Dementia Overview

David Weidman, MD

Goals of Presentation

1. Differentiate between normal cognitive changes with aging, mild cognitive impairment and dementia, defining MCI and dementia
2. Describe the most common types of dementia and their different presentations
3. Review appropriate evaluation of MCI and dementia, with cognitive screening tests, laboratory and imaging tests
4. Review treatment of dementia
5. Review delirium, diagnosing and managing

Cognitive Changes with Aging

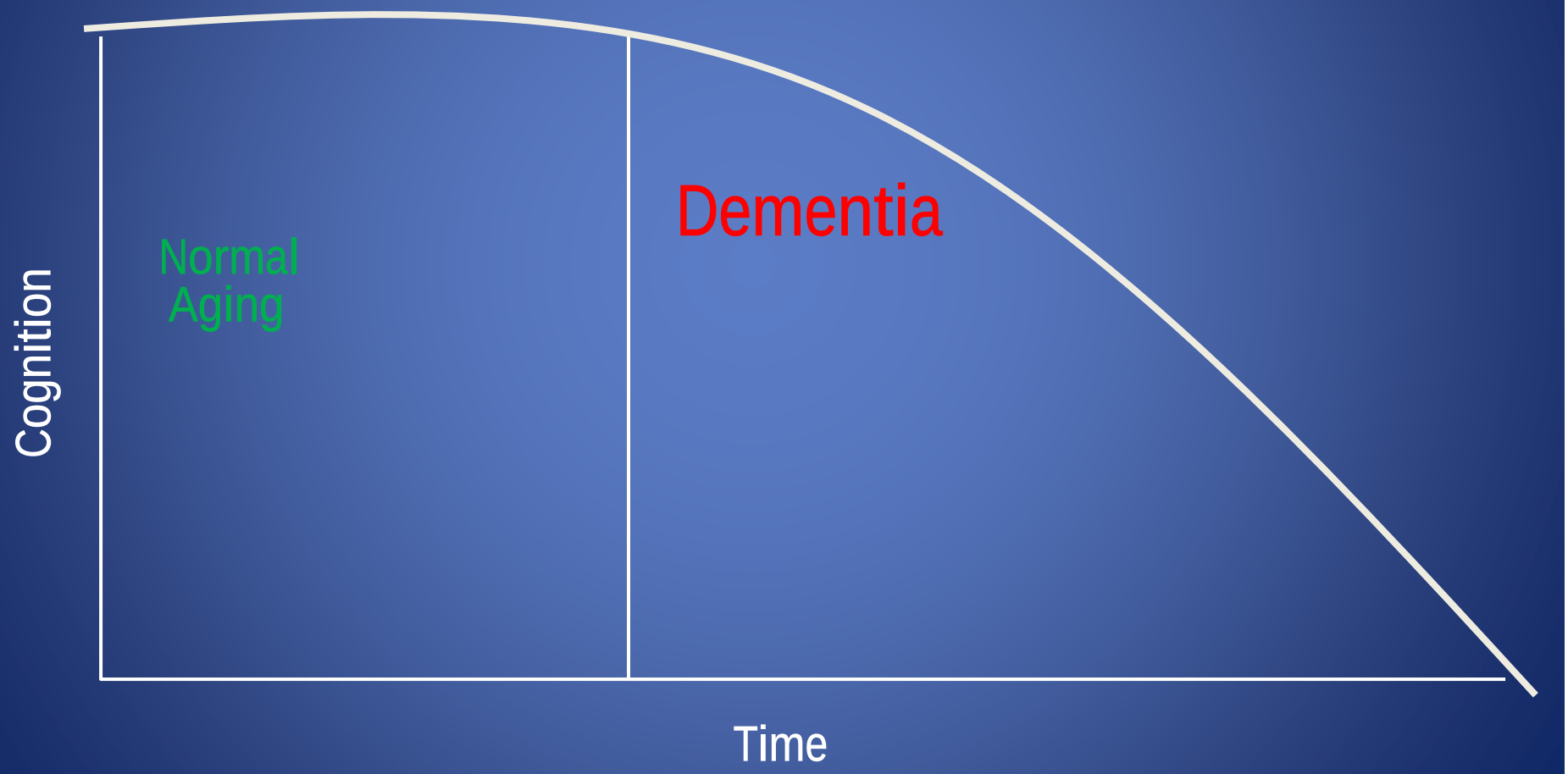
- Mild changes in memory
  - decline in rate of learning new information but not in memory retention
- Rate of information processing slows
- More difficulty with multi-tasking
- Mild word finding difficulty (especially names)
- “Sometimer’s”

