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Neuropsychological Aspects of Aging: Implications for Assessment & Intervention

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Disciplines

Psychology

Comments

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Neuropsychological Aspects of Aging: Implications for Assessment & Intervention

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Disclosures

- Employee of Dordt College

Overview

- Identify primary brain regions & describe their central functions
- Describe some of the “normal” neuropsychological changes that occur with advancing age
- Develop a broader understanding of most common aging-related Neurocognitive Disorders & their characteristic signs / symptoms
- Identify at least one key aspect of each primary type of Neurocognitive Disorder through basic neurocognitive screening
- Identify key treatment-, referral-, & continuum-of-care considerations / options for those struggling with neurocognitive impairment

Note:

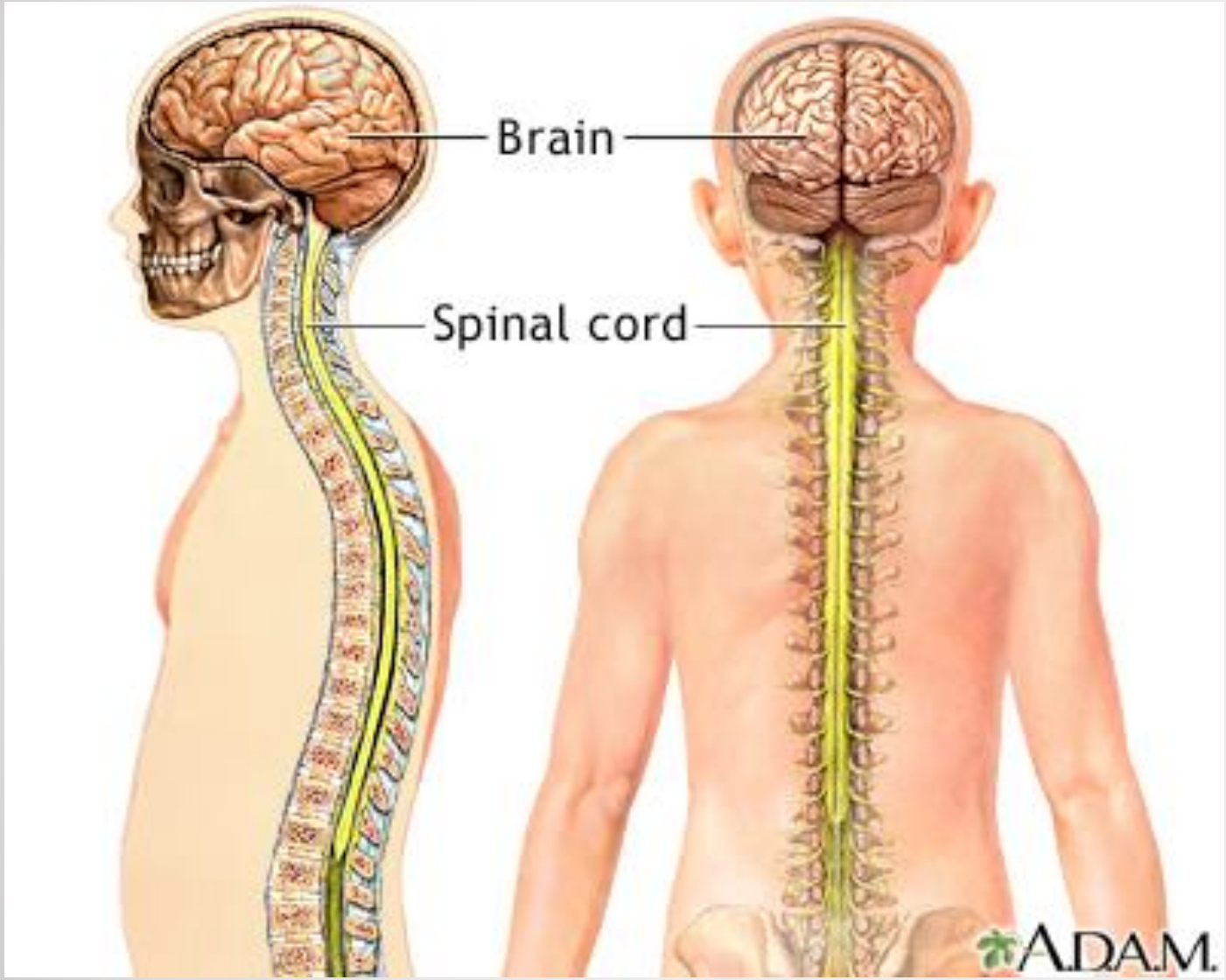
Although completion of this workshop will NOT make you a qualified neuropsychologist, it WILL give you clinically useful information and tools for appropriate *neurocognitive screening* of older adults within the scope of your current practice.

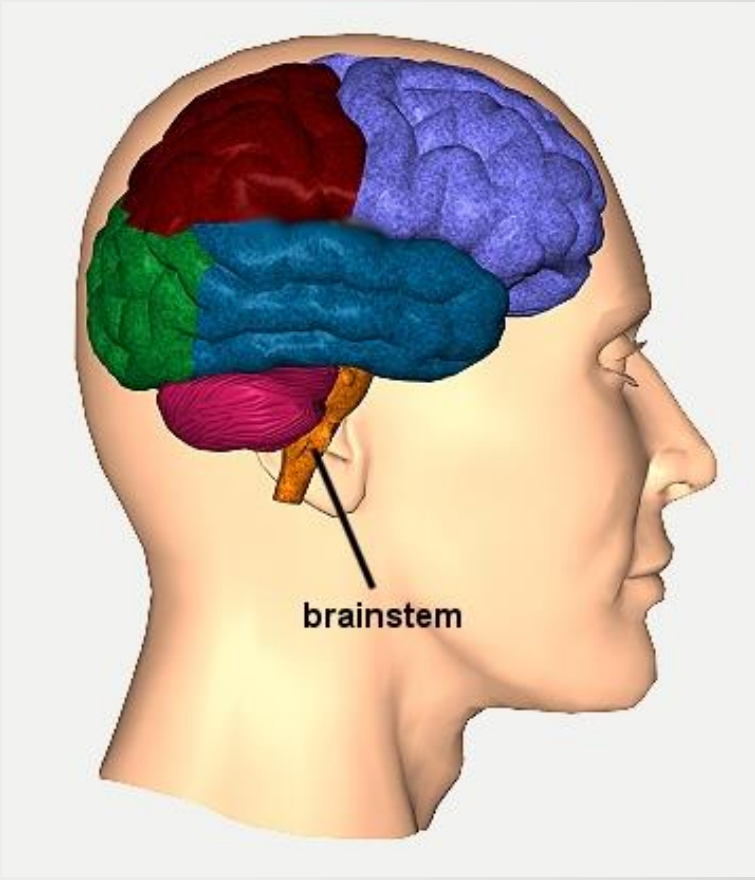
Older Adults in South Dakota

- In 2017, approx. 20% of South Dakota residents were ≥ 60 years old (≈ 174,000)

- Future Projections (U.S. Census Bureau 2009 estimates):
  - 2020 ≈ 22.5% of pop.
  - 2030 ≈ 27.5% of pop.

