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**Disciplines**
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**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
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

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

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

“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

Lobes of the Brain

General Lobe Functions

Frontal Lobe
- movement
- thinking initiation
- reasoning (judgement)
- behaviour (emotions)
- memory
- speaking

Parietal Lobe
- knowing right from left
- sensation
- reading
- understanding spacial relationships

Occipital Lobe
- vision
- colour blindness

Temporal Lobe
- understanding language
- behaviour
- memory
- hearing

Brain stem
- breathing
- blood pressure
- heartbeat
- swallowing
- alertness/sleep
- body temperature
- digestion

Cerebellum
- balance
- coordination
- fine muscle control

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
• 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 nonverbal 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
  o Early identification of those at risk of decline
  o Indication of need for referral for additional evaluation / treatment
  o 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](http://www.mocatest.org))
  - Mini-Mental Status Examination (MMSE)
  - Geriatric Depression Scale – Long Form (GDS)
  - Geriatric Anxiety Scale (GAS) ([www.uccs.edu/agingandmentalhealthlab/scale](http://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.

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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

- 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”?  
  o 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*
  o Aβ\textsubscript{42} very sticky $\Rightarrow$ easily forms plaques *among* axon terminals
  o Interferes w/ neural transmission $\Rightarrow$ *Eventual* neuron death

• Neurofibrillary Tangles: Abnormal accumulations of *tau*
  o Tangles form *inside of* neurons $\Rightarrow$ Neuron death
Image: https://www.alz.org/braintour/plaques_tangles.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) \(\rightarrow\) 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 Neurocognitive 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 significant 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

Subcortical Ischaemic Vascular Disease

Detecting / Diagnosing Vascular Neurocognitive Disorder

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

• *Third* most common form of Neurocog. Disorder
  o 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

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