Decades ago

- Normal cognitive aging vs. dementia
  - Satisfactory distinction to make
  - Didn't define dementia as a specific stage of cognitive impairment
  - Alzheimer's vs. other dementias* was a focus
  - Even before that, "Pre-senile" and "Senile" dementia

*EG, "Hardening of the arteries"

Hypothetical Course of Cognitive Function in a Dementia Patient

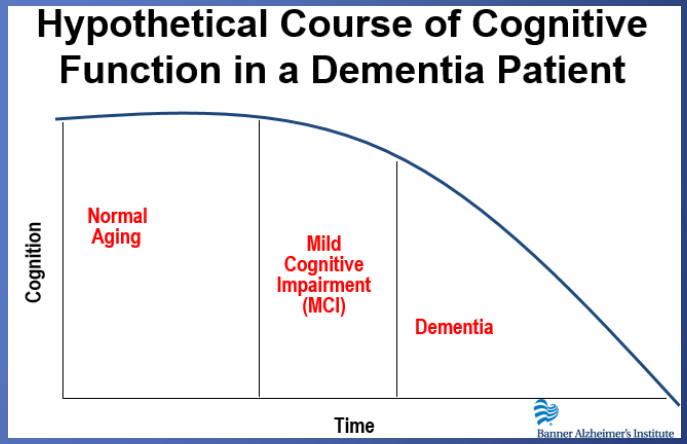


MCI is "Construct"

- Core criteria or attributes medical researchers and clinicians define as entity to examine
- Plausible biologically
- Somewhat man-made,

  like a canal, so not even

  a syndrome



- The criteria may evolve over time

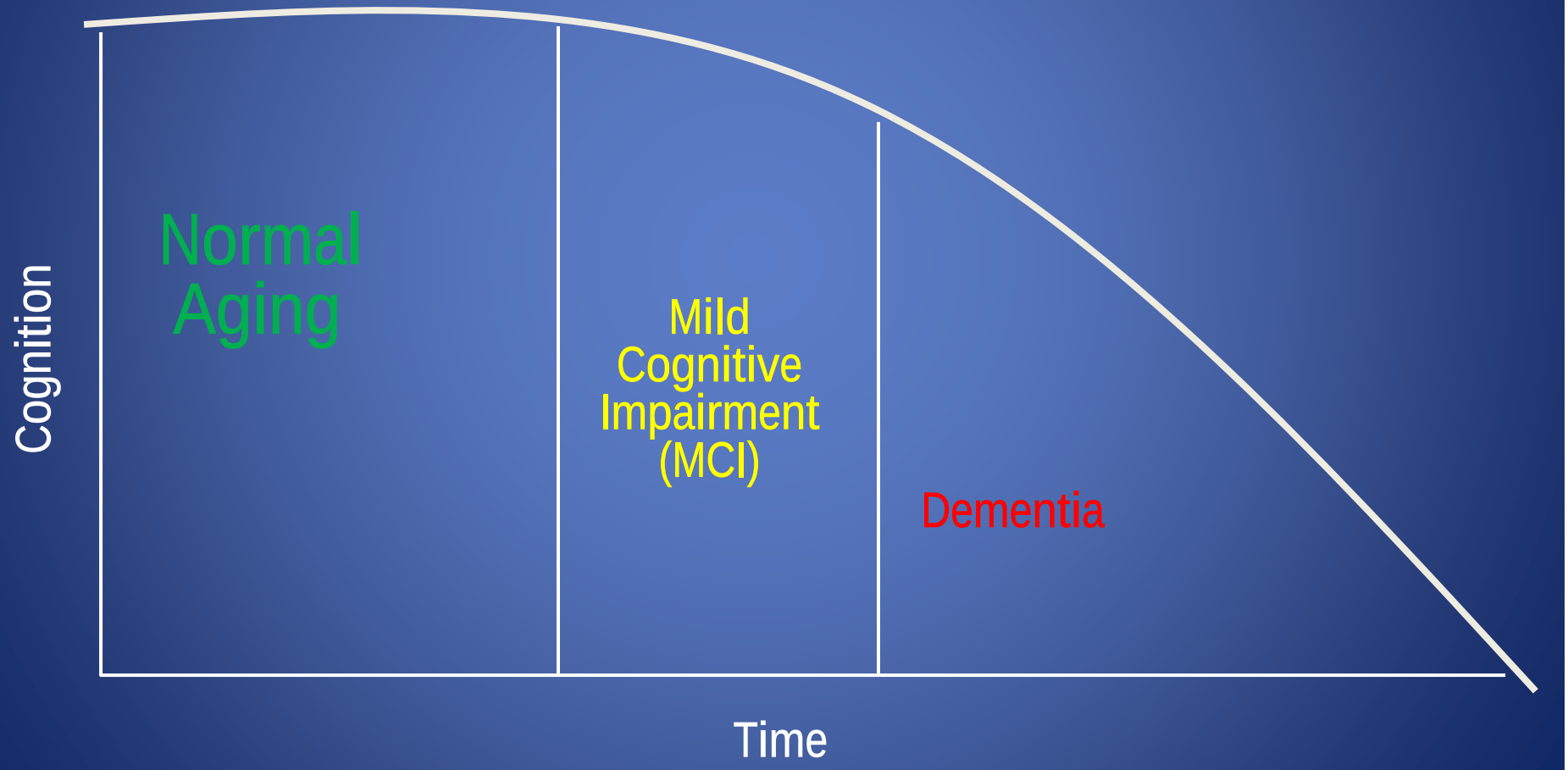
Mild Cognitive Impairment (MCI)