The Central Nervous System





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Neurons

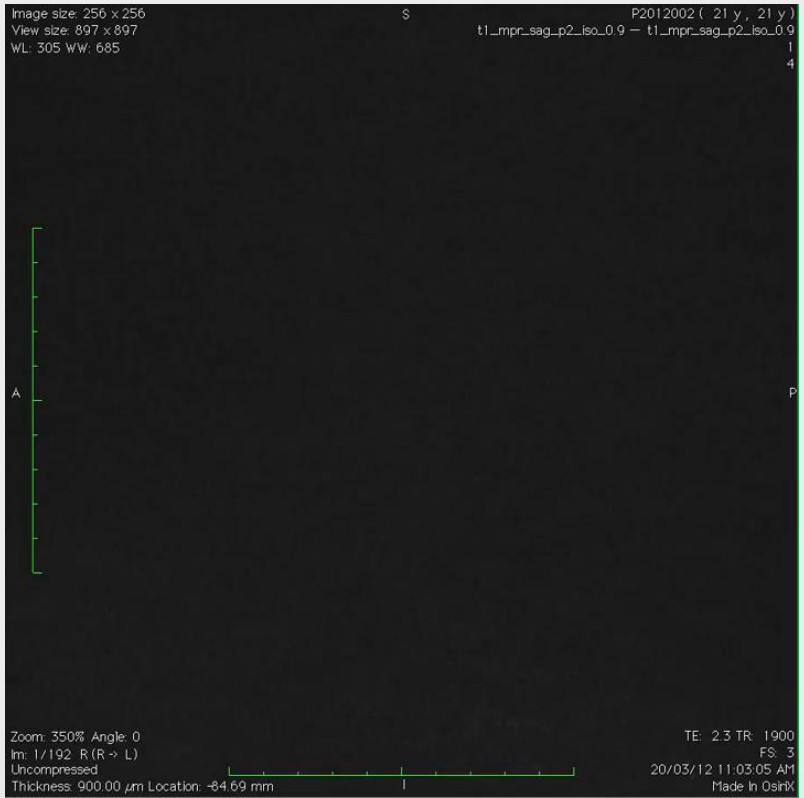
- 50-100 billion throughout CNS (approx. 20 billion in neocortex alone)
- Most all present ***at birth***
- Can move & grow
- No replication of cells
- Each can receive 100,000 + contacts
- Starting in our 20's, we naturally lose ≈ 10,000-100,000/day

Image: https://alsnewstoday.com/2017/07/21/als-researchers-find-natural-mechanism-to-prevent-harmful-tdp-43-protein-clumping/

Neurons - 2

- Neural Impulse (Action Potential) = *Electrochemical* Process
- Action potential expends energy & electromagnetic "fields" (glucose, oxygen, blood flow)

  ➔Basis of neuroimaging technology (CT, MRI, PET, etc.)



Glial Cells

- Likely >100 billion in neocortex alone
- Do *not* transmit information…
- …Instead, implicated in synaptic functioning & neural signaling
- Provide

  --Structural support

  --Nutritional & scavenger functions

  --Release of growth factors

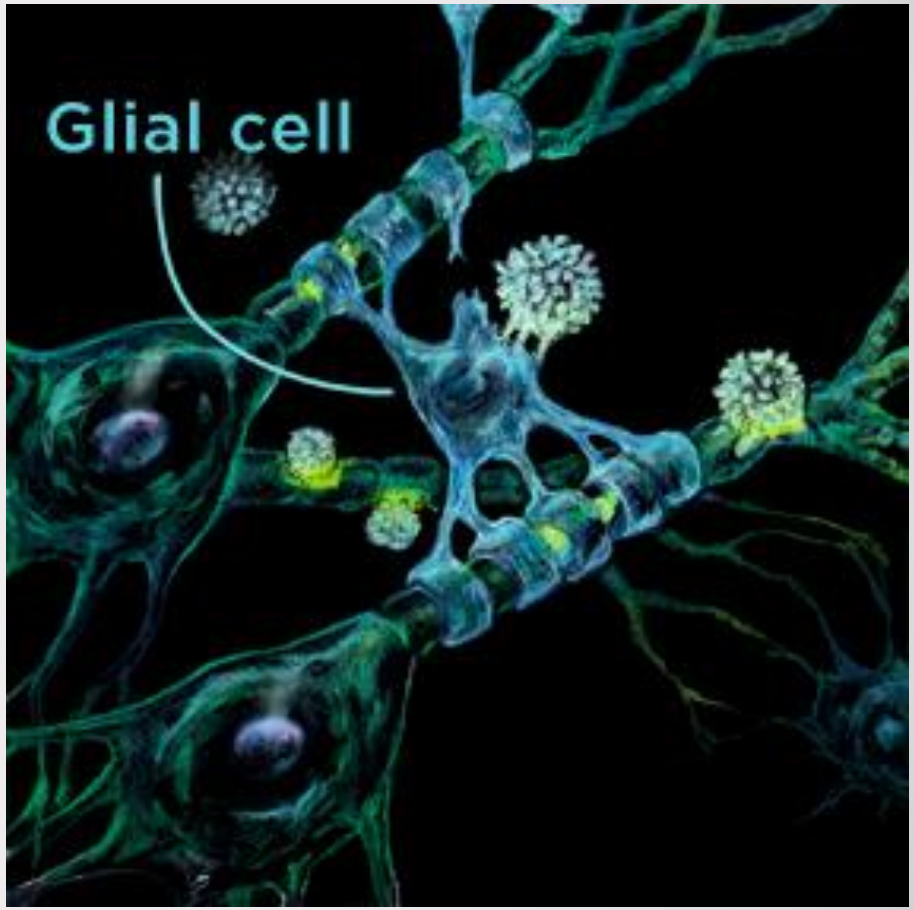


Image: https://jeevanshu.wordpress.com/2015/05/29/do-glial-cells-have-any-role-in-creativity-and-genius-nearly-90-percent-of-the-brain-is-composed-of-glial-cells-not-neurons/

"Normal" Brain Aging

- Aging-related decline begins in one's 20's-30's
- Cortical atrophy evident by 40's
- Decreased Gray Matter Volume:
  - Reduced dendrite length / arborization
  - Fewer neocortical synapses
- Decreased White Matter Volume
  - ≡ Most signif. overall brain shrinkage
- *Some* senile plaques & neurofibrillary tangles

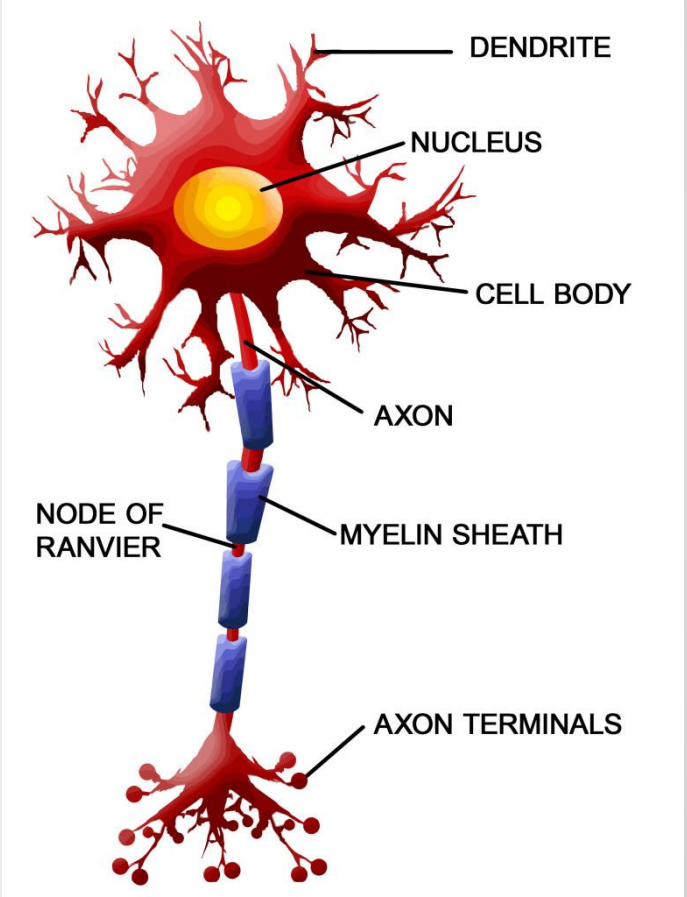


Image: http://www.interactive-biology.com/3247/the-neuron-external-structure-and-classification/

