COGS101

Causes of agnosia:
- brain damage
- dementia
- drug use
- developmental: typically prosopagnosia

Amnesia:

- amnesia means loss/failure to form a memory
- retrograde amnesia - loss of memory of times passed - typical Hollywood amnesia, not very common, but can happen w/ severe psychological trauma
- most common - anterograde amnesia: failure to lay down new memories

What is amnesia?:
- loss of memory
- 2 different kinds:
  - *retrograde*: loss of existing amnesia; forgetting things from earlier parts of life
  - *anterograde:* failure to form new memories; forgetting what has just happened

Causes of amnesia:
- Alzheimer's disease
- stroke
- head injury
- Epilepsy
- can affect different sides of the brain and so different areas can be affected - e.g. left hemisphere - language & verbal knowledge

Retrieval failure amnesia:
  - enduring memories of experiences are created; access to them impaired
    - tests requiring recall failed
    - tests requiring recognition may reveal memory - e.g. hints used
- frontal lobe dysfunction may be responsible
- not primary problem w/ memory
  - often other deficits: attention, planning, general info retrieval not associated with self experience
- traumatic brain injury: frontal lobes, diffuse axonal damage
- depression: neurochemical imbalance in fronto-striatal circuitry
- stroke: anterior circulation - frontal lobes; thalamus (major info relay nucleus)
- Wernicke-Korsakoff syndrome: vitamin B1 (thiamine) deficiency; can result from long term alcoholism
- anterograde amnesia: new info can be learned but not recalled
- retrograde amnesia: existing memories cannot be retrieved
- unitary account - retrieval failure blurs anterograde/retrograde distinction

Apperceptive agnosia – A form of visual agnosia in which a person cannot reliably name, match, or discriminate visually presented objects, despite adequate elementary visual function (visual fields, acuity, and color vision).
Associative agnosia – A form of visual agnosia in which a person cannot use the derived perceptual representation to access stored knowledge of the object's functions and associations but is able to copy and match the drawing even though unable to identify it.
Balint's syndrome – Agnosic syndrome that results from large bilateral parietal lesions and is composed of three deficits: (1) paralysis of eye fixation with inability to look voluntarily into the peripheral visual field, (2) optic ataxia, and (3) disturbance of visual attention such that there is neglect of the peripheral field.
Dorsal simultanagnosia – An inability to detect more than one object at a time, with difficulty shifting attention from one object to another.
Dorsal stream – The stream of cortical visual projections from primary visual cortex to posterior parietal cortex, concerned primarily with the visual control of action.
Inferotemporal cortex – Inferior surface of the temporal lobe that is particularly important for object recognition.
Integrative agnosia – A form of visual agnosia in which one retains the ability to recognize elements of objects but is unable to integrate these elements together into comprehensible percept.
Optic aphasia – A condition in which a person cannot name a visually presented object, despite being able to indicate the identity of the object through gesture and sort the visual stimuli into categories.
Prosopagnosia – A form of visual agnosia in which a person cannot recognize familiar faces, despite adequate elementary visual function (visual fields, acuity, and color vision).
Ventral simultanagnosia – A reduction in the ability to rapidly recognize multiple visual stimuli, such that recognition proceeds in a part-by-part fashion.
Ventral stream – The stream of cortical visual projections from primary visual cortex to the inferotemporal cortex, concerned primarily with representing the identity of stimuli by characteristics such as shape and color.

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Consolidation failure amnesia:
- enduring memories of current experiences not created
    - tests requiring recall failed
    - tests requiring recognition also failed - e.g hints used
- memory not formed in first place
- primarily memory problem:
    - temporal lobe
    - or hippocampal amnesia
    - these receive multimodal perceptual info & are linked to knowledge stores distributed across cortex
    - hippocampus= associates contiguous (co-occurring) inputs forming memory links (what/ when/where aspects of experience)
- stroke: posterior circulation - hippocampus
- transient global amnesia - rare, usually only happens 1x in life- sudden onset, temporary (up to few hrs); suspected to do w/ blood supply
- temporal lobe epilepsy: seizures —> hippocampal sclerosis
- transient epileptic amnesia: similar to transient global amnesia: lasts up to few hrs but = accelerated long-term forgetting; possible patchy retrograde amnesia
- herpes simplex encephalitis: damages hippocampus
- Alzheimer's disease:
    - most common cause of amnesia
    - neurodegeneration starts in temporal lobes —> spreads to rest of brain
    - anterograde amnesia usually 1st sign
        - unremindable: memory not formed
        - contrasts w/ retrieval-based amnesia of late-onset depression (other v likely cause)
    - perhaps graded retrograde amnesia?
        - hard to tell w/ insidious onset of disease

- anterograde: new links not formed
- retrograde: recently formed, partially consolidated memories affected
- dualistic account: specific mechanisms to explain antero & retro

Theoretical accounts of amnesia:
- Standard Consolidation Theory (SCT):
    - hippocampus involved in memory formation & integration of new memory traces w/ existing traces (located in cortical regions)
    - explains anterograde amnesia
    - consolidation occurs over long period of time w/ hippocampus 'handing over'
    - memories become independent of hippocampus
    - explains temporally graded retrograde amnesia (Ribot's Law) w/ hippocampal damage
    - Ribot's law: more recent memories affected more than older memories (which are independent of hippocampus)
    - criticisms: trouble explaining non-graded retrograde amnesia
        - JB: patchy retrograde loss
        - old memories can affected just as badly as recent memories

- Multiple Trace Theory (MTT):
    - hippocampus involved in memory formation & integration of new memory traces w/ existing traces in cortical regions
    - explains anterograde amnesia
    - consolidation occurs over long period of time w/ hippocampus creating new, schematised trace each time memory is recollected
    - however, memory does not become independent to hippocampus, but keeps a clearer 'master copy' of memory
    - can explain temporally graded retrograde amnesia w/ limited hippocampal damage
        - recent memories = fewer traces