Cognitive decline more than expected for normal aging

- Cognitive decline from a previous level of performance
- Cognitive impairment **does not** impair everyday activities (work, IADLs, ADLs)
- Does not occur exclusively during the course of delirium
- Not accounted for by another mental disorder (depression, schizophrenia, etc.)

DSM V – Minor Neurocognitive Disorder

Hypothetical Course of Cognitive Function in a Dementia Patient



More recently

- Emerging in late 1980's: A borderland or "grey zone" transition state, before functional decline, patients still accomplishing tasks independently, but problems forgetting noticed by patient, family and care partners, or his or her physician
- Termed Mild Cognitive Impairment: helpful to describe, detection needed to start treatment earlier, including drug trials
- Impairment in thinking skills which goes beyond normal age-related cognitive changes, but not dementia (and can't state "not yet" with certainty, doesn't always progress!)

MCI Causes

Reversible, Readily treatable Conditions

- Depression
- Severe stress
  - anxiety
  - Occupational burnout
- Obstructive sleep apnea
- Metabolic disturbance
  - B12 lack; hypothyroid
- Alcohol
- Other toxins
- Infection

Neurodegenerative disorders

- Alzheimer's disease
- Vascular dementia
- Lewy Body dementia, PD
- Frontotemporal dementias
- Mixes of the above
- PSP/CBD, CJD, NPH, Amyloid angiopathy

MCI

- There is no single cause of MCI
- Symptoms may remain stable for years, improve over time, or progress to dementia
- No FDA approved treatment at this time

Symptom domains

- Cognitive domains
  - Memory, Learning
  - Language
  - Visuospatial ability; perceptual-motor
  - Executive function, problem solving
  - Attention
- Social, behavior

Functional Decline – Symptoms

1. Occupation
2. Social
3. Instrumental ADLs (IADLs)

usually affected earlier in the disease process

- Housework
- Shopping
- Using the telephone
- Medications
- Managing money
- Transportation

4. Basic ADLs –

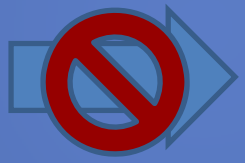
affected later in disease process

- Functional mobility
- Bathing/showering
- Dressing
- Grooming and hygiene
- Toileting

Mild Cognitive Impairment

Cognition

Cognitive decline from a previous level of performance



Function

Cognitive impairment **does not** impair everyday activities (work, IADLs, ADLs)

Mild Cognitive Impairment (MCI) Amnestic Type: Clinical Definition

- DOMAIN
  - Memory *only*
  - Memory impairment corroborated by friends/family
  - Intact ADLs
- TEMPO
  - Gradual onset, variable decline
  - Conversion to AD: ~ 16 – 20% per year
- ASSOCIATED FINDINGS
  - Otherwise normal
- CONTEXT
  - Otherwise healthy

Petersen R. Mild Cognitive Impairment. New York: Oxford, 2003

Mild Cognitive Impairment (MCI) Amnestic Type: Research Definition

- Subjective memory impairment corroborated by other informants
- Performance on objective memory tests > 1.5 SD below norms for age & education
- Intact ADLs
- Otherwise normal cognitive function

Albert MS et al. Alz & Dem 2011; 7: 270-279

Dementia

Cognition

Cognitive decline from a previous level of performance

Function

Cognitive impairment **does** impair everyday activities

- Occupation
- Social
- IADLs
- ADLs

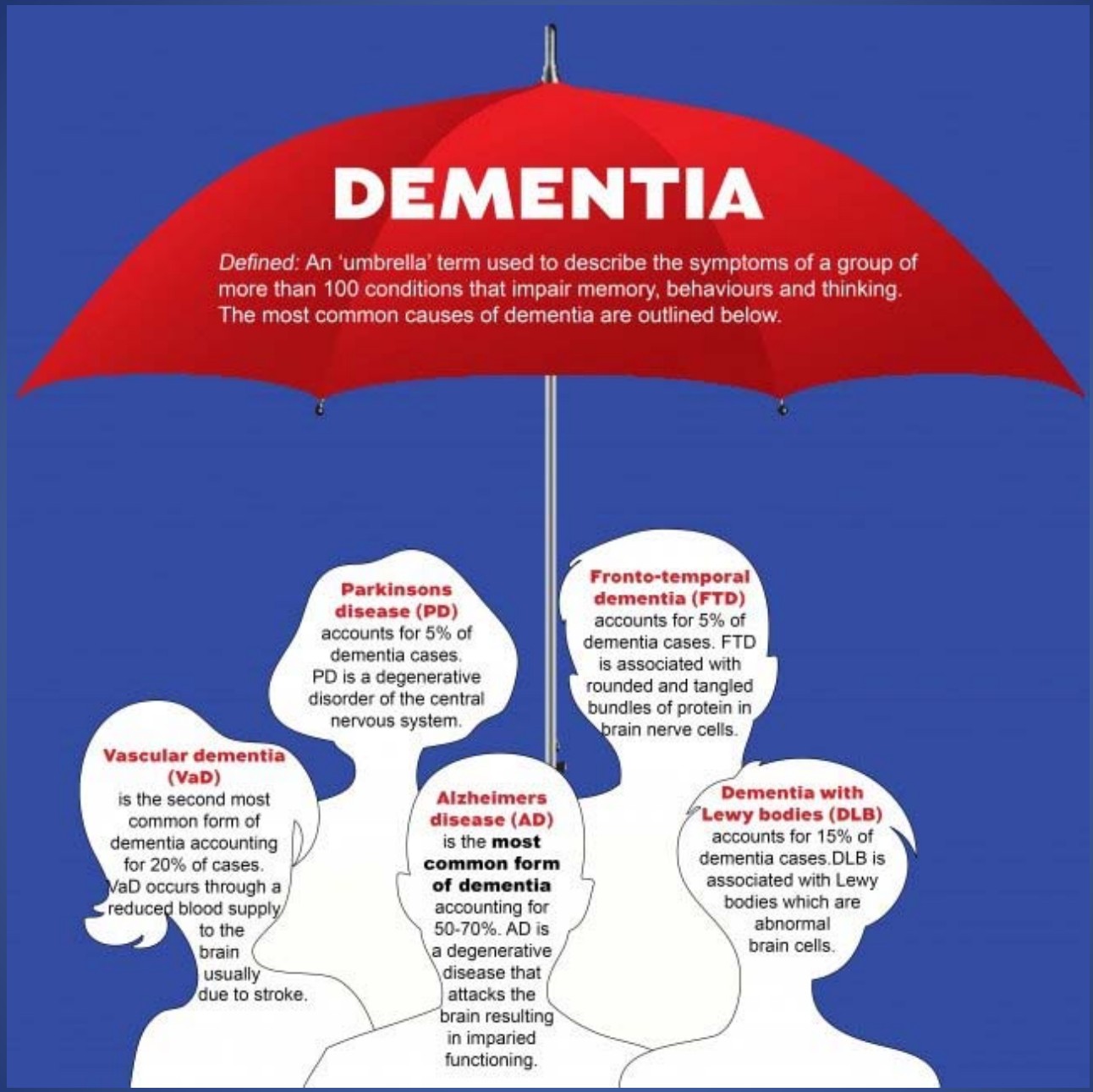
What is “Dementia?”

- It is not a specific disease, but is a descriptive term
- Clinical syndrome (collection of symptoms)
- There are many different causes or types of dementia
- Clinical diagnosis vs. Pathologic diagnosis

Dementia - Definition

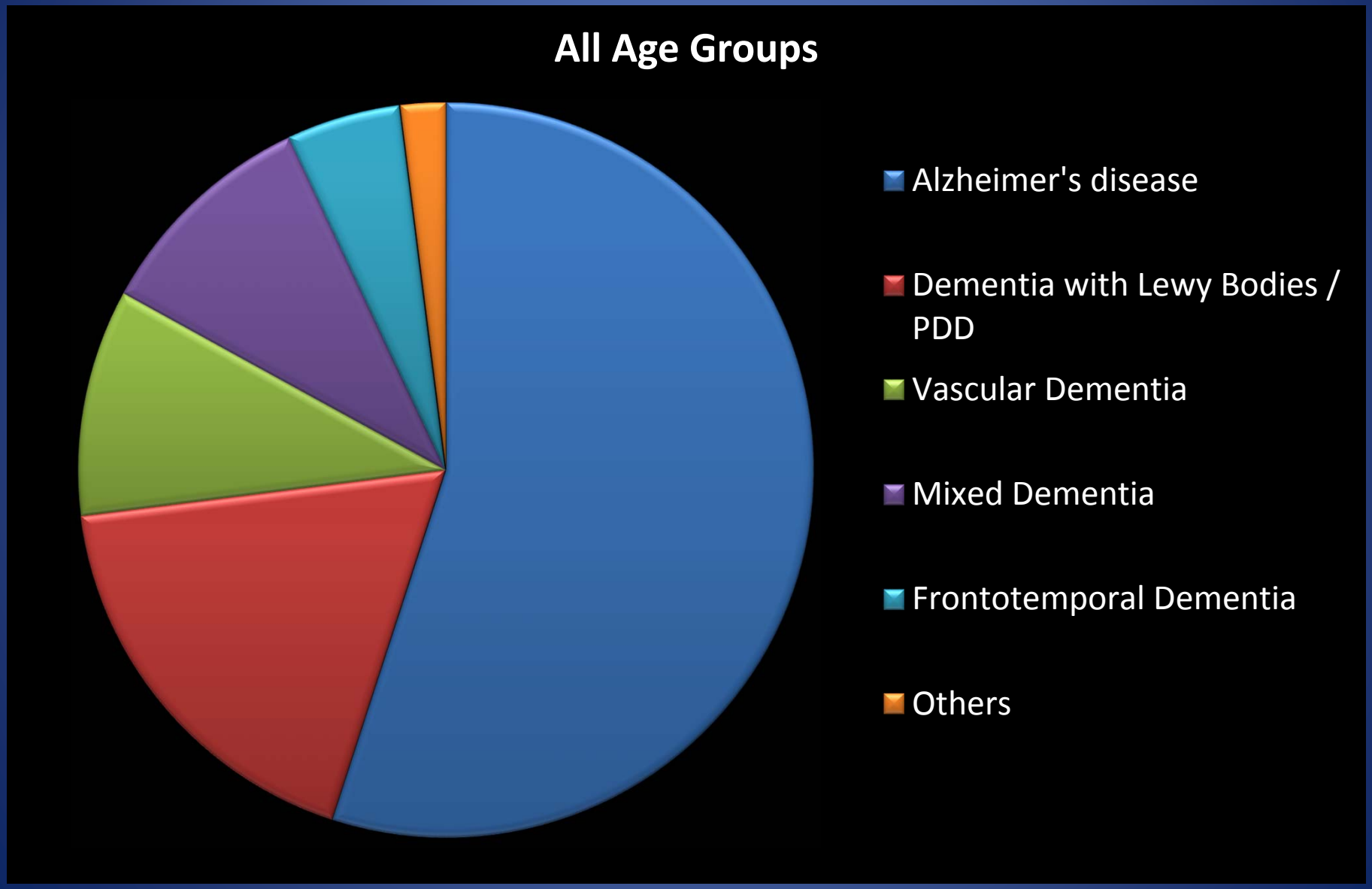
- Cognitive decline from a previous level of performance
- Cognitive impairment **does** impair everyday activities (work, IADLs, ADLs)
- Does not occur exclusively during the course of delirium
- Not accounted for by another mental disorder (depression, schizophrenia, etc.)

DSM V – Major Neurocognitive Disorder



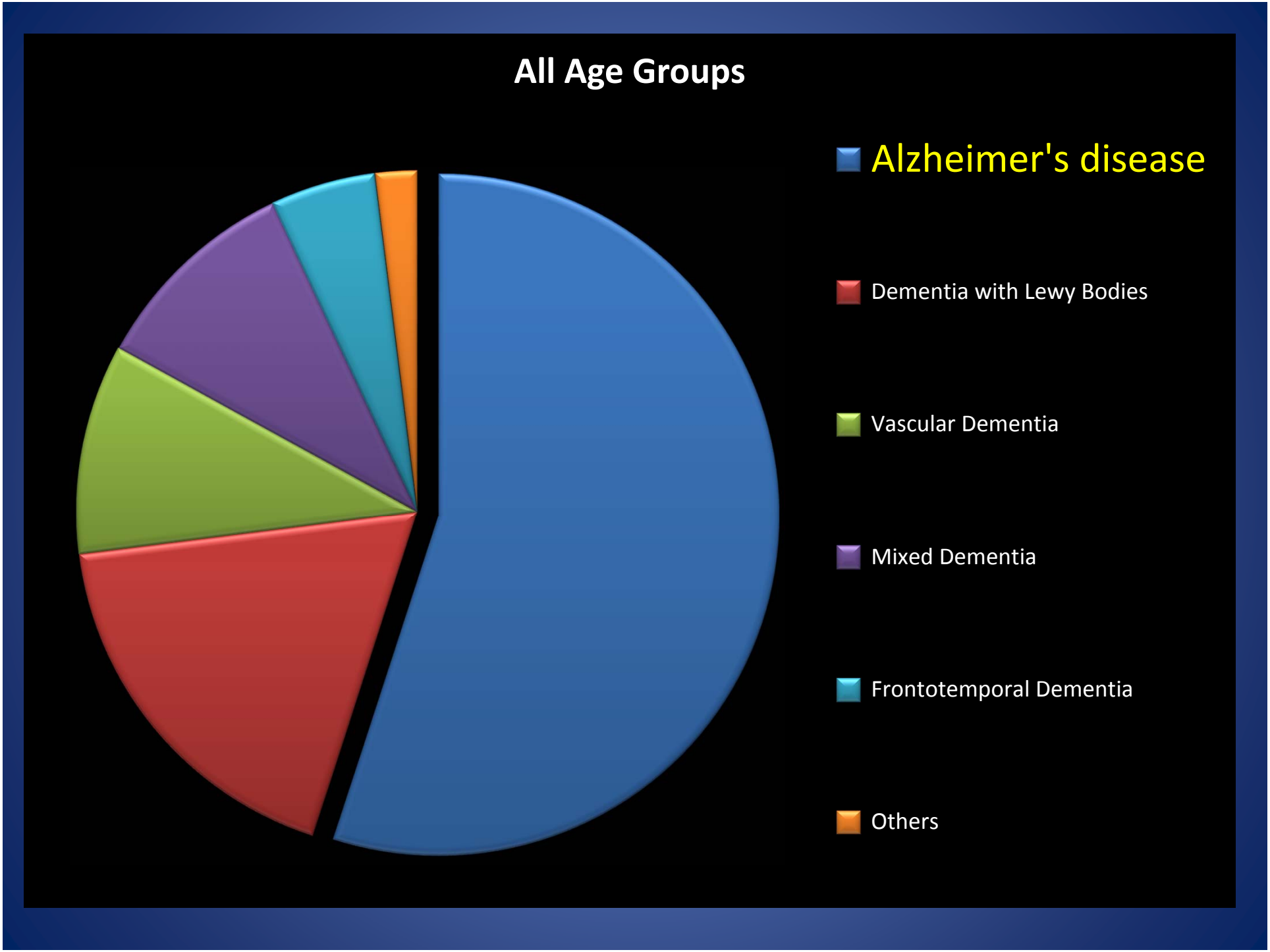
dementiaforward.org.uk

Dementia


All Age Groups
Alzheimer's disease
Dementia with Lewy Bodies / PDD
Vascular Dementia
Mixed Dementia
Frontotemporal Dementia
Others

Types of Dementia

- Alzheimer's disease
- Dementia with Lewy Bodies and Parkinson's disease with Dementia
- Vascular Dementia
- Mixed Dementia
- Frontotemporal Dementia
- Other (eg, Normal Pressure Hydrocephalus)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Alzheimer's Disease

- Most common form of dementia
- Progressive neurodegenerative disorder that damages and eventually destroys brain cells
- Greatest known risk factor is age
- 5% of people with the disease are < 65 years
- Microscopic changes in the brain begin long before the first signs of memory loss (preclinical phase)

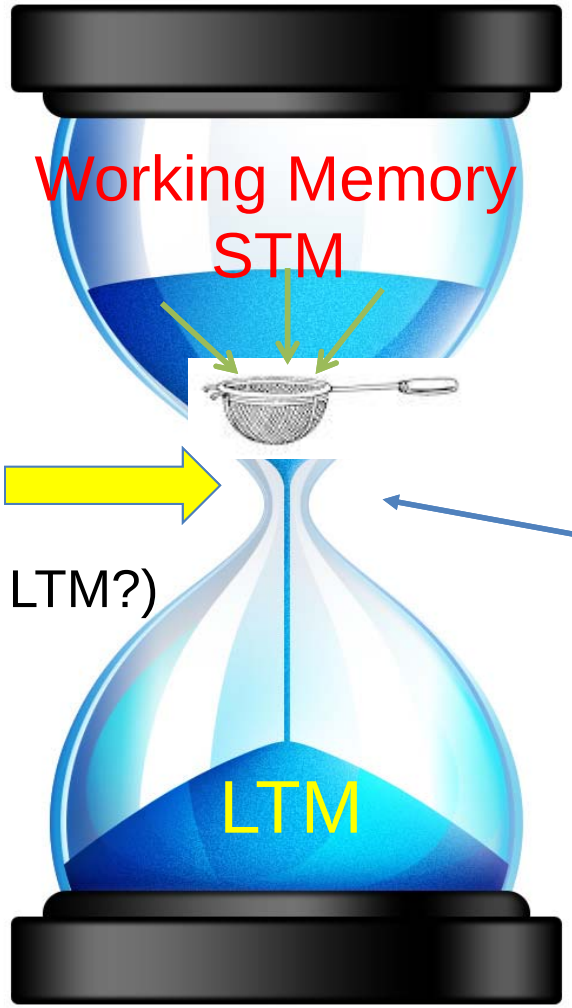
Key Concepts of AD

- Age is the single greatest risk factor
- Acetylcholine is the main transmitter affected
  - Glutamate, NE, 5-HT, & others are affected
- Apo-E status increases risk
- Role of insulin-like growth factors unclear
- Aβ-42 is increased in brain but *low* in CSF
- Tau is increased in CSF
- PET CT and β-amyloid CT may soon be approved for general use

Alzheimer’ s Disease - Symptoms

Insidious onset and progressive decline

- ★ Memory changes
- Increasing forgetfulness
- Mild confusion
- Repetitive questions or stories
- Misplacing items
- Difficulty planning
- Personality and behavior changes
  - Neuropsychiatric Symptoms
  - Depression, anxiety, withdrawal, irritability, aggression, etc.

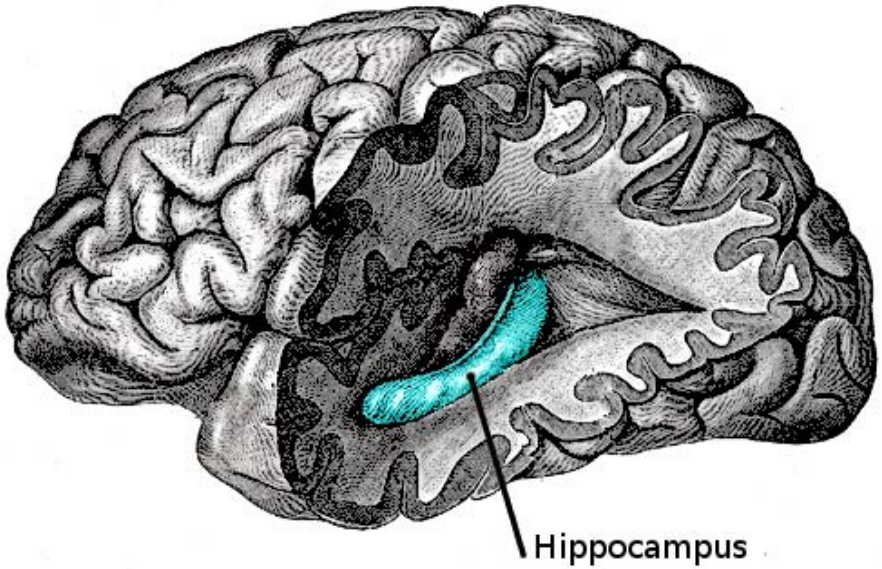


So what brain area filters all new info?

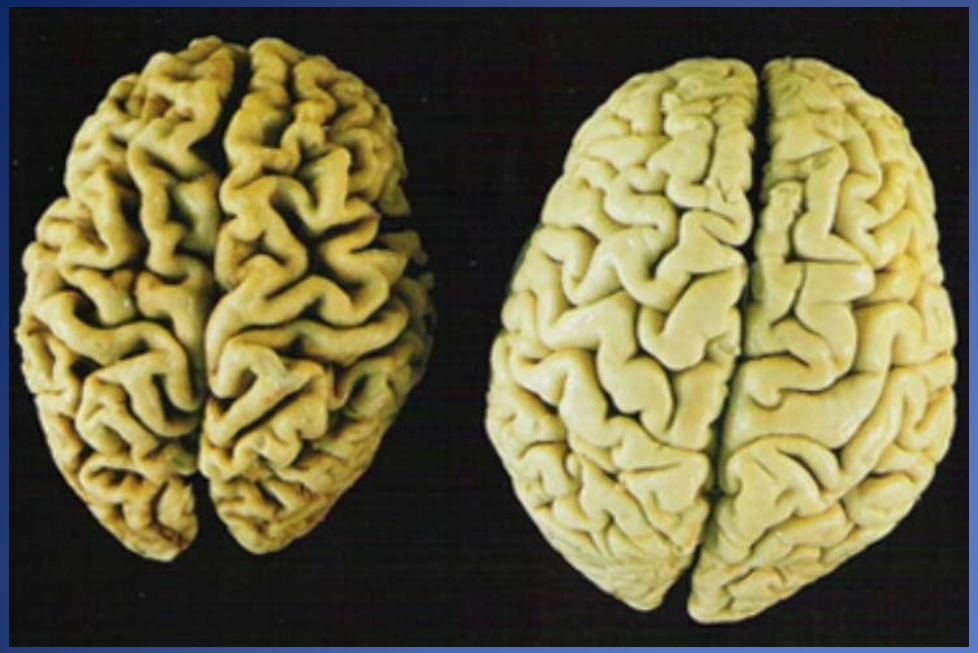
(if a STM is to become a LTM?)

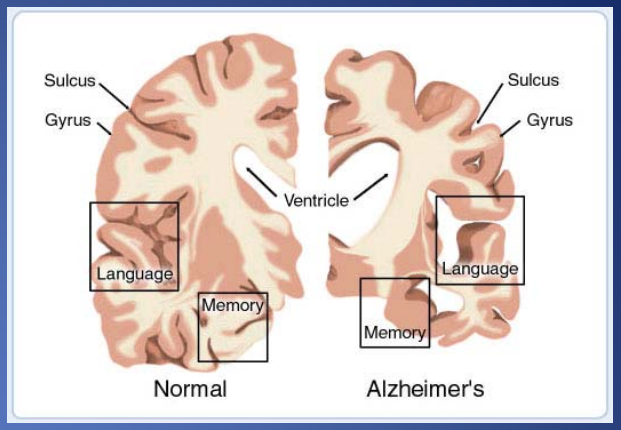
Encoding

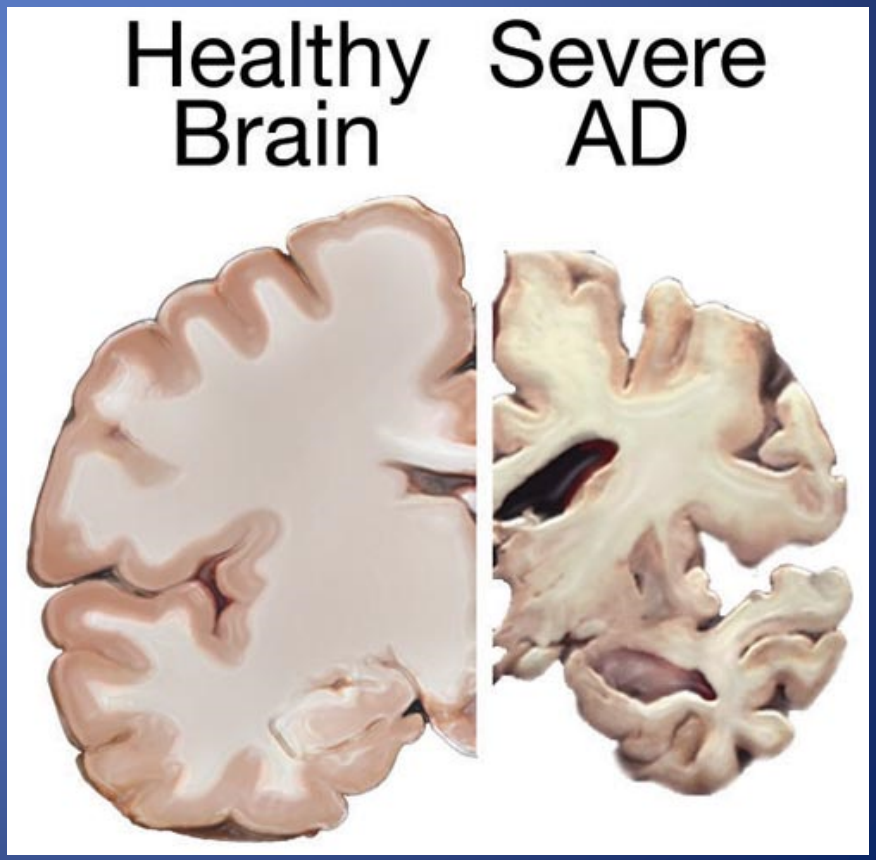
Banner Alzheimer's Institute