Lobes of the Brain

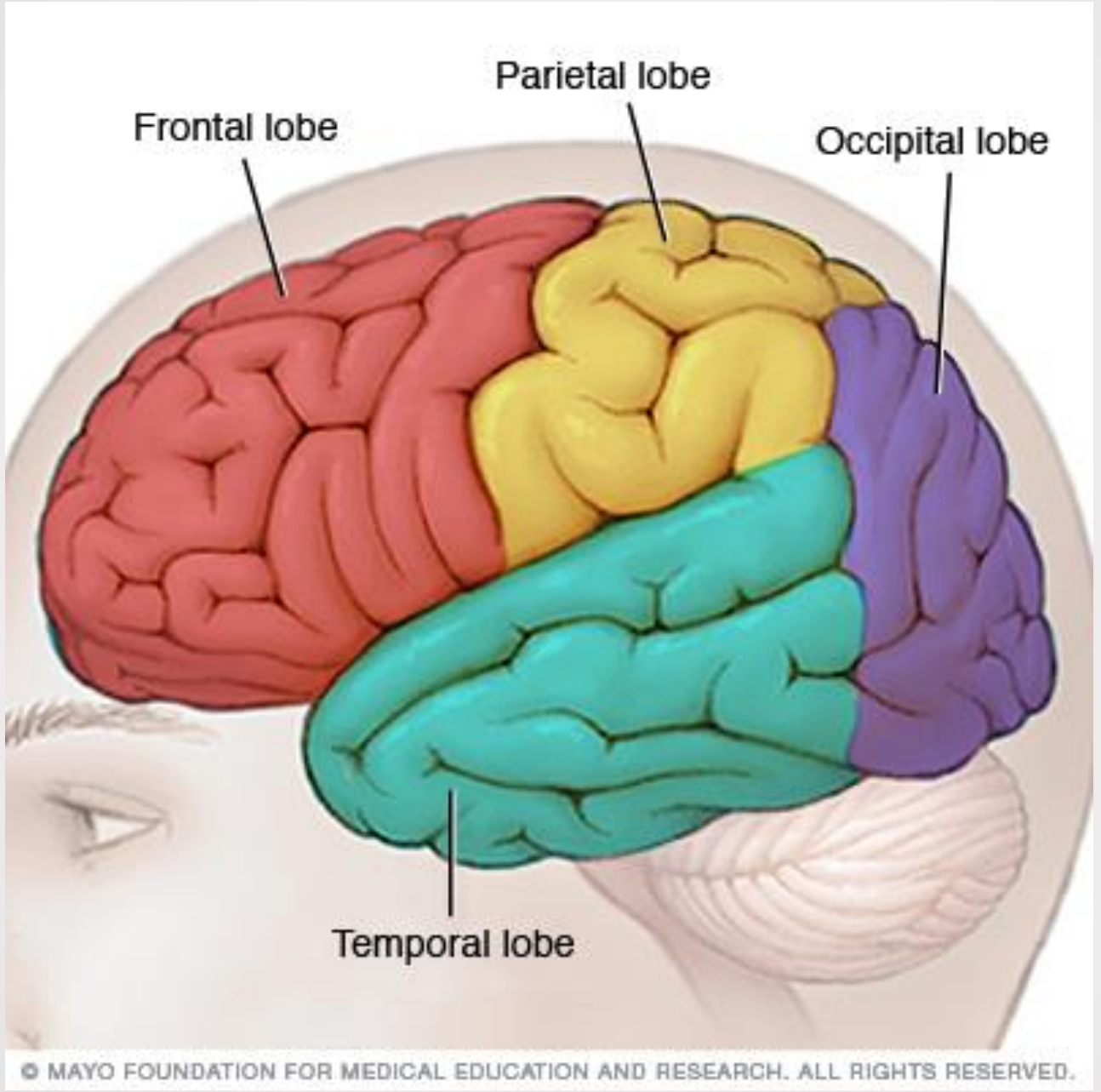


Image: http://www.mayoclinic.org/brain-lobes/img-20008887

General Lobe Functions

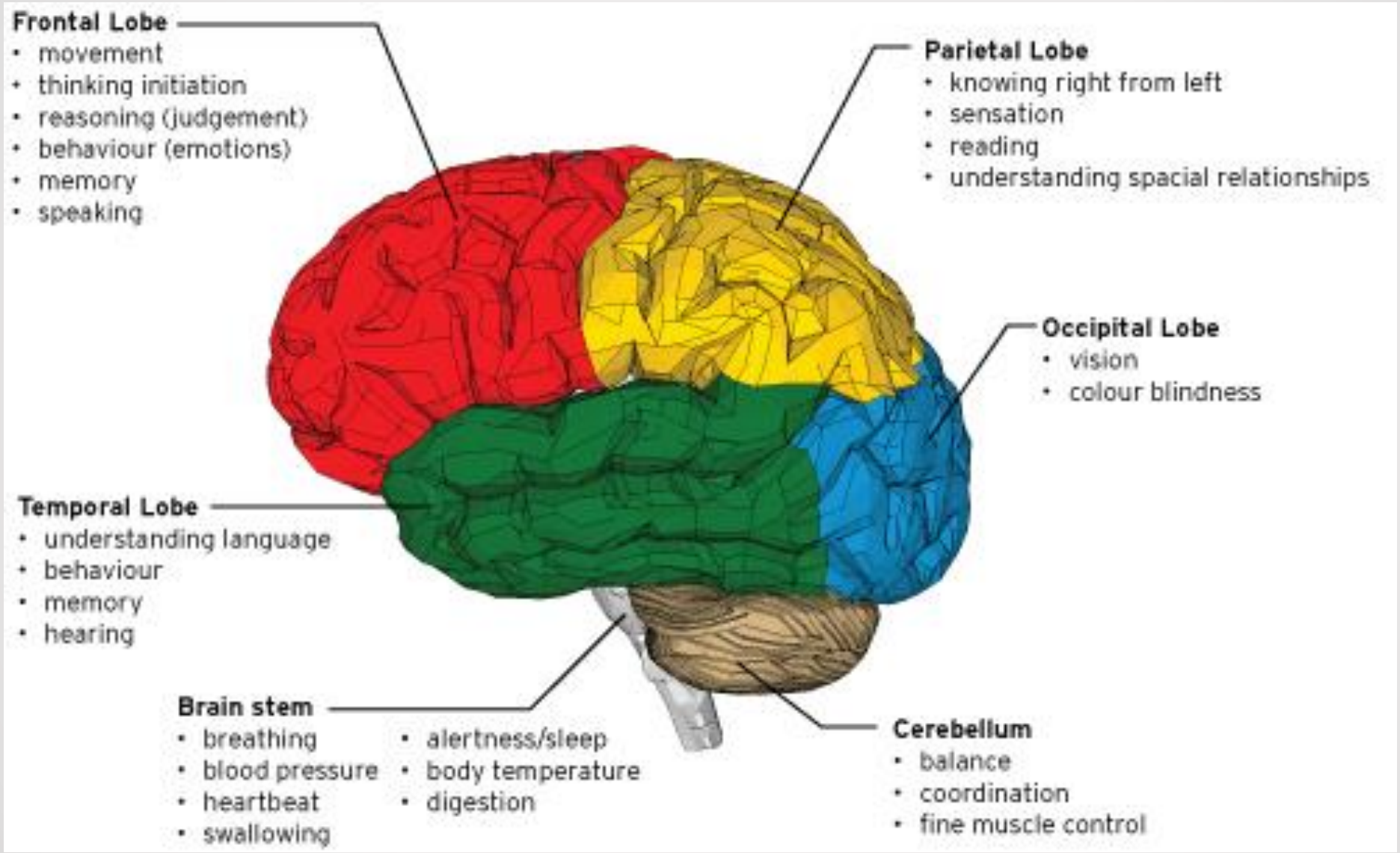


Image: http://www.familyhealthonline.ca/fho/familymedicine/FM_stroke_FHc13.asp

Neurocognitive Domains (DSM-5)

American Psychiatric Association (2013)

A. Learning & Memory
B. Complex Attention
C. Executive Function
D. Language
E. Perceptual-Motor
F. Social Cognition

Aging & Neurocognitive Domains: General Principles

Lezak, Howieson, Bigler, & Tranel (2012)

- Slowed *information processing speed* implicated in many "normal" cognitive changes
- Education level, etc. influence "brain reserve capacity" & cognitive preservation into later years ➔ Cognitive Reserve
- Number of potentially confounding research variables increase with advancing subject age…

  …So, following data must be discerned carefully (is not exhaustive)

A. Learning & Memory

- Long-Term
  - Autobiographical
  - Semantic (Context-free; general knowledge of symbols & concepts + the rules for manipulating them)

- Short-Term
  - Verbal—Nonverbal
  - Immediate – Delayed
  - Recall – Recognition

A. Learning & Memory - 2

- "Normal" Aging Effects:
  - Mild word-finding difficulty (esp. proper names)
  - Immediate short-term mem. affected slightly
  - Acquisition < retention
  - Short-term *non*verbal mem. typically *more compromised* than short-term verbal mem.
  - Recognition mem. retained well
  - Implicit & procedural mem. fairly robust

B. Complex Attention

- Sustained
- Divided
- Selective
- Processing Speed

- "Normal" Aging Effects:
  - Simple span intact into 80's
  - Slower responses & more errors on tasks of divided attn.
  - Difficulty shifting attn. when given an invalid cue
  - Deficits in sustained & selective attn. + distractibility

C. Executive Function

- Abstraction / Reasoning
- Decision-Making
- Mental Flexibility / Planning
- Working Memory

- "Normal" Aging Effects:
  - Reasoning w/ familiar material good (yet more concrete)…but compromised w/ unfamiliar, complex material
  - Abstraction declines (concept formation, too—but in 80's)
  - Working mem. declines

D. Language

- Expressive:
  - Naming
  - Fluency
  - Word-finding
  - Grammar
  - Syntax

- Receptive:
  - Comprehension

D. Language - 2

- “Normal” Aging Effects:
  - Verbal abilities retained well, generally
  - Verbal fluency changes (variable research results)
  - Verbal comprehension changes (variable research results)

E. Perceptual-Motor

- Visual Perception
- Visuoconstruction
- Praxis

- "Normal" Aging Effects:
  - Object- & shape-recognition preserved well
  - Visuo-perceptual judgment declines gradually / steadily into 90's (basic analysis OK, but integration / reasoning decline)
  - Diminished accuracy & complexity on some construction tasks

F. Social Cognition

- Emotional Recognition
- Social & Behavioral Propriety

- "Normal" Aging Effects:
  - Generally well-preserved
  - Declining perceptual abilities may negatively influence emotional recognition

Cognitive Aging & Intellectual Ability

Lezak et al. (2012)

- Crystallized Intelligence:
  - Over-learned, well-practiced, familiar…

    …Skills, ability, & knowledge
  - *Gains* through 60's, stable through 70's

- Fluid Intelligence:
  - Reasoning & problem-solving for which familiar solutions are not available
  - Slow *decline* until late 50's – early 60's, then pace of decline increases

Neurocognitive Screening: Characteristics

Roebuck-Spencer, Glen, Puente, Denney, Ruff, Hostetter, & Bianchini (2017)

- Narrow in scope
- Minimal administrator training needed
- Brief administration (< 30 min.)
- Provides
  - Early identification of those at risk of decline
  - Indication of need for referral for additional evaluation / treatment
  - Means of monitoring symptom progression or treatment response
- Does NOT provide definitive diagnosis

Neurocognitive Screening: Procedures

- Obtain / Review Medical Documentation from PCP, incl.
  - Recent, relevant primary care medical notes
  - Neuroimaging reports (if avail.)
  - Specialist reports (neurology, psychiatry, etc.)

- Thorough, Comprehensive Evaluative Interview
  - Interview family / others when possible, also
  - The best available strategy you have (likely)!

Neurocognitive Screening: Procedures - 2

- Select Screening Instrument(s), *e.g.*
  - Montreal Cognitive Assessment Test (MoCA) (www.mocatest.org)
  - Mini-Mental Status Examination (MMSE)
  - Geriatric Depression Scale – Long Form (GDS)
  - Geriatric Anxiety Scale (GAS) (www.uccs.edu/agingandmentalhealthlab/scale)

Neurocognitive Screening: Procedures - 3

- Recommend Referral(s) p.r.n.
  - Neuropsychology
  - Radiology (neuroimaging)
  - Neurology
  - Psychiatry

- Treatment p.r.n.
  - Client
  - Spouse
  - Family

Abnormal Cognitive Decline in Older Adults: The "3 D's"

- Differential Diagnosis / Rule-Out's
  - Delirium
  - Depression
  - Dementia (Major Neurocognitive Disorder)

Delirium: DSM-5 Criteria

A. A disturbance in attention (i.e., reduced ability to direct, focus, sustain, and shift attention) and awareness (reduced orientation to the environment).
B. The disturbance develops over a short period of time (usually hours to a few days), represents a change from baseline attention and awareness, and tends to fluctuate in severity during the course of a day.
C. An additional disturbance in cognition (e.g., memory deficit, disorientation, language, visuo-spatial ability, or perception).
D. The disturbances in Criteria A and C are not better explained by another preexisting, established, or evolving neurocognitive disorder and do not occur in the context of a severely reduced level of arousal, such as coma.
E. There is evidence from the history, physical examination, or laboratory findings that the disturbance is a direct physiological consequence of another medical condition, substance intoxication or withdrawal, or exposure to a toxin, or is due to multiple etiologies.

Delirium: Key Characteristics

- * *Acute* onset (typically)
- * Clouding or loss of consciousness (usu. unexplained)
- Impaired cognition (incl. memory & language)
- Confused, "out of touch", disoriented
- Hallucinations (poss.)
- * Course: Hours ➔ days (some forms = weeks ➔ months)
  - Waxing/waning fairly common
- Typically caused by a medical condition…

Etiology of Delirium

- Encephalopathy due to…
  - Urinary tract infection (esp. older adults)
  - Dehydration
  - Medication reactions: Intolerance, interactions
  - Substance-induced (Note specific coding in DSM-5)
  - *High* fever (esp. children)
  - Sleep deprivation (excessive)
  - Etc.

Treatment & Prevention of Delirium

- Treatment
  - Address precipitating medical problems
  - Psychosocial interventions
    - Reassurance/comfort, coping strategies, inclusion of pt. in treatment decisions (when poss.)
- Prevention
  - Utilize proper medical care for illnesses
  - Emphasize proper use of, & adherence to, therapeutic drugs

"Dementia" ➔ Neurocognitive Disorder

- DSM-IV: "Dementia" & "Organic Mental Disorder"
  ➔ DSM-5: "**Neurocognitive Disorder**"

- Memory impairment *no longer essential* for diagnosis
- Includes range of disorders in which principal manifestation is an *acquired* loss of cognitive ability (objective decline from baseline) due to known (or assumed) brain damage or disease

"Dementia" ➔ Neurocognitive Disorder - 2

- All age groups*
- Greater specification of behavioral symptoms / syndromes
- Active use of objective neurocognitive assessment data
- Increasing *role* of biomarkers in diagnosis (but not yet required)

Neurocognitive Disorder

- Mild
  - Cognitive Deficits approx. 1-2 SD's below mean on neuropsychological testing
  - Cognitive deficits ***do not*** interfere w/ capacity for independence in daily activities
- Major
  - Cognitive Deficits > 2 SD's below mean on neuropsych. testing
  - Cog. deficits ***interfere*** w/ independence in daily activities

Neurocognitive Disorder: Specifiers

- Differentiation ***must be made*** between "possible" & "probable"
- Medical disease / problem that is *causing* the disorder ***must be specified***…

Types (Sources) of Neurocognitive Disorder

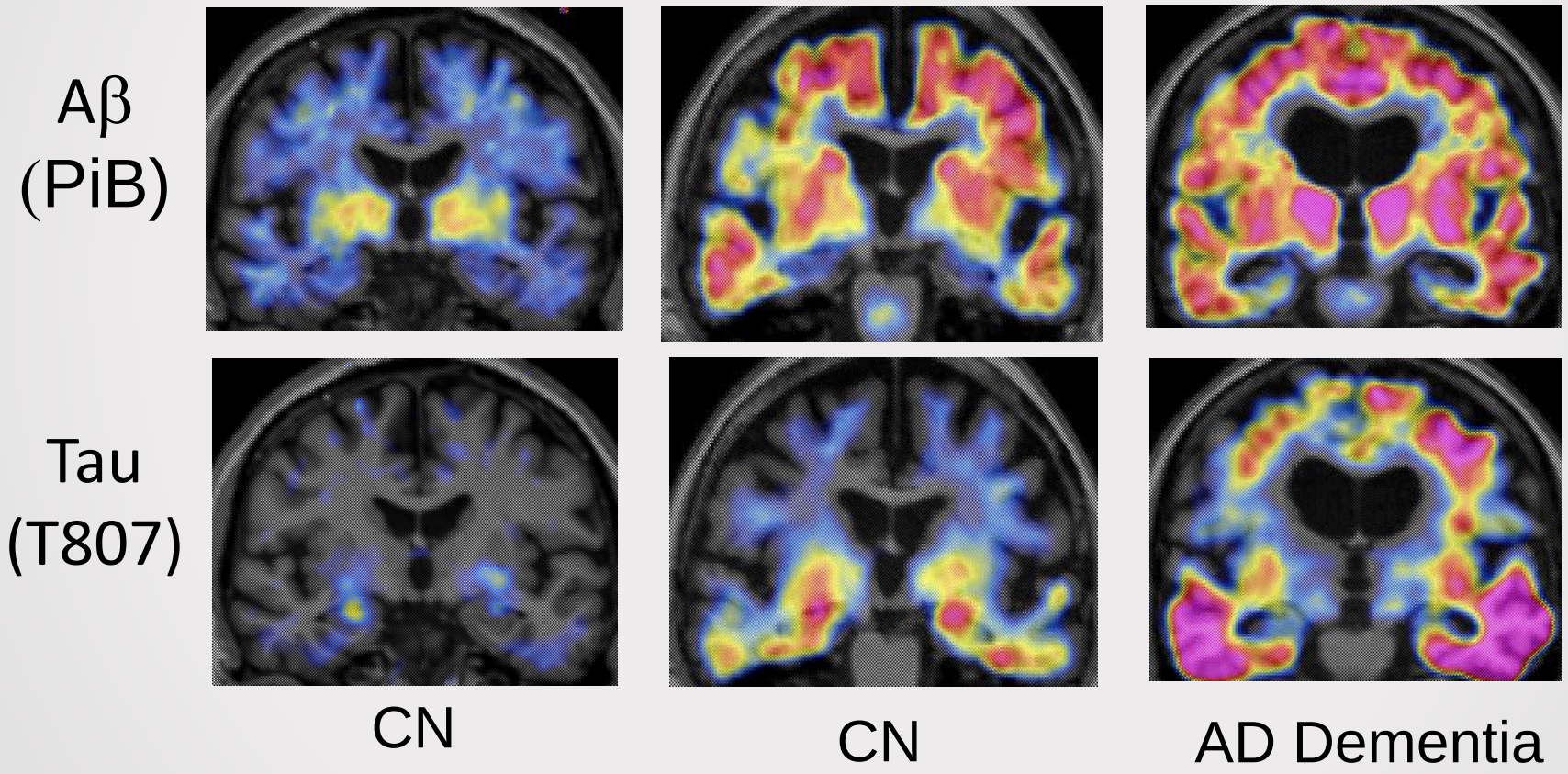
- Due to Alzheimer's Disease*
- Frontotemporal
- Vascular*
- With Lewy bodies*
- Due to Traumatic Brain Injury
- Substance/medication induced
- Due to HIV infection
- Due to prion disease
- Due to Parkinson's Disease
- Due to Huntington's Disease
- Due to another medical condition
- Due to multiple etiologies
- Unspecified

Subjective Cognitive Decline (SCD)

- Older adults express concern about perceived decline in cognitive abilities—yet, assessment WNL + IADL's remain intact (Jessen et al., 2014)
- "Worried well"? (Tuokko & Smart, 2018) or "CRS"?
  - Not necessarily—Seems distinct
- SCD ➔ Increased risk of AD when relevant biomarkers present
- Preclinical Alzheimer's Disease (up to 15 yrs before AD) (Sperling et al., 2011)

PET Amyloid & Tau Imaging

Sperling, Mormino, & Johnson (2014)



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Alzheimer's Disease (AD)

- ***Most common*** form of Major Neurocog. Disorder
- Identified by Dr. Alois Alzheimer in 1906 (Auguste D.)
- Char. by ***progressive*** brain deterioration & impaired cognitive function (esp. memory)
- *Primary* Cortical Structures Involved:
  - Hippocampus
  - Thalamus
  - Temporal Lobe
  - Basal Forebrain

Notable AD Characteristics

- Plaques (Senile…): Clusters of ***amyloid beta 42***
  - $A\beta_{42}$ very sticky ➔ easily forms plaques ***among*** axon terminals
  - Interferes w/ neural transmission ➔ *Eventual* neuron death

- Neurofibrillary Tangles: Abnormal accumulations of ***tau***
  - Tangles form ***inside of*** neurons ➔ Neuron death

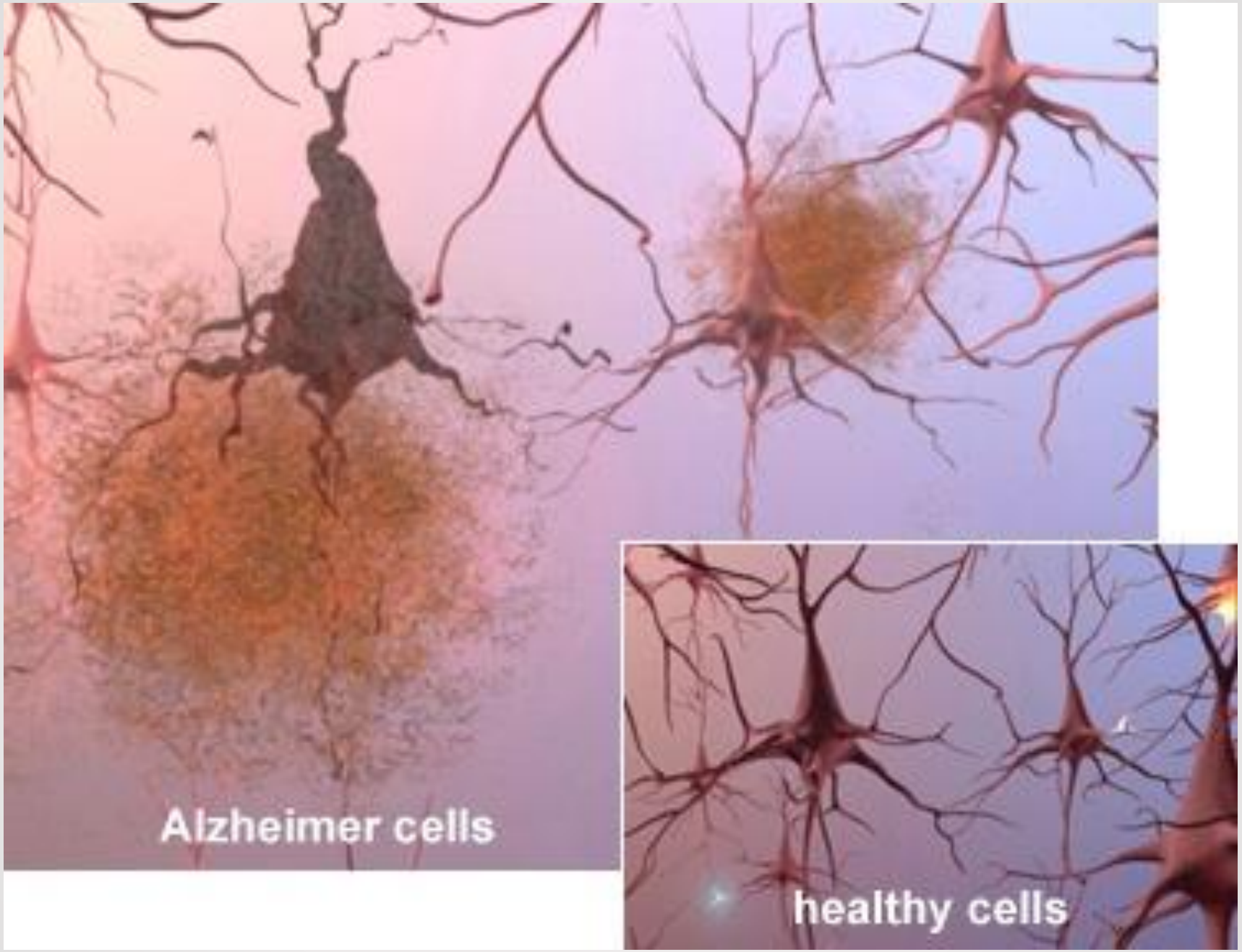

Image: https://www.alz.org/braintour/plaques_tangles.asp

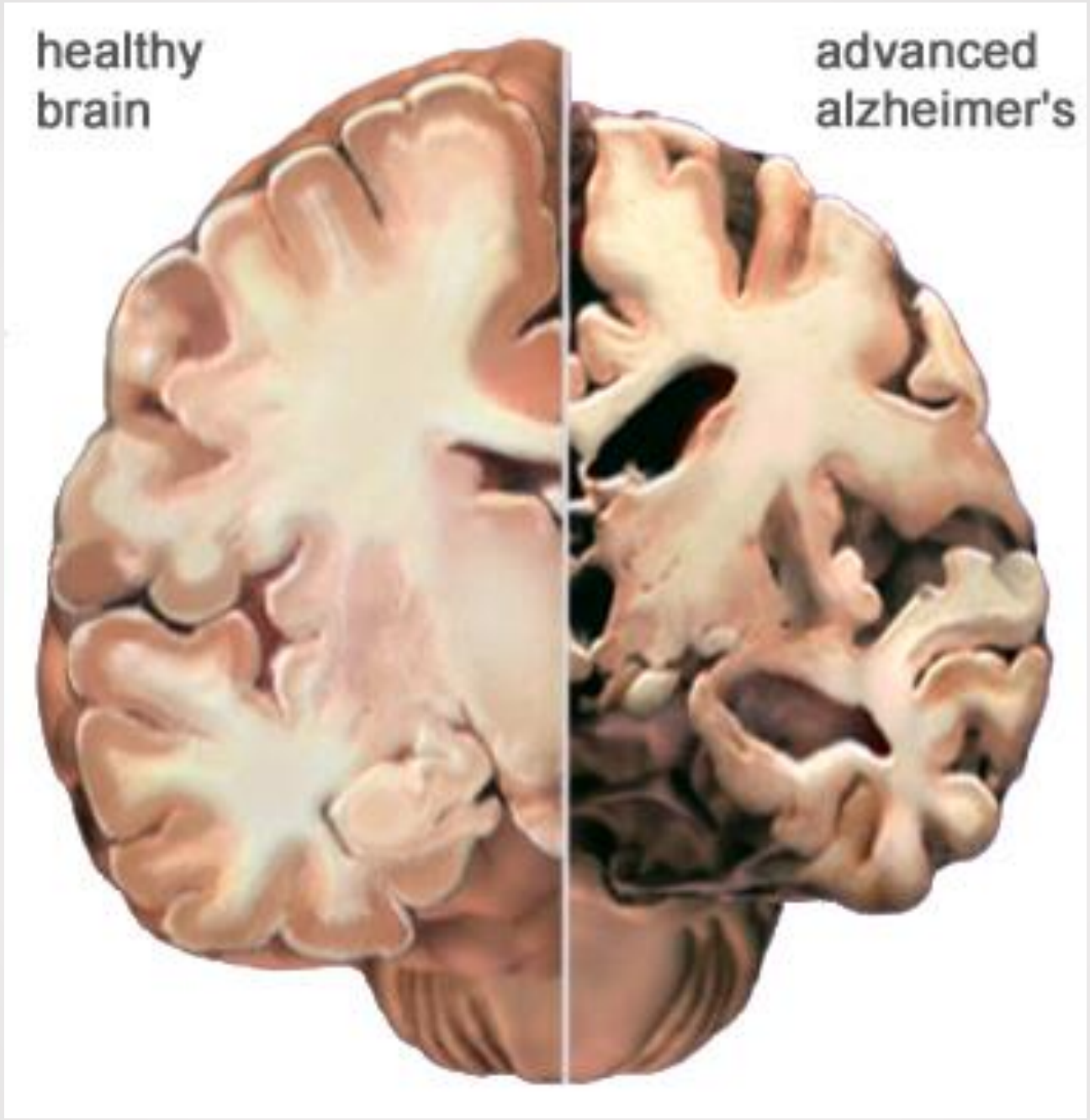

Image: https://www.alz.org/braintour/healthy_vs_alzheimers.asp

Heredity & Alzheimer's Disease

- A key aspect of AD (accounts for just > 50% of cases)
- Four Known Genes (& associated chromosome):
  - APP (21)
  - Presenilin 1 (14)
  - Presenilin 2 (1)
  - APOE ε4 (19) ➔ *Interacts* w/ tau to exacerbate pathogenic cascade (Shi et al., 2017)
- *Early*-Onset AD: Related to APP, Presenilin 1, & APOE-ε4 genes
- *Late*-Onset AD: Related to Presenilin 2 gene

Detecting / Diagnosing AD

- Autopsy: Most Common / Definitive
- PET Scan
- Comprehensive Medical Evaluation (*a rule-out process*)
- Neuropsychological Evaluation / Neurocognitive Screening:
  - Look for insidious onset & gradual progression
  - Anosognosia common (➔ Denial)
  - Short-term *verbal* memory impairment often primary symptom
  - Word-finding problems common

Current Alzheimer's Medication Options

- Acetylcholinesterase Inhibitors
  - A.k.a. Aricept, Razadyne, Exelon
  - Inhibits acetylcholinesterase from breaking down ACh, thereby *preserving* neuronal transmission
  - Effectively *slows progression* of AD—but does not stop it

Current Alzheimer's Medication Options - 2

- NMDA [N-methyl-D-aspartate] Blockers
  - A.k.a. Namenda
  - Limits NMDA receptor sensitivity to glutamate
  - Mechanism: Some dying AD neurons trigger release of glutamate ➔ excitotoxicity (overstimulation of NMDA receptors) ➔ neuron death
  - Also FDA indicated for Neurocog. Disorder w/ Lewy Bodies (DLB)

Vascular Neurocognitive Disorder

- ***Second*** most common form of Neurocog. Disorder
- Cause:
  - Damage to, or deterioration of, the vascular integrity of brain
- Sources:
  - Cerebrovascular disease, cardiac disease, hypertension, high cholesterol, smoking, etc.
- *Variable pattern* of neurocognitive impairment

Vascular Neurocognitive Disorder - 2

- "Probable" if (≥ 1 of following)…
  - Clinical criteria supported by neuroimaging evidence of signif. lesions, attributed to cerebrovascular disease
  - Neurocog. syndrome is *temporally* related to ≥ 1 documented cerebrovascular events
  - Clinical *&* genetic evidence of cerebrovascular disease is present

Stroke: Primary Types

- Ischaemic
  - Arterial clogging / blockage
  - TIA if < 24 hrs. (usu. w/ symptom remission), CVA / Stroke if > 24 hrs.

- Hemorrhagic
  - Arterial rupture ➔ Intracranial bleeding

Neuroimaging of Ischaemic Stroke

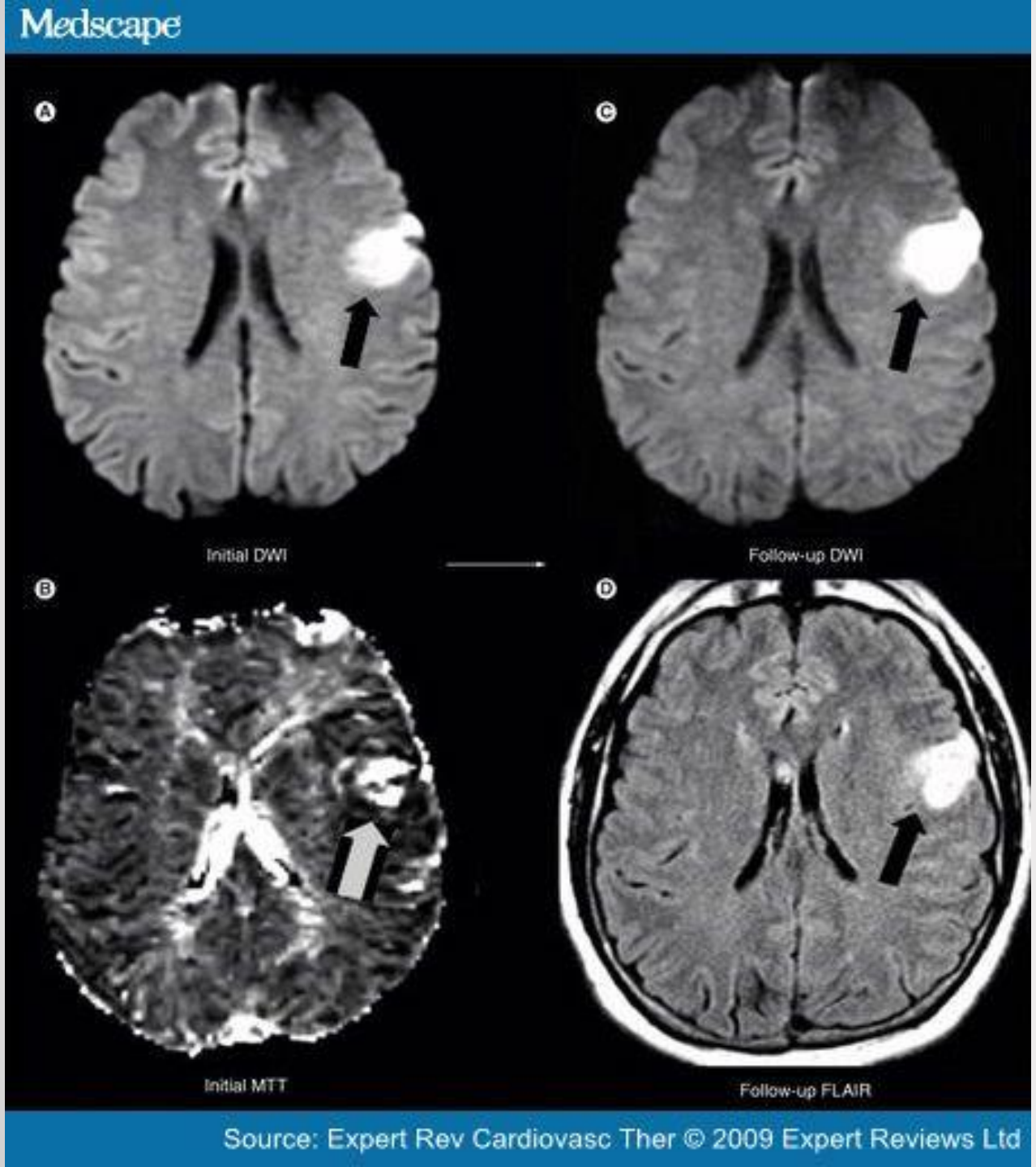

Image: http://www.medscape.com/viewarticle/587073_6

Subcortical Ischaemic Vascular Disease

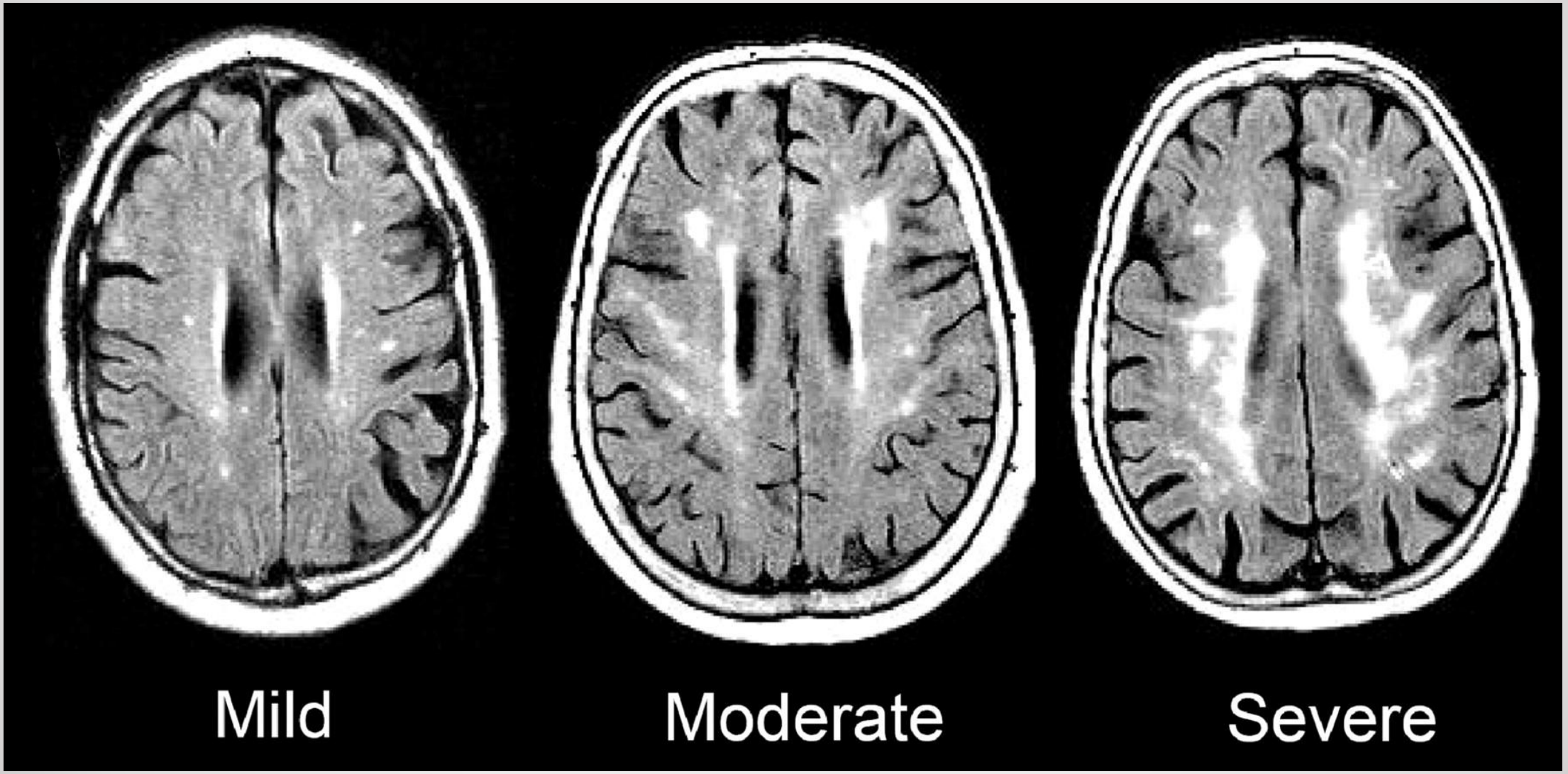

Image: http://ischemicskolik.blogspot.com/2015/08/chronic-ischemic-changes-in-brain.html

Detecting / Diagnosing Vascular Neurocognitive Disorder

- Documented Evidence of Cerebrovascular Disease
- *Stepwise progression* of cognitive impairment common
- Neuropsychological Evaluation / Neurocognitive Screening:
  - Look for temporal causation
  - Look for correlation between lesion location(s) & affected cognitive function
  - Client often (not always) aware of problems

Neurocognitive Disorder w/ Lewy Bodies

- ***Third*** most common form of Neurocog. Disorder
  - A.k.a. DLB or Lewy Body Disease
- Apparent Hybrid: Symptoms / characteristics of Parkinson's Disease & Alzheimer's Disease…
  - Episodes of seemingly unexplained clouding or loss of consciousness
  - Episodes of seemingly unexplained falling

Neurocognitive Disorder w/ Lewy Bodies - 2

- Apparent Hybrid [cont.] ….
  - Spontaneous development of "parkinsonism" (tremors, etc.) ***after*** dev't of cognitive problems (often starting w/ executive dysfunction)
  - Recurrent visual hallucinations (well-formed, detailed)
  - Severe neuroleptic sensitivity
  - REM Sleep Behavior Disorder criteria met

Lewy Bodies

- Abnormal protein accumulation ***inside*** neurons
- Found in pts w/ Parkinson's disease (PD), Lewy Body Disease (Neurocognitive Disorder due to…), & a few others
- Identified under microscope when histology is performed on the brain…

Lewy Bodies - 2

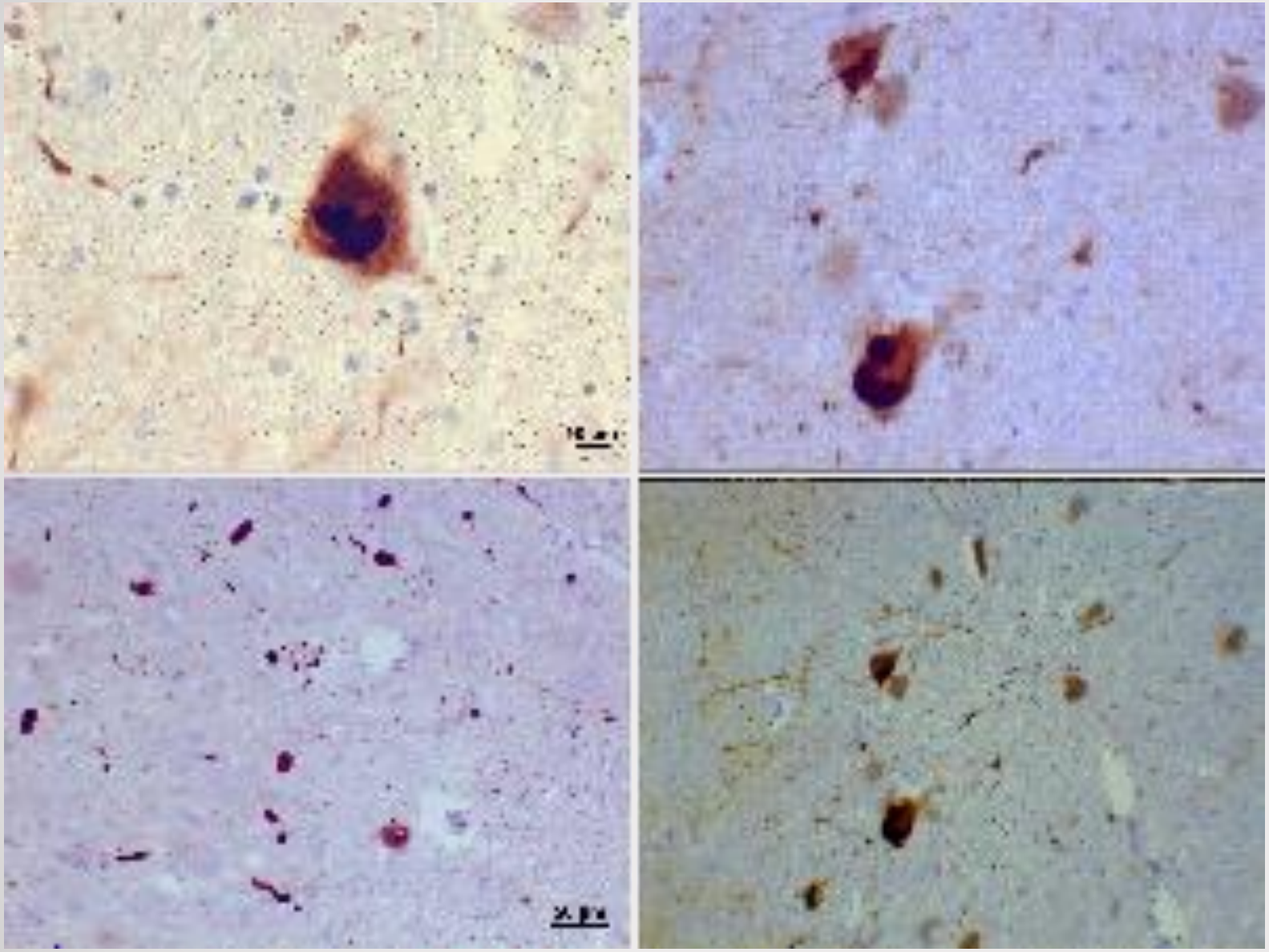

Image: https://en.wikipedia.org/wiki/Lewy_body

Detecting / Diagnosing Neurocognitive Disorder w/ Lewy Bodies

- Documented Evidence of Clouding / Loss of Consciousness and / or Unexplained Falling
- Histologic Evidence of Lewy Bodies
- Neuropsychological Evaluation / Neurocognitive Screening:
  - Look for relatively *intact* short-term memory functioning early
  - Executive dysfunction often an early issue
  - Do cognitive problems precede Parkinsonism?

References - 1

- Administration for Community Living (2012). *Policy academy state profile: South Dakota's population*. Retrieved from https://www.acl.gov/sites/default/files/programs/2016-11/South%20Dakota.pdf
- American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.
- Jessen, F., Amariglio, R.E., van Boxtel, M., Breteler, M., Ceccaldi, M., Chételat, G.,…Wagner, M. (2014). A conceptual framework for research on subjective cognitive decline in preclinical Alzheimer's disease. *Alzheimer's and Dementia, 10,* 844-852. doi:10.1016/j.jalz.2014.01.001
- Lezak, M.D., Howieson, D.B., Bigler, E.D., & Tranel, D. (2012). *Neuropsychological assessment* (5th Ed.). New York, NY: Oxford University Press.
- Roebuck-Spencer, T.M., Glen, T., Puente, A.E., Denney, R.L., Ruff, R.M., Hostetter, G., & Bianchini, K.J. (2017). Cognitive screening tests versus comprehensive neuropsychological test batteries: A National Academy of Neuropsychology education paper. *Archives of Clinical Neuropsychology, 32,* 491–498. doi:10.1093/arclin/acx021
- Sachdev, P.S., Blacker, D., Blazer, D.G., Ganguli, M., Jeste, D.V., Paulsen, J.S., & Petersen, R.C. (2014). Classifying neurocognitive disorders: The DSM-5 approach. *Nature Reviews Neurology, 10,* 634–642. doi:10.1038/nrneurol.2014.181

References - 2

- Shi, Y., Yamada, K., Liddelow, S.A., Smith, S.T., Zhao, L., Luo, W., Tsai, R.M.,…Holtzman, D.M. (2017). ApoE4 markedly exacerbates tau-mediated neurodegeneration in a mouse model of tauopathy. *Nature, 549*, 523-527.
- Sperling, R.A., Aisen, P.S., Beckett, L.A., Bennett, D.A., Craft, S., Fagan, A.M., Iwatsubo, T.,…Phelps, C.H. (2011). Toward defining the preclinical stages of Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's and Dementia, 7*, 280-292. doi:10.1016/j.jalz.2011.03.003
- Sperling, R.A., Mormino, E., & Johnson, K. (2014). The evolution of preclinical Alzheimer's disease: Implications for prevention trials. *Neuron, 84*, 608-622. http://dx.doi.org/10.1016/j.neuron.2014.10.038
- Suburban Stats (2018). *Current Population Demographics and Statistics for South Dakota by age, gender and race*. Retrieved from https://suburbanstats.org/population/how-many-people-live-in-south-dakota
- Tuokko, H.A., & Smart, C.M. (2018). *Neuropsychology of cognitive decline: A developmental approach to assessment and intervention*. New York: Guilford Press.