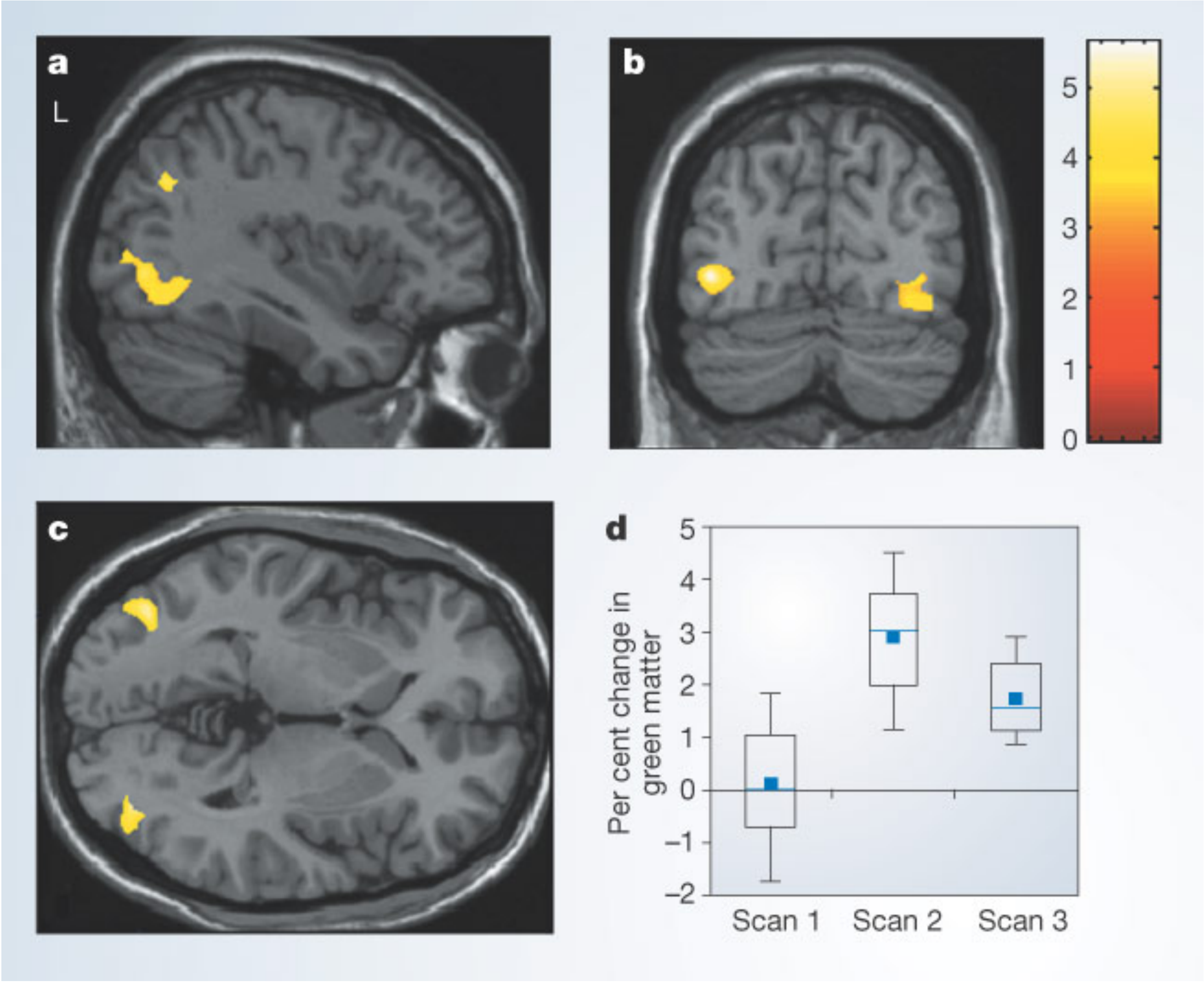
London cabbies have more hippocampal gray matter

Learning to juggle

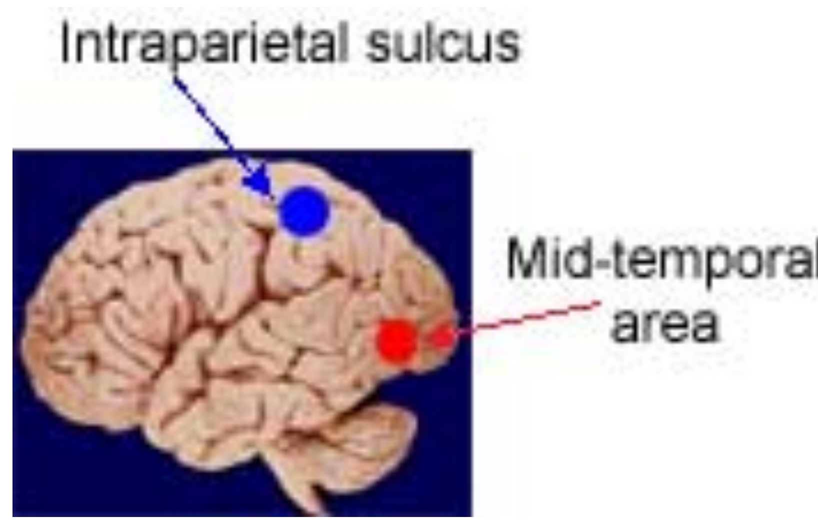
- Juggling Group: 12 subjects learned a three-ball cascade juggling routine. They were considered to be skilled jugglers when they could juggle for 60 seconds.
- Non-Juggling Group: 12 subjects had no juggling practice.



The brain of a juggler



These data suggest that learning new skills can alter brain structure. However, it is unclear what exactly caused the brain changes. The expansion in the two brain areas may have been caused by an increase in the number of nerve cells, glial cells or synapses.



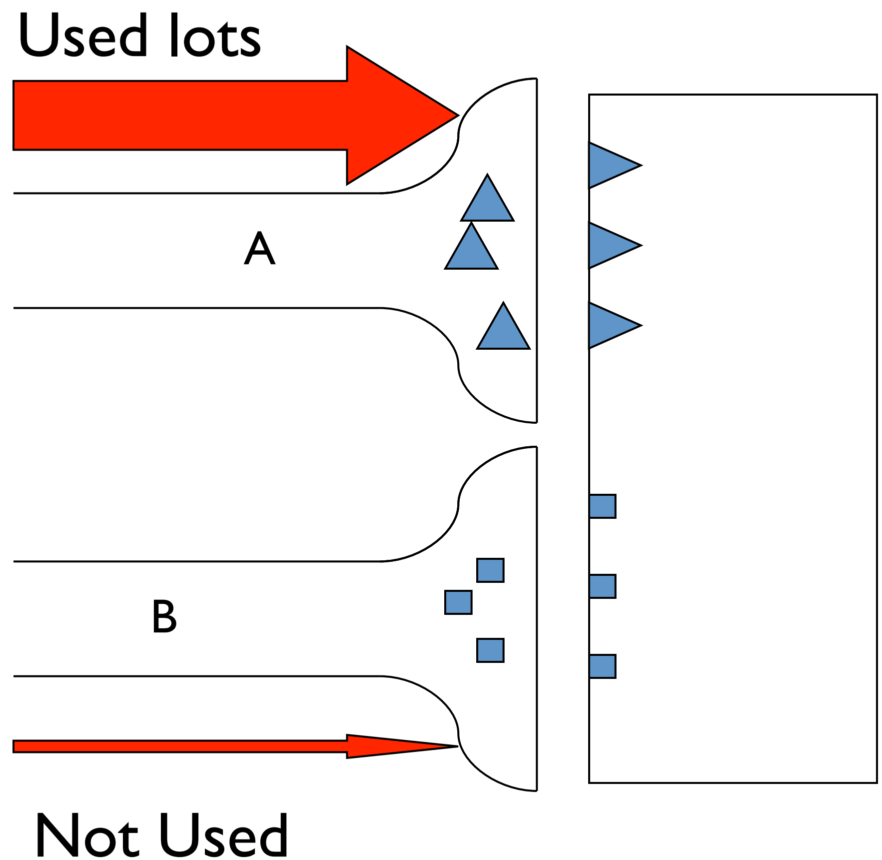
One more time…

- Memory & consolidation
- Amnesia and localization of memory
- Learning: changes in the brain
- Learning, consolidation and memory
- Changes in the brain as a function of learning
- London cabbies and jugglers

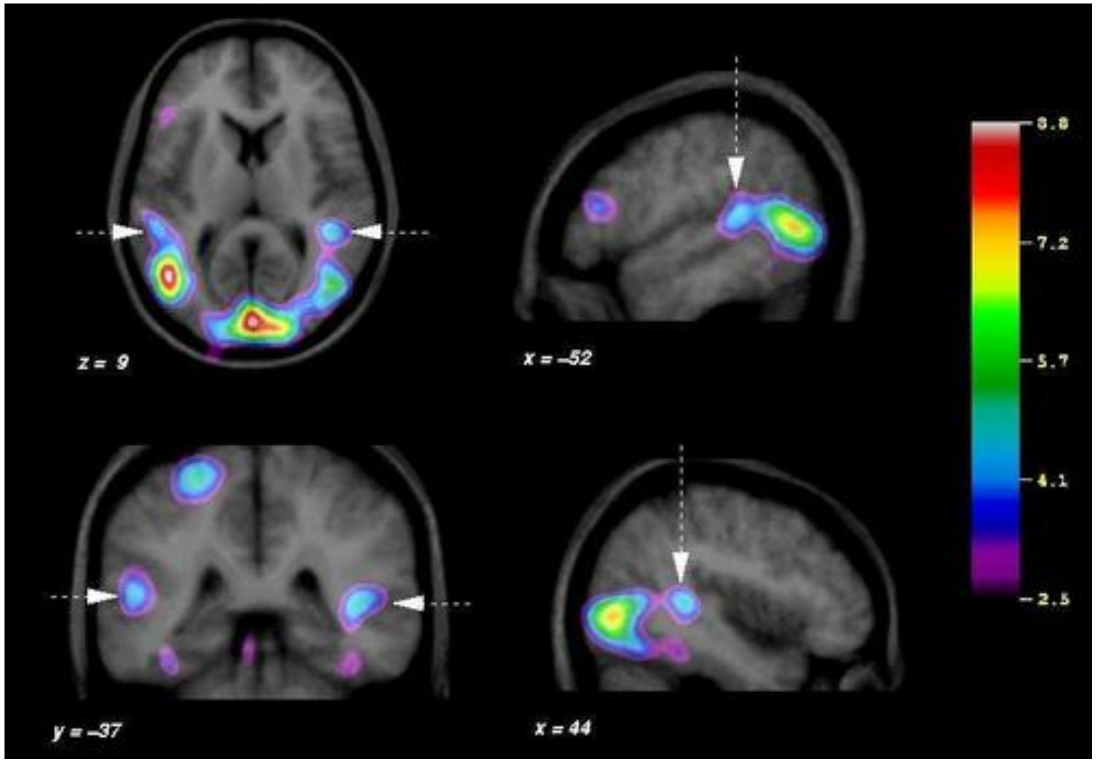
- NEXT: plasticity

Strengthening synapses

- Synapses used often are strengthened (A)
- Those not used are weakened and can become ineffective (B)



Cross-modal plasticity in congenitally deaf



- These PET/MR images show increased neural activity in the superior temporal gyrus in congenitally deaf subjects when they viewed signs or sign-like movements, suggesting that auditory cortical regions may contribute to the processing of visual information in the deaf

Optic nerve hypoplasia

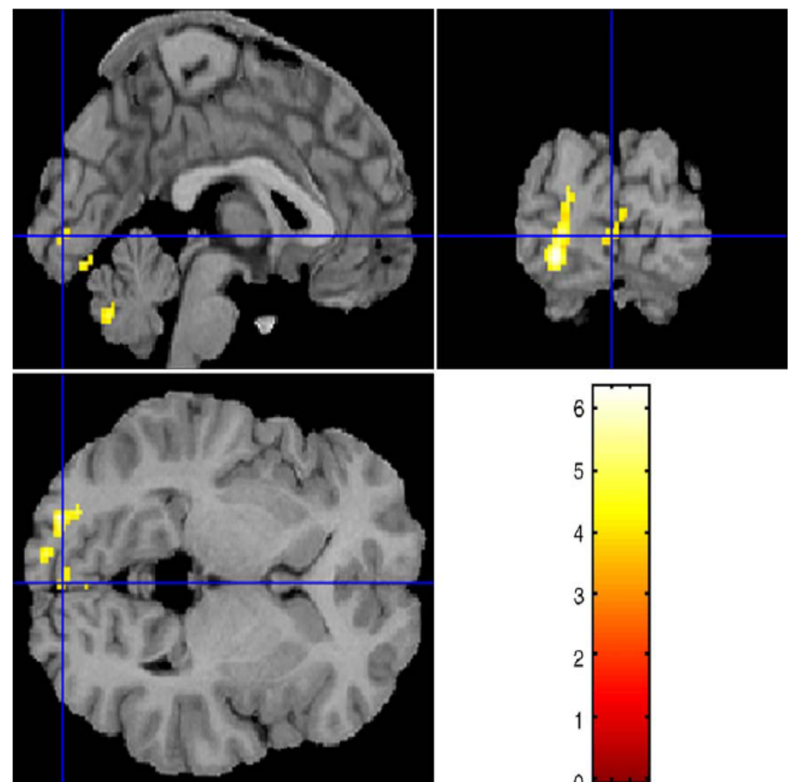
- Sometimes referred to as ONH autism
- Or DeMorsier' s syndrome
- Congenital blindness (optic nerve to one or both eyes impaired before birth)
- Extremely sensitive to sound; for example have perfect pitch.
- Musical savants can have ONH.

VIDEO OF A MUSICAL SAVANT

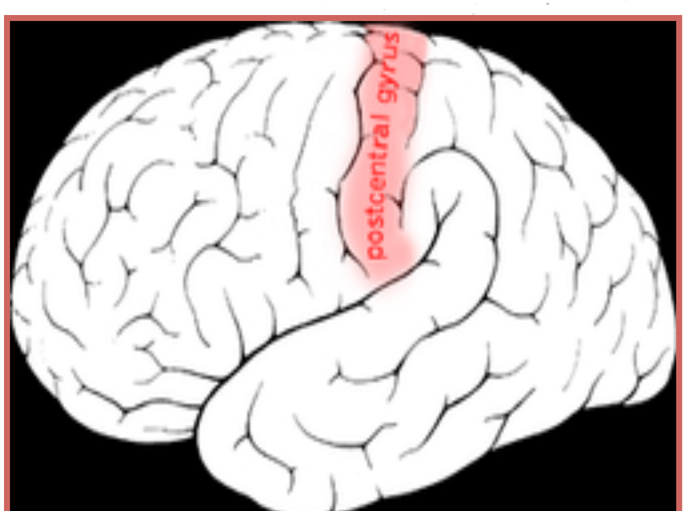
Auditory Motion Perception

- Early blind subjects show activation of the visual cortex during an auditory motion tracking task
- Visual areas become involved in auditory processing
- Functional Re-mapping of the cortex

Poirier et. al. (2006)



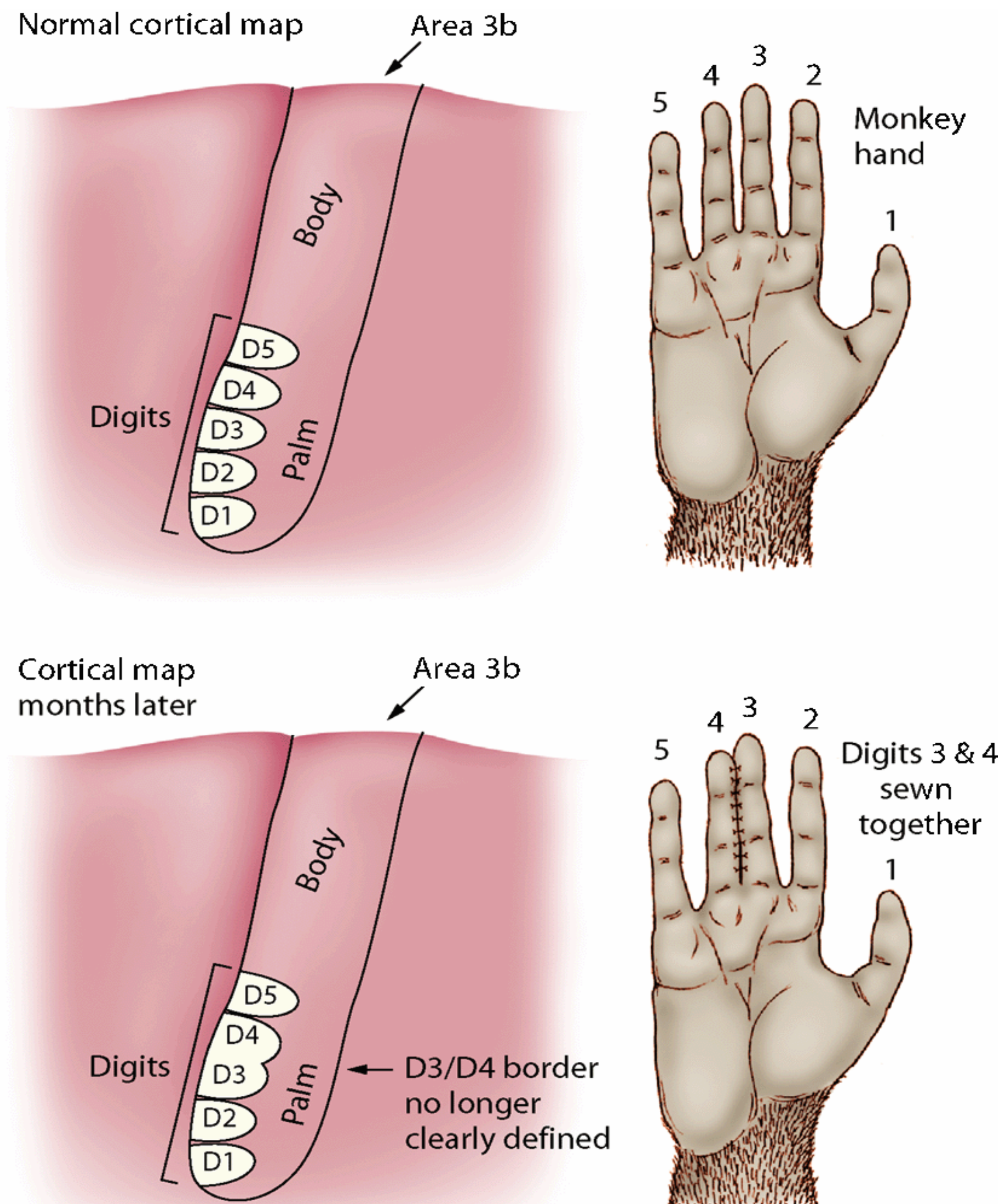
Re-Writing the Cortical Map



- Primary Sensory Cortex (S1)
- Located posterior to the central gyrus (behind M1)
- Arranged in a somatotopic map – the sensory homunculus

Cortical Re-Mapping

- Motor cortical areas for each digit mapped
- Digits 3 and 4 are sewn together (now functionally act as one digit)
- Months later, the cortical areas for D3 and D4 have merged

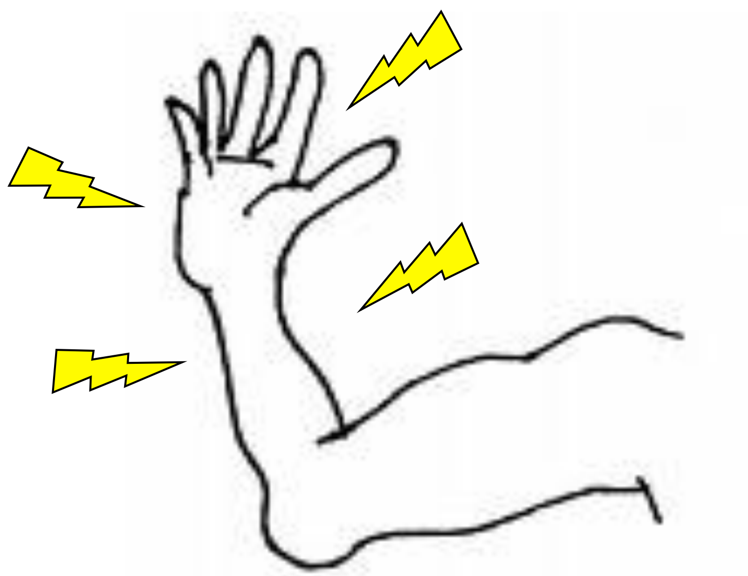


Plasticity and phantom limbs

- Re-mapping and Rewiring of the cortex seems to occur under many circumstances
- Imaging studies have shown massive cortical re-organization
- Somatotopic map can redraw itself in response to new circumstances
- This is called **Plasticity of the brain** or **Neuroplasticity**

What are Phantom Limbs?

- **Phantom limbs** – the vivid impression that a lost limb is present; can be painful and distressing



Clinical Phenomena

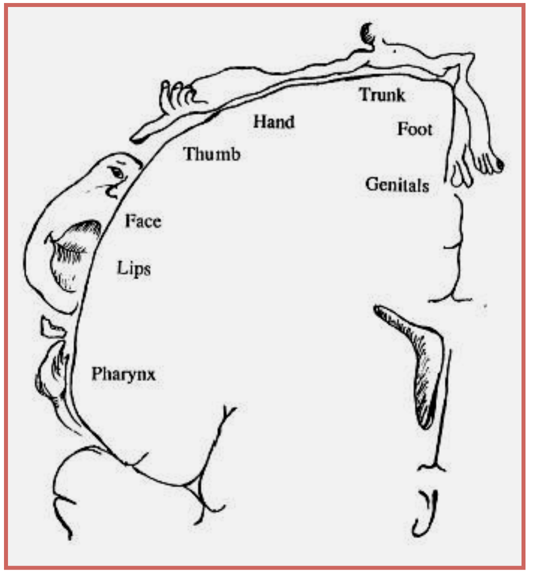
- Phantom limbs have been observed throughout history (Moby Dick – Capt. Ahab)
- 1st clinical description during the American Civil War -*The case of George Dedlow* by Dr. Silas Weir Mitchell

Clinical Phenomena

- Incidence: 90 - 98% of patients who lose a limb experience phantom sensations
  - Less prevalent in early childhood (perhaps because brain topography is changing)
- Onset: Immediate in 75% of cases
- Duration: Few days to decades (57 yrs!)
- Body Part: Arm, leg, breast, face, organs
- Posture: “habitual” posture, can change

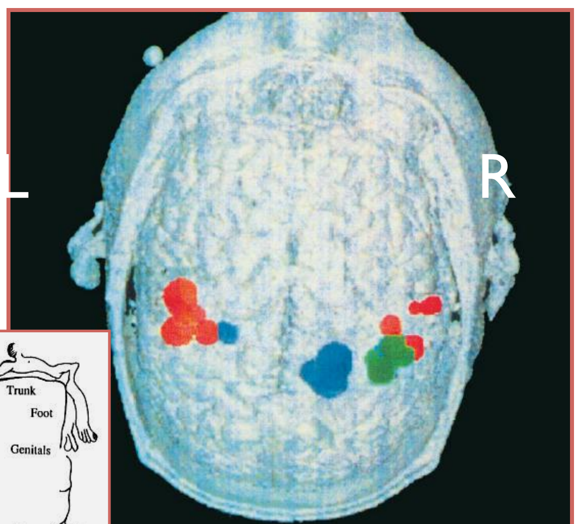
Experimental Studies

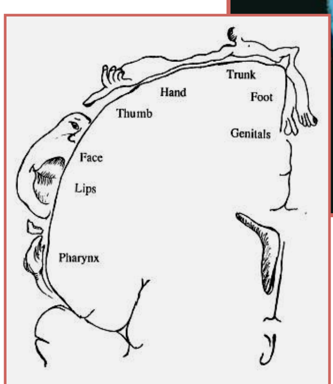
- Primate Amputation
  - When middle finger was amputated, motor cortex for that finger started responding to adjacent finger stimulation within months (Merzenich et al., 1984)
  - After 12 years of arm amputation, the "hand cortex" responded to stimulation on the face (Pons et al., 1991)



Experimental Studies

- Human Brain Imaging
  - Patients with arm amputations demonstrated activation of the “hand cortex” when the face and upper arm were stimulated (Ramachandran, 1993)





MEG scans from a patient whose right arm was amputated. Right hemisphere activity is normal, while left activity shows reorganization.

Red = face, Green = hand, Blue = upper arm

Referred Sensation

- Ramachandran was able to elicit sensation in the amputated arm (the phantom limb) by stroking the patient’s face

Experimental Studies

- Referred sensations of phantom fingers map onto the face (Ramachandran, 1993)

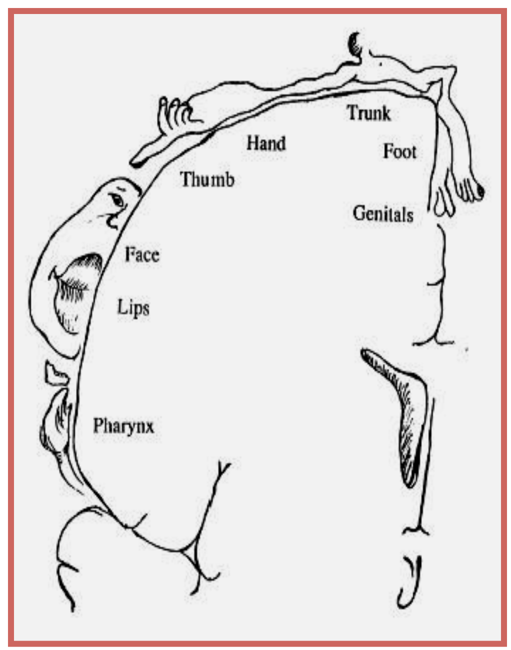
Left face regions elicited localized sensations in the phantom fingers.

P = pinkie, I = index finger, T = thumb, B = ball of thumb

Experimental Studies

- Referred sensations also map onto upper limbs (shoulder)

- Mapping onto adjacent cortical areas



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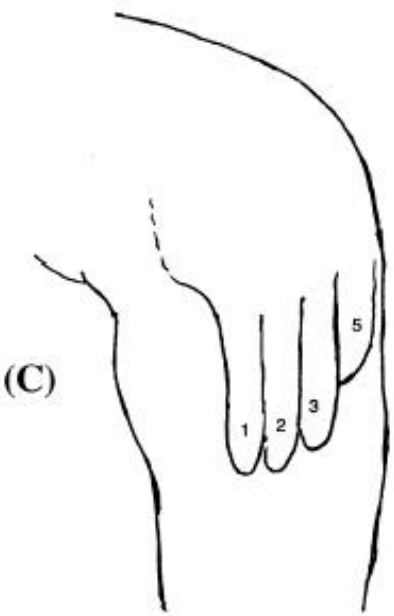


Phantom Limb Treatment

- Painful sensations (e.g. clenched fists, spasms) can be treated with a **mirror box**
- With treatment, the phantom limb can change its posture or disappear entirely

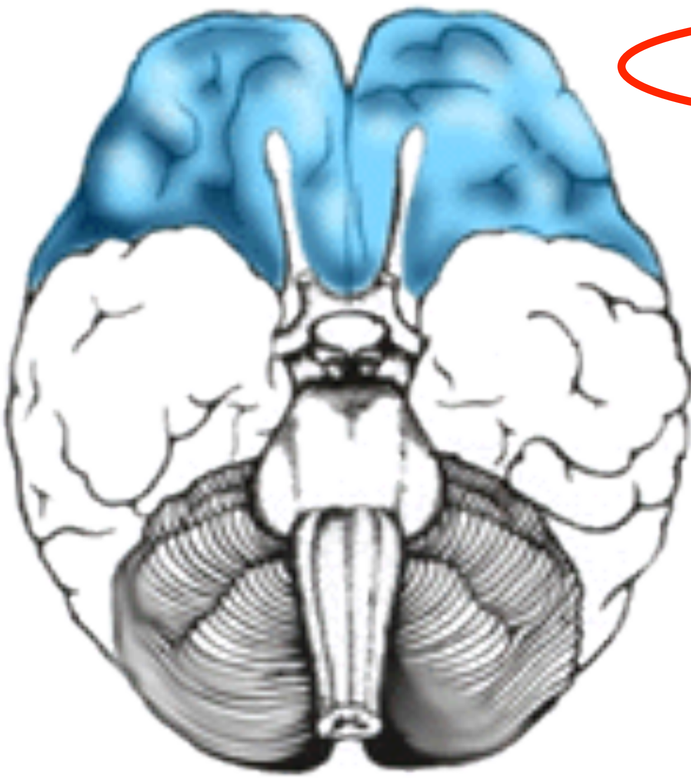
Phantom Limb Implications

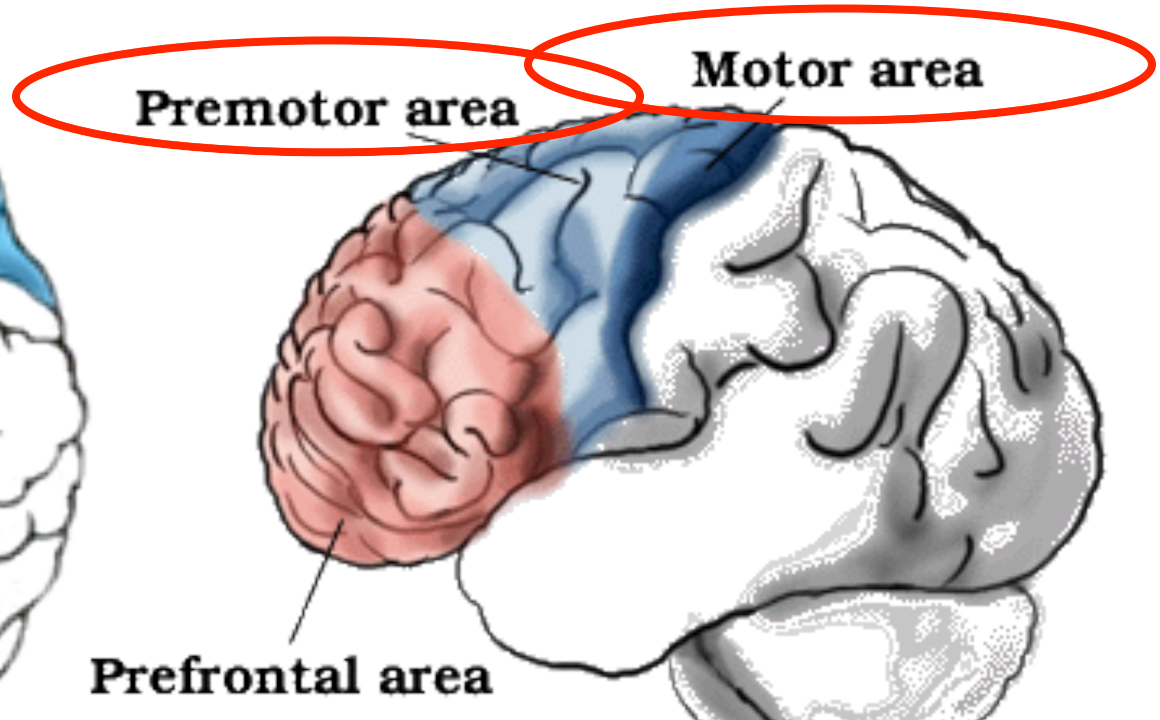
- Brain **topography** is **dynamic** and neural organization can be altered (Neuroplasticity)
- Phantom limb sensations are **real**, they have identifiable neural correlates

Phantom Limb Implications

- Painful and distressing phantom limbs can be treated with visual feedback
- Increasingly studied in the USA as a result of Afghanistan and Iraq wars
- Body image is a transitory internal construct

Anatomy of the Frontal Lobes





Dorsolateral PFC

Ventral/Medial Orbital PFC

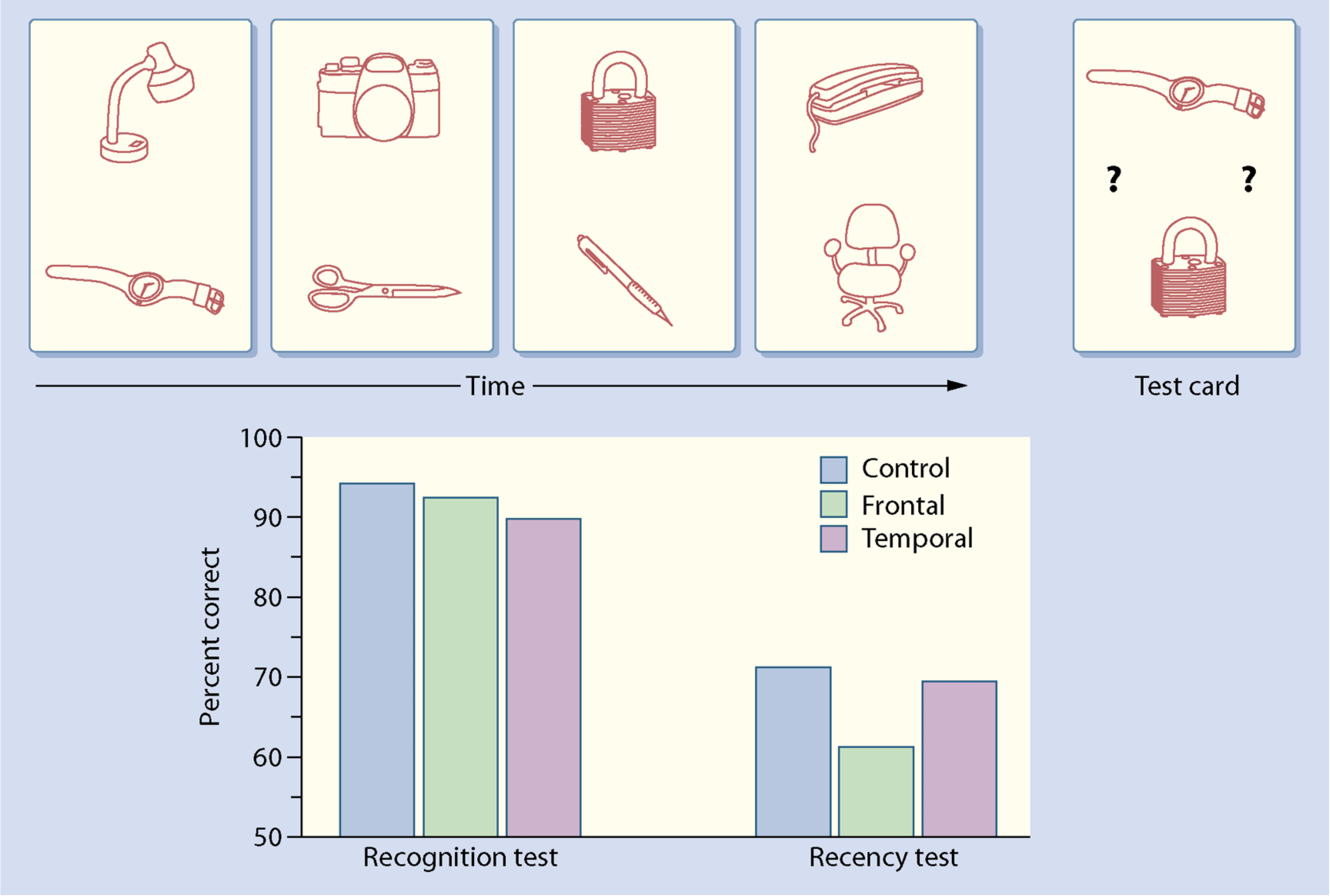
Some symptoms associated with frontal lobe damage

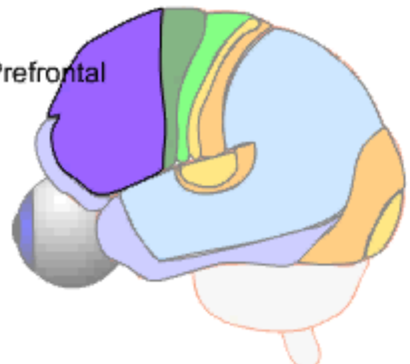
- Working memory deficits
- Temporal memory / Source memory
- Perseveration
- Loss of spontaneous behavior
- Apathy
- Planning deficits/impaired goal-directed behavior
- Disinhibition/impulsive behavior
- Impaired attention
- Depression
- Elevated mood

Memory-related deficits

- Working memory: delayed response tasks
- Temporal memory: when did the event occur?
- Source memory: where did I get this information from?

Temporal memory example





Prefrontal association area
Function: motor planning through the use of working memory

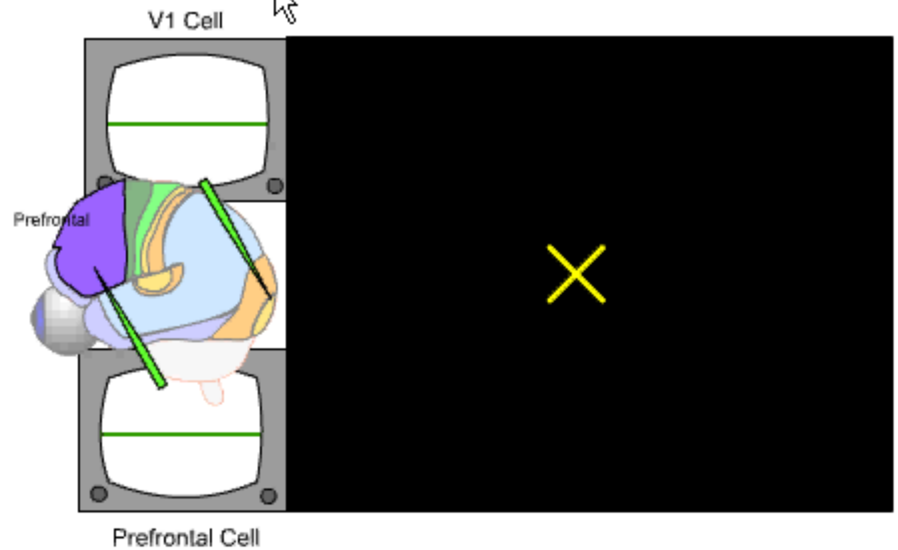
An example of spatial working memory.

Can you remember where the egg is?

If you cannot you may have a lesion of the prefrontal association areas. Lesions here produce deficits in tasks that are spatial and delayed.

Children prior to the age of 1 yr
have not developed this working memory.
If a toy is covered by one of two covers, the child cannot find it.
Out of sight is out of mind.

Neurons show
a) activity which starts when a stimulus appears in a particular location
b) unlike neurons in V1, here activity continues even when the stimulus disappears.
This tonic activity holds the object location in working memory.
Different cells hold the memory of objects in different target locations.



THE STROOP EFFECT

RED
BLUE
GREEN
RED
BLUE
YELLOW
BLUE
GREEN
RED
YELLOW

RED

BLUE

GREEN

RED

BLUE

YELLOW

BLUE

GREEN

RED

YELLOW

Inhibition-Related Deficits

- Stroop task (unable to do this task).
- Perseveration
  - Wisconsin Card Sorting Task
- Social Disinhibition/Impulsivity
- Loss of spontaneous behavior
- Poor planning/goal-directed behavior
- Attentional deficits

Personality Changes in FL Dementias

- Impairments in social skills
  - inappropriate or bizarre social behavior
  - “loosening" of normal social restraints (e.g., using obscene language or making inappropriate remarks)

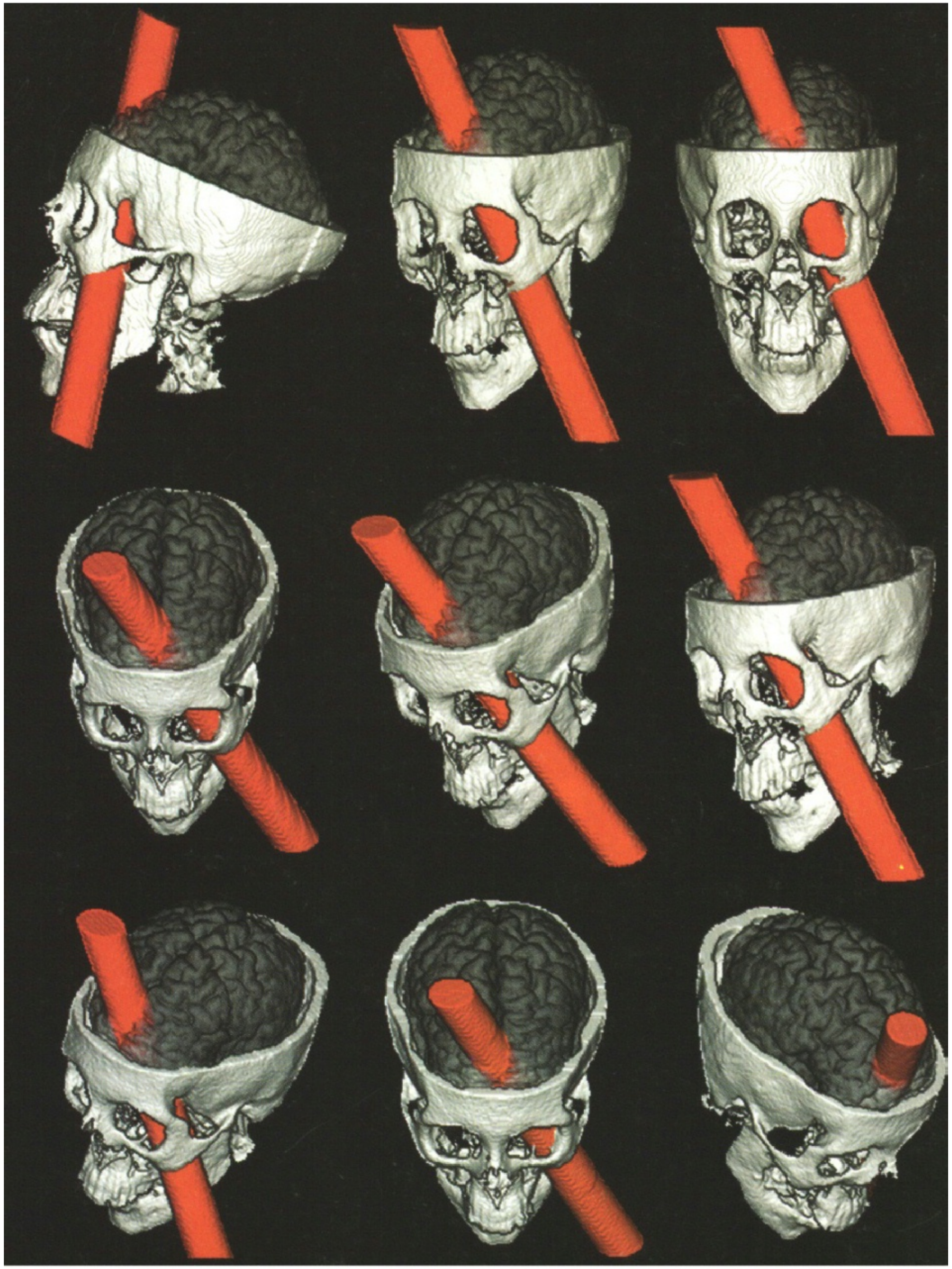
- Change in motivation
  - apathy, withdrawal, lack of interest, and initiative which may appear to be depression but the patient does not experience sad feelings.

Personality Changes in Anterior Strokes (2000, *Neurology*)

- Depression
  - In 75% of anterior CVA pts
  - In 50% of TL CVA pts
- Emotional Problems
  - Present in 18% of stroke pts
    - In 100% of anterior CVA pts
    - In 0% of pts with TL or OL CVA
- Presence of post-stroke depression and emotional ability is greatly influenced by lesion location

Phineous Gage

- Railroad foreman
- Well-respected, hard-working
- 1848: tamping iron accident
- He never lost consciousness, and had no obvious neurological symptoms
- But he was “no longer Gage”

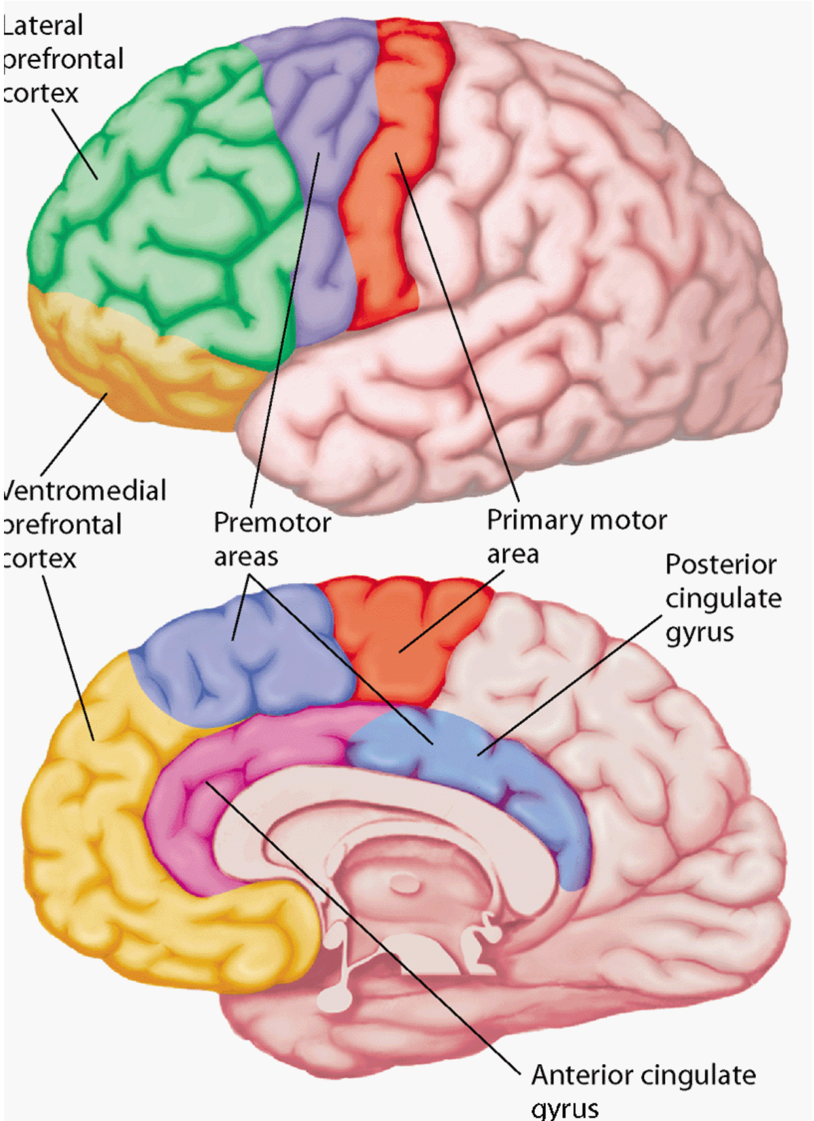


Phineous Gage

Gage's Doctor described Gage's post accident personality as

Fitful, irreverent, indulging at times in the greatest profanity which was not previously his custom, manifesting but little deference for his fellows, impatient of restraint and advice when it conflicts with his desires, at times pertinaciously obstinate, yet capricious and vacillating, devising many plans of future operation, which are no sooner arranged than they are abandoned … a child in his intellectual capacity and manifestations, he has the animal passions of a strong man.

Anatomical Subdivisions of Frontal Cortex



Two Broad Frontal Syndromes

1. Dorsolateral prefrontal syndrome
   - Lowered general arousal
   - Impaired attention/distractable
   - Apathy, depressed mood
   - Perseverative behavior
   - Working memory deficits
   - Diminished spontaneous behavior
   - Difficulty with goal-directed behavior

Two Broad Frontal Syndromes

2. Ventromedial prefrontal syndrome
    - Disinhibition of drives
    - Impulsivity
    - Elevated mood
    - Hyperactive
    - Impaired attention

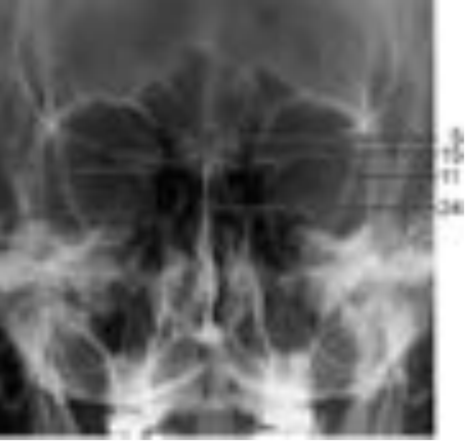
Theories of Prefrontal Function

- There have been many
  - Attentional function, reasoning and planning, inhibition/ selection, "personality", working memory, temporal integration
- None capture the full range of deficits
- But clearly, prefrontal cortex, sitting between sensory and limbic systems on the one hand and motor systems on the other, is in a position to integrate information in the sensory environment, with internal motivations, goals, previous experience, etc.

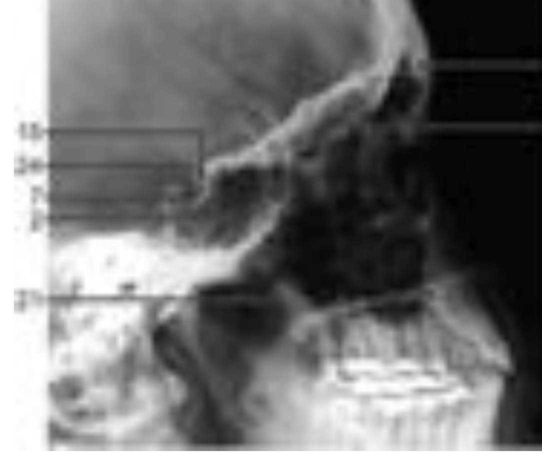
Causes of FL Dysfunction

- changes in motivation
- impaired social skills

- Traumatic Brain Injury (TBI)
- Tumors
- Vascular Lesions
- Neuropsychiatric Diseases
  - Schizophrenia
  - Frontal lobe dementias (Pick’s Disease)

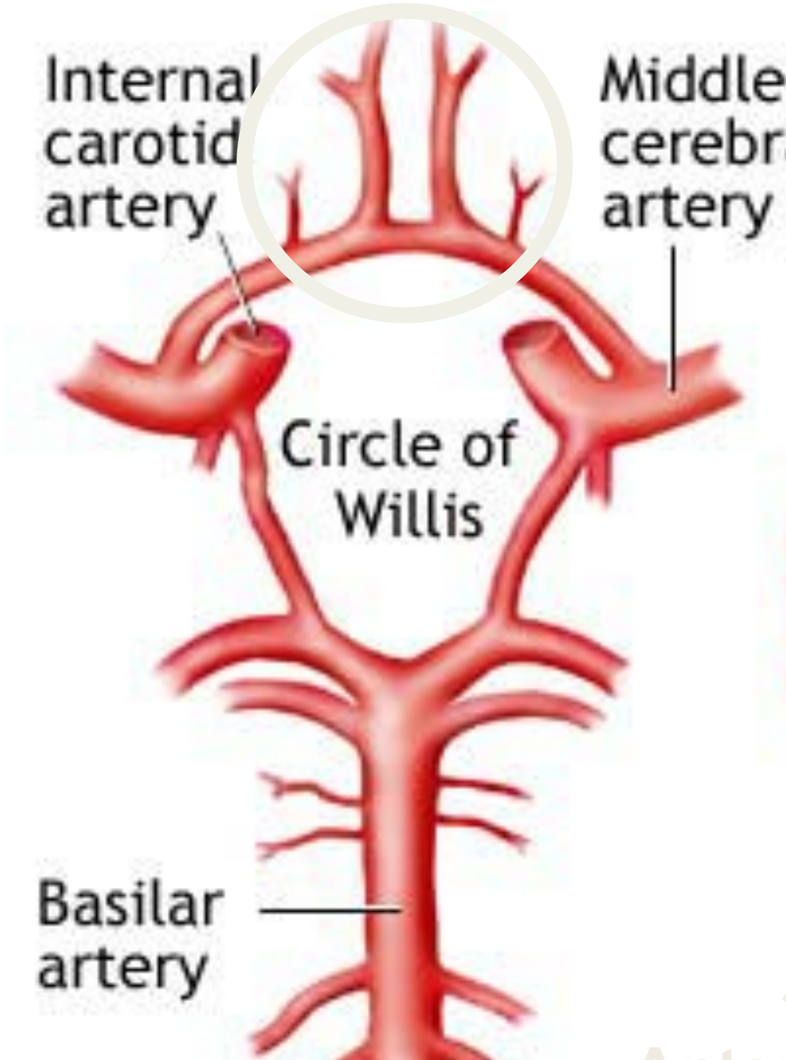


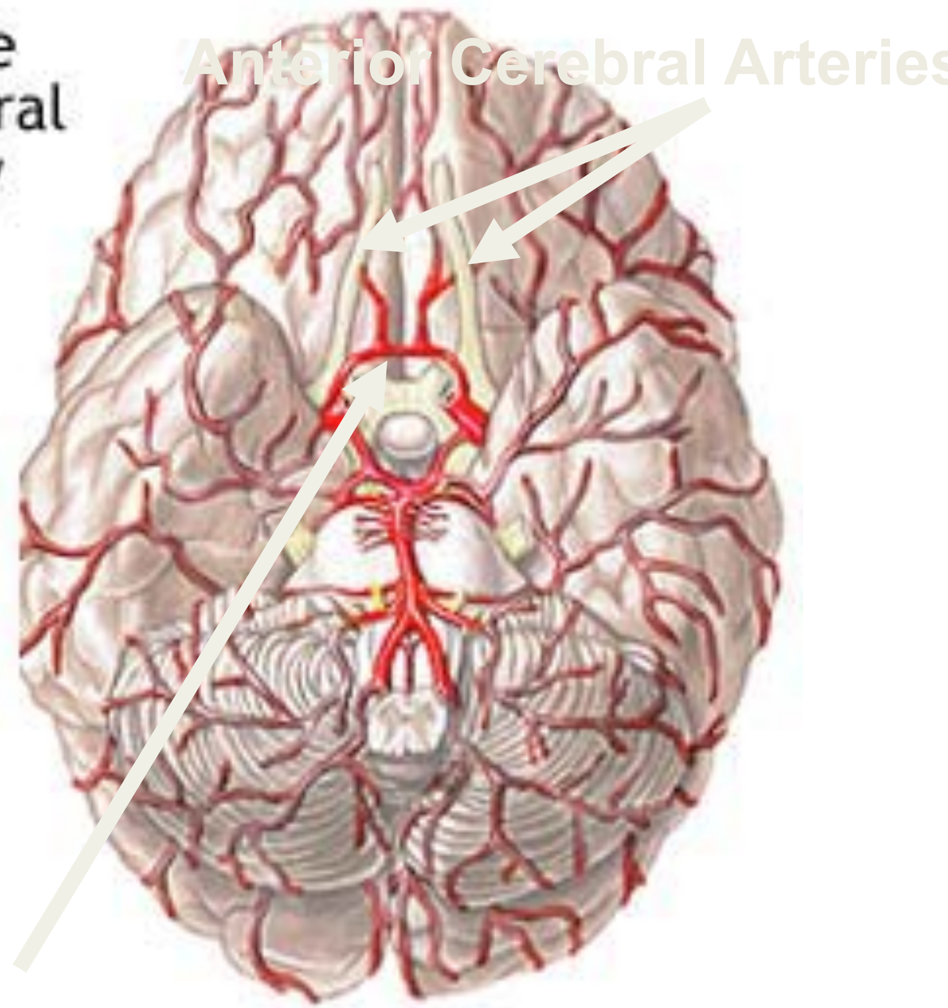
FL Injury in TBI



- The frontal lobes are *extremely vulnerable* to injury due to their location at the front of the cranium, proximity to the *sphenoid wing* and their *large size*.
- MRI studies have shown that the frontal area is the *most common region of injury* following mild to moderate TBI (Levin et al., 1987).
- 2 main types of TB injuries resulting in “FL” dysfunction
  - Injury at the site of impact (“coup”) or where the brain hits the cranium on the opposite side (“contrecoup”).
  - Diffuse axonal shearing (white matter connections w/FL)

Frontal Lobe Dysfunction in CVA





70% of all ischemic strokes occur in the anterior circulation.

Common Symptoms of TBI (Mild to Moderate)

- Behavior problems
  - aggression and violence, impulsivity, disinhibition, acting out, noncompliance, social inappropriateness, emotional outbursts, childish behavior, impaired self-control, impaired self-awareness, inability to take responsibility or accept criticism, inappropriate sexual activity
- Emotional problems
  - depression, apathy, anxiety, irritability, anger, paranoia, confusion, frustration, agitation, and mood swings
- Executive/Cognitive problems
  - such as problems with planning, organizing, abstract reasoning, problem solving, and making judgments,
  - may make it difficult to resume pre-injury work-related activities
- Anosmia (inability to discriminate odors)

Vascular Lesions and Frontal Lobe Dysfunction

- Large vessel strokes (unilateral damage)
  - LH: speech/language (Broca's), right-sided motor deficits, and depression
  - RH: spatial deficits, left-sided motor deficits, elevated mood
- Ruptured aneurysm of the ACoA (anterior communication artery)
  - Personality changes
- Small Vessel/Microvascular Disease
  - Variable presentation of symptoms

Frontal Lobe Dysfunction in Tumors

- *Intrinsic tumors* (e.g., gliomas – tumors arising from glia) are most common and typically begin unilaterally and spread through corpus callosum.
- Can cause gradual or abrupt changes in personality/ mood with or without cognitive changes
- Symptoms determined by tumor features:
  - Size
  - Rate
  - Location

One more time….

- Frontal lobe function
- Damage to Frontal lobe
- How is this related to emotions and high-level cognition?

The sense of time

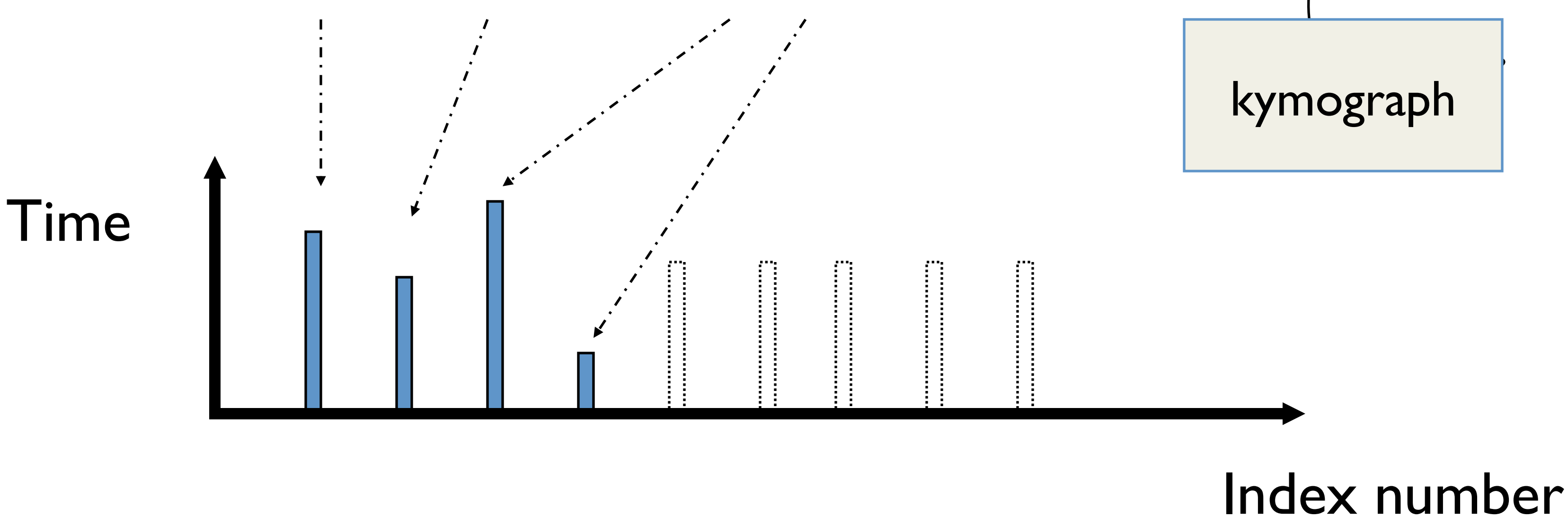
- How does the CNS know time?
- Are there circuits in the brain for the perception of time?
- Neural event counters.
- Long range rhythms vs time in the short range.

Possible mental operations/computations	Example: playing piano	
1. Select	Match fingers to keys, notes.	→ Supplementary motor area
2. Sequence	Group notes into a phrase.	→ Frontal lobe (planning)
3. Force	Strike accented notes with greater force.	→ Basal Ganglia
4. Timing	Establish rhythm.	→ Cerebellum

CORTICAL KEYBOARD METAPHOR

Event times

- Time time series
  - Paced; 250 - 2000 ms
  - 30 - 50 free responses



The Cerebellum and Timing

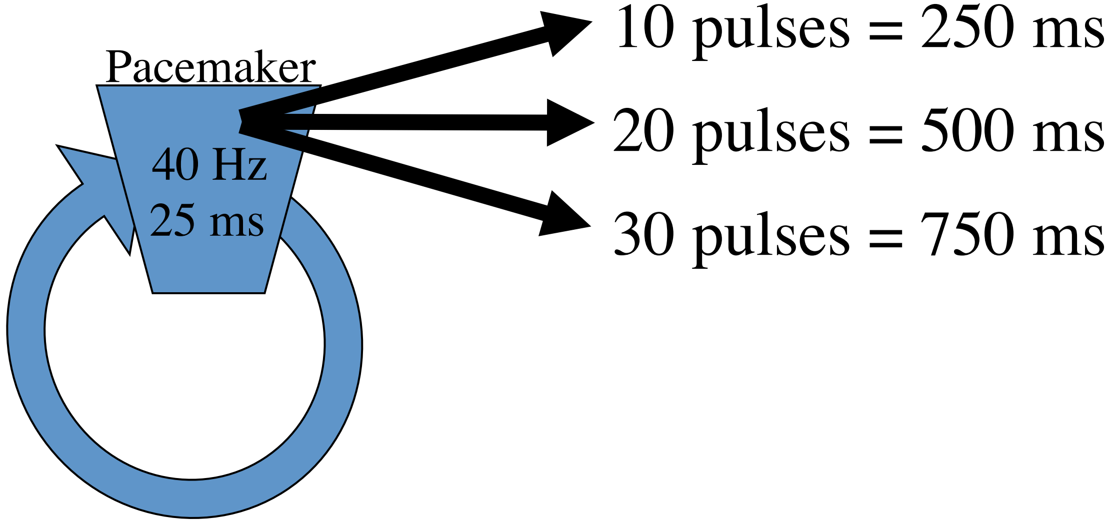
Cerebellum is thought be involved in the timing of movements because
cerebellum lights up in fMRI study of complex/novel timing tasks (Penhune et al. 1998)
cerebellar patients are imparied at tasks like tapping along to a metronome beat

Two basic models:

Clock counter model

Pacemaker produces output to counter

Longer intervals represented by more pacemaker outputs in counter



Interval model

Different intervals represented by distinct elements

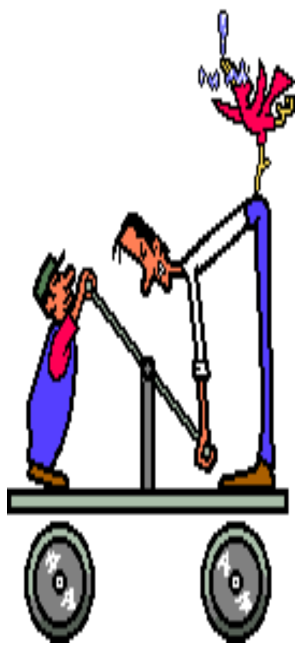
Each corresponds to a specific duration

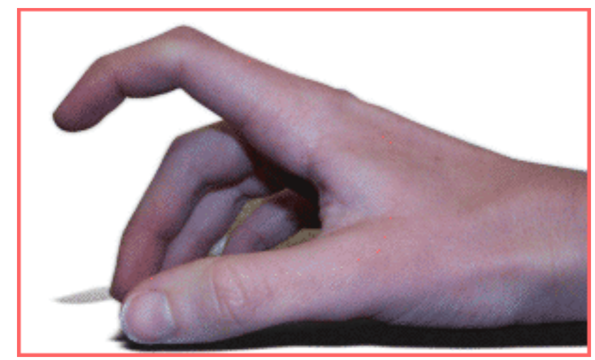
250 ms | 500 ms | 750 ms

Spencer et al (2003): Cerebellum is only responsible for stop-start movements, not continuous motion.

Continuous movements

Discontinuous movements





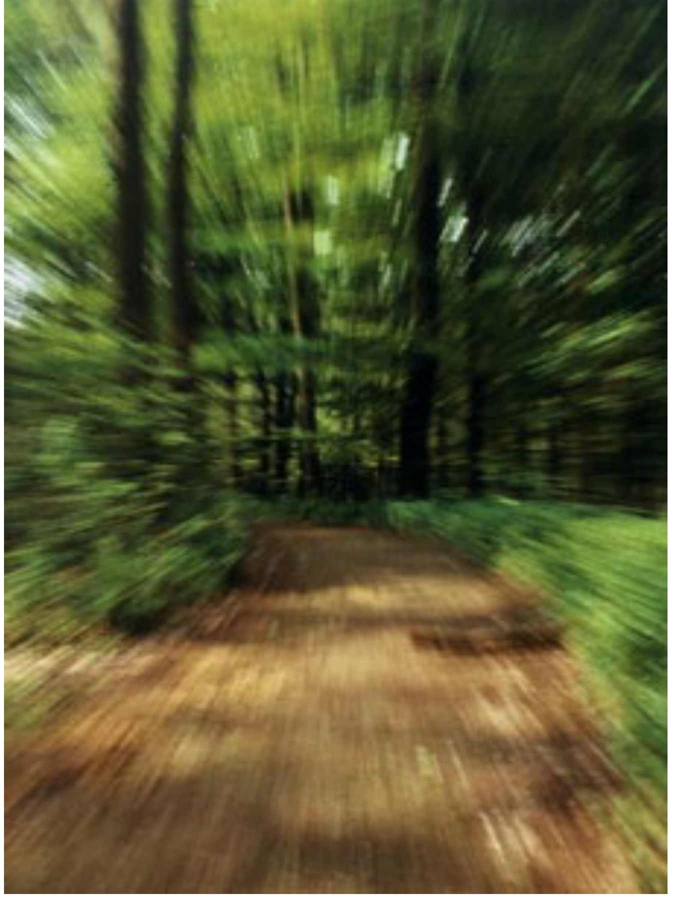
have a specific temporal goal

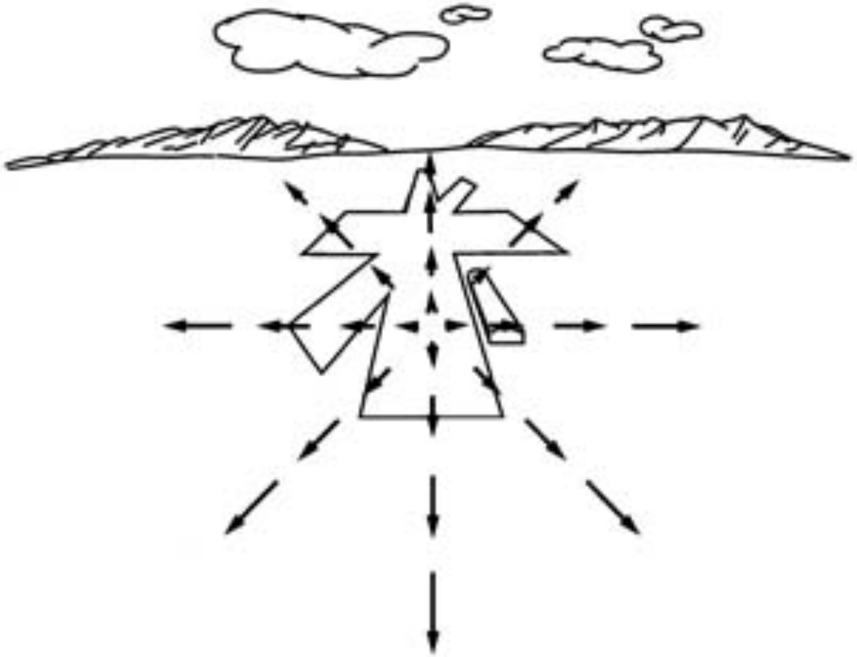
can be set going and left

This is what is controlled by the cerebellum.

Time to contact

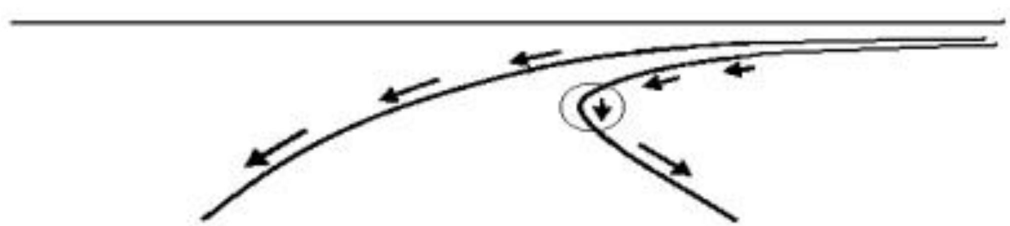
- Is this the same mechanism for timing that is used in locomotion & navigation?

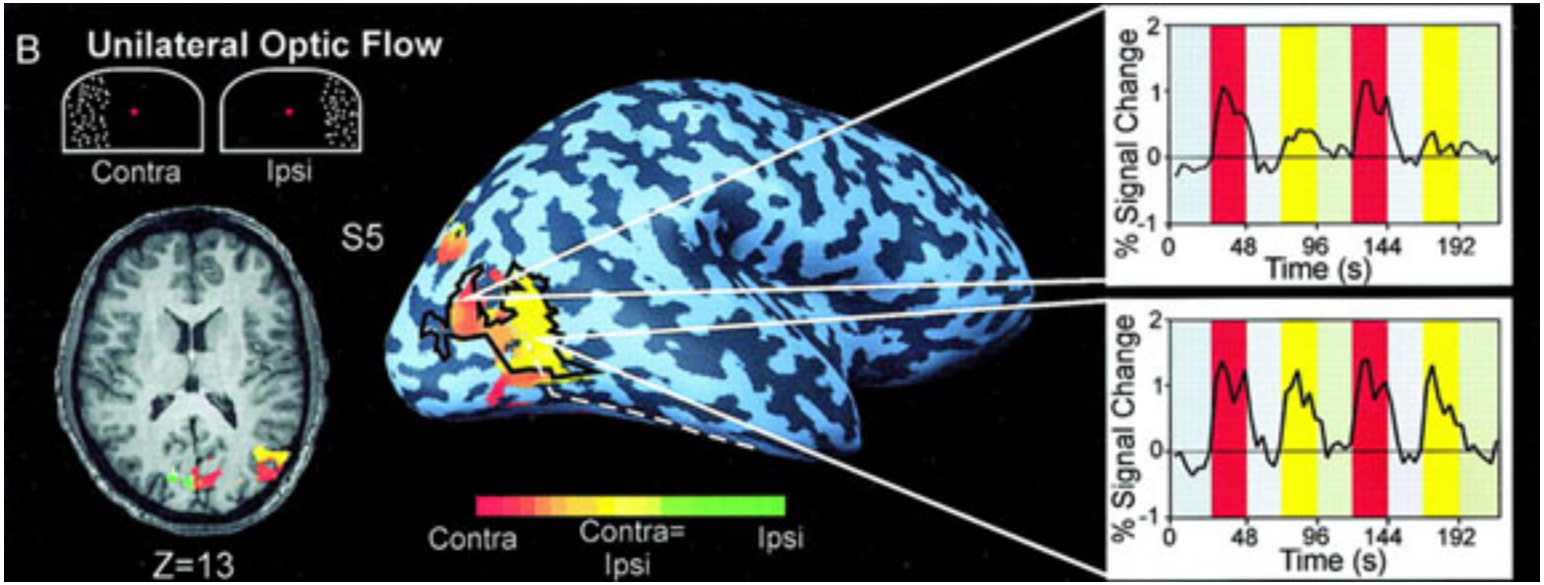




FOCUS OF EXPANSION

LINES OF FLOW





-Visual cortical motion processing area encodes basic elements of motion and might be involved in perception of object and self-motion (i.e. optic flow), larger receptive fields

Sense of time now comes from optics (not internally generated by subcortical circuits).

Biological Rhythms

- Many of our behaviors display rhythmic variation
  - Circadian rhythms ("about a day")
    - One cycle lasts about 24 hours (e.g. sleep-waking cycle)
    - Light is an external cue that can set the circadian rhythm
    - Some circadian rhythms are endogenous (do not require light) suggesting the existence of an internal (biological) clock
    - Entrainment
  - Monthly rhythms
    - Menstrual cycle
  - Seasonal rhythms
    - Aggression, sexual activity in male deer
    - Seasonal Affective disorder

Biological clocks:

- systems that control the temporal organization of biological processes

- inferred existence due to cyclical pattern of various functions ie blood level of glucocorticoids, body temperature, sleep.

- cycle of a clock is defined by its **period:** interval of time required to return to its initial state

- different types of cycles that vary as a function of period:

    - **circannual -** about 1 year ie SAD
    - **one month -** ie human menstrual cycle
    - **ultradian -** longer than 24 hours
    - **circadian rhythm -** about 24 hours.
        - sleep-wake cycle
    - **infradian -** less than 24 hours.
    - **Short term clocks**: cerebellum.

Sleep Disorders

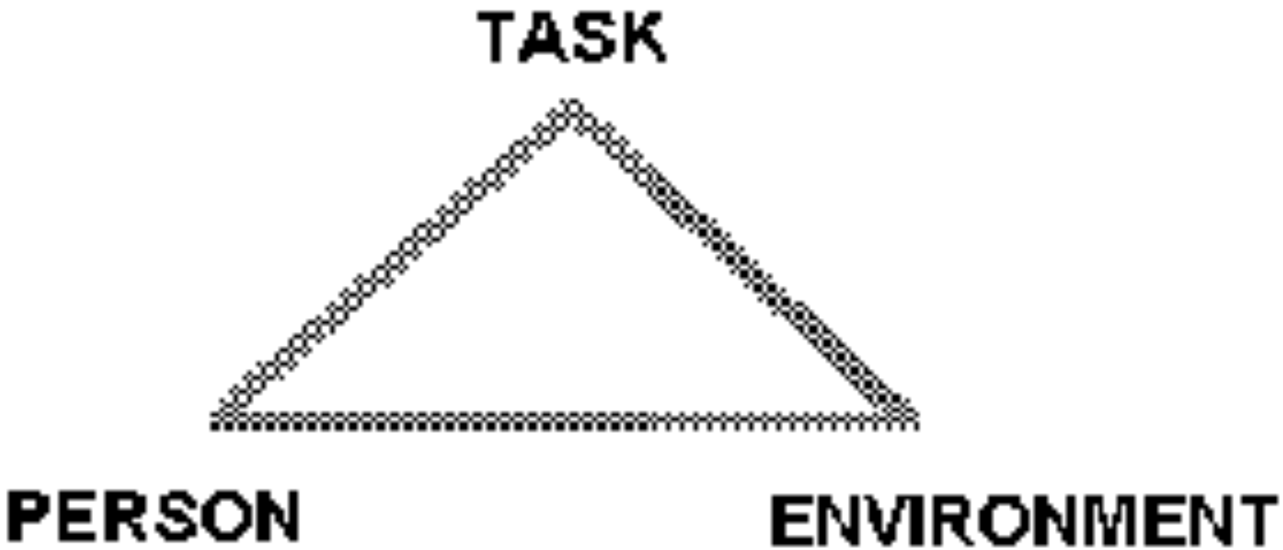
- Insomnia refers to a difficulty in getting to sleep or remaining asleep and has many causes
  - Situational
  - Drug-induced: Use of sleeping pills can result in insomnia
  - Sleep apnea: person stops breathing and is awakened when blood levels of carbon dioxide stimulate breathing
- Narcolepsy: Sleep appears at odd times
  - Sleep attack: urge to sleep during the day
  - Cataplexy: REM paralysis occurs, person is still conscious
    - Sleep paralysis: REM paralysis that occurs just before or just after sleep
  - Narcoleptics have reduced levels of the neuropeptide orexin or altered activity of the orexin-B receptor

What is

MOTOR LEARNING?

What is learned in motor learning?

- Adams: closed-loop theory
- Schmidt: motor schema theory
- Generalized motor programs
- How to work with patients who have lost movement function?



NEWELL's SCHEME FOR MOTOR LEARNING

The "internal reference of correctness"

- J.A. Adams (1971) used this term in his influential theory of motor learning. He proposed that several processes become increasingly congruent for motor learning to occur:

- A motor memory must initiate movement

- The movement thus initiated must produce internal feedback, which "lays down" in the central nervous system another memory, a "perceptual trace." The more accurate the movement, the more useful the perceptual trace that is collected and retained.

Closed-loop theory

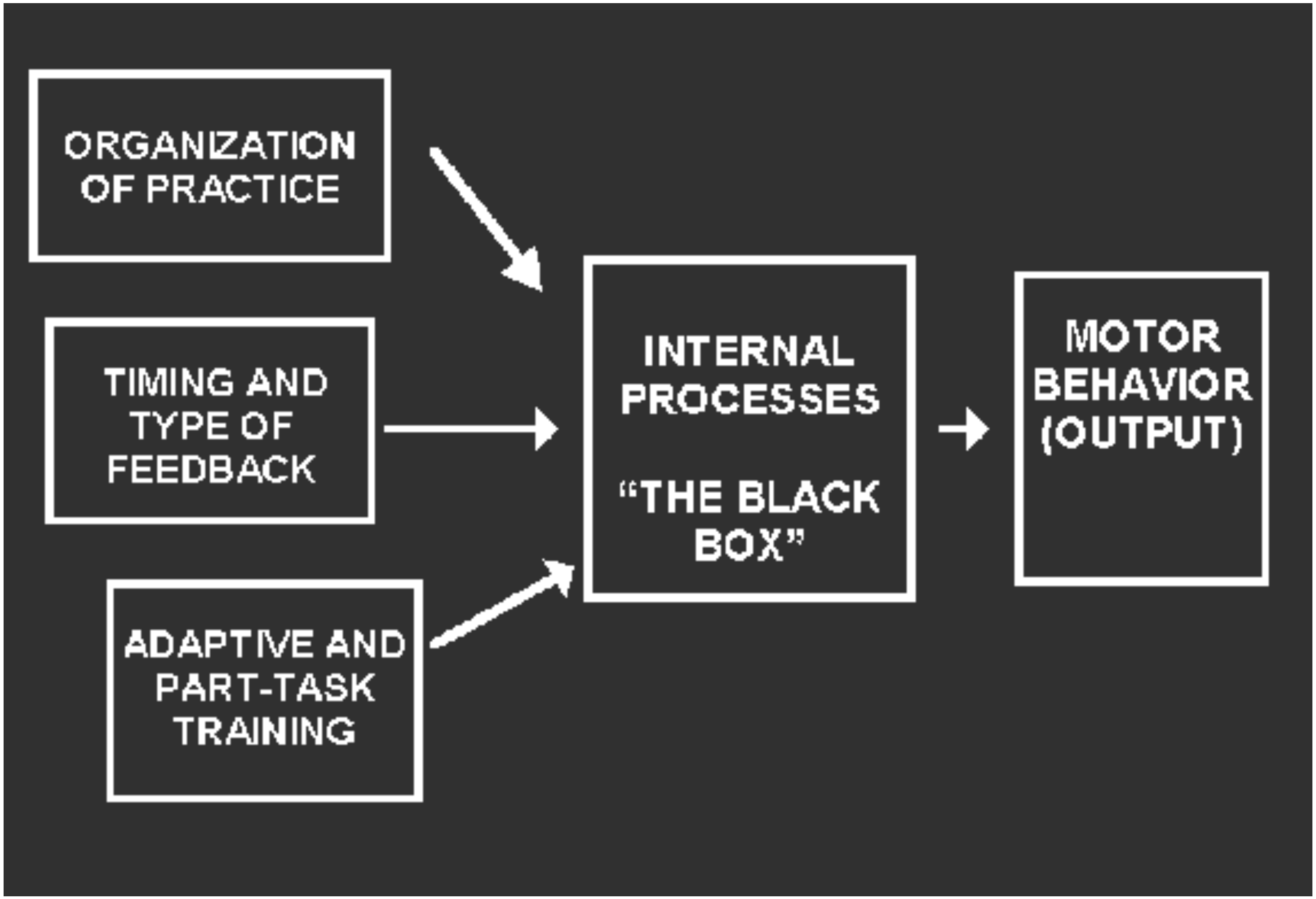
- The system must compare the feedback produced by a current movement against the accumulated perceptual trace.
- Finally, the system must detect any "error" or difference between the actual and the expected feedback, and correct the movement accordingly.
- Adams' theory implies a "closed-loop" type of learning in which accuracy and repetition are important for refinement of skill.

Schema theory

- **Richard Schmidt developed a schema theory of motor learning to explain evidence that variety of practice and latitude for errors also produces motor learning.**
- **People don't learn specific movements. Instead, they construct "generalized motor programs." They do this by exploring programming rules, learning the ways in which certain classes of movement are related. Then they learn how to produce different movements within a class by varying the parameters that determine the way in which movements are constructed.**

Recall vs recognition

- In Schmidt's theory, this relationship between the parameters and outcomes are collected in two "schemes" or "schema," hence the name by which his theory is known.
- Recall schema: relates outcomes to parameters like those listed above, movement duration, overall force production, etc.
- Recognition schema : relates expected sensory consequences of a movement to the movement's outcome. This is reminiscent of Adams' earlier ideas of an "internal reference of correctness"


ORGANIZATION OF PRACTICE
TIMING AND TYPE OF FEEDBACK
ADAPTIVE AND PART-TASK TRAINING
INTERNAL PROCESSES
"THE BLACK BOX"
MOTOR BEHAVIOR (OUTPUT)

Rehab programs

- Rehab Programs are similar to schooling
- Rehabilitation is done ***with*** the patient rather than ***to*** the patient
- Learning new movements from generalized motor programs and novel dynamics.
- More details in APA 4150: Motor Rehabilitation.

CONSTRAINT INDUCED THERAPY

One more time….

- With lots of feeling.
- Course overview:
- Neural control of movement: neurophysiology, pathologies & disorders, measurement techniques
- Plasticity and Learning: the dynamic brain
- Neuro-rehabilitation

Future Directions

- Advances in brain imaging
- Matching spatial and temporal resolution
- Real-time study of the brain
- Implanting electrodes into the brain
- Neuro-rehab engineering, artificial brains?

One more time ….

"Whoa! *That* was a good one! Try it, Hobbs—just poke his brain right where my finger is!"

THE END

Advanced course suggestions

KIN 4P03

KIN 4CN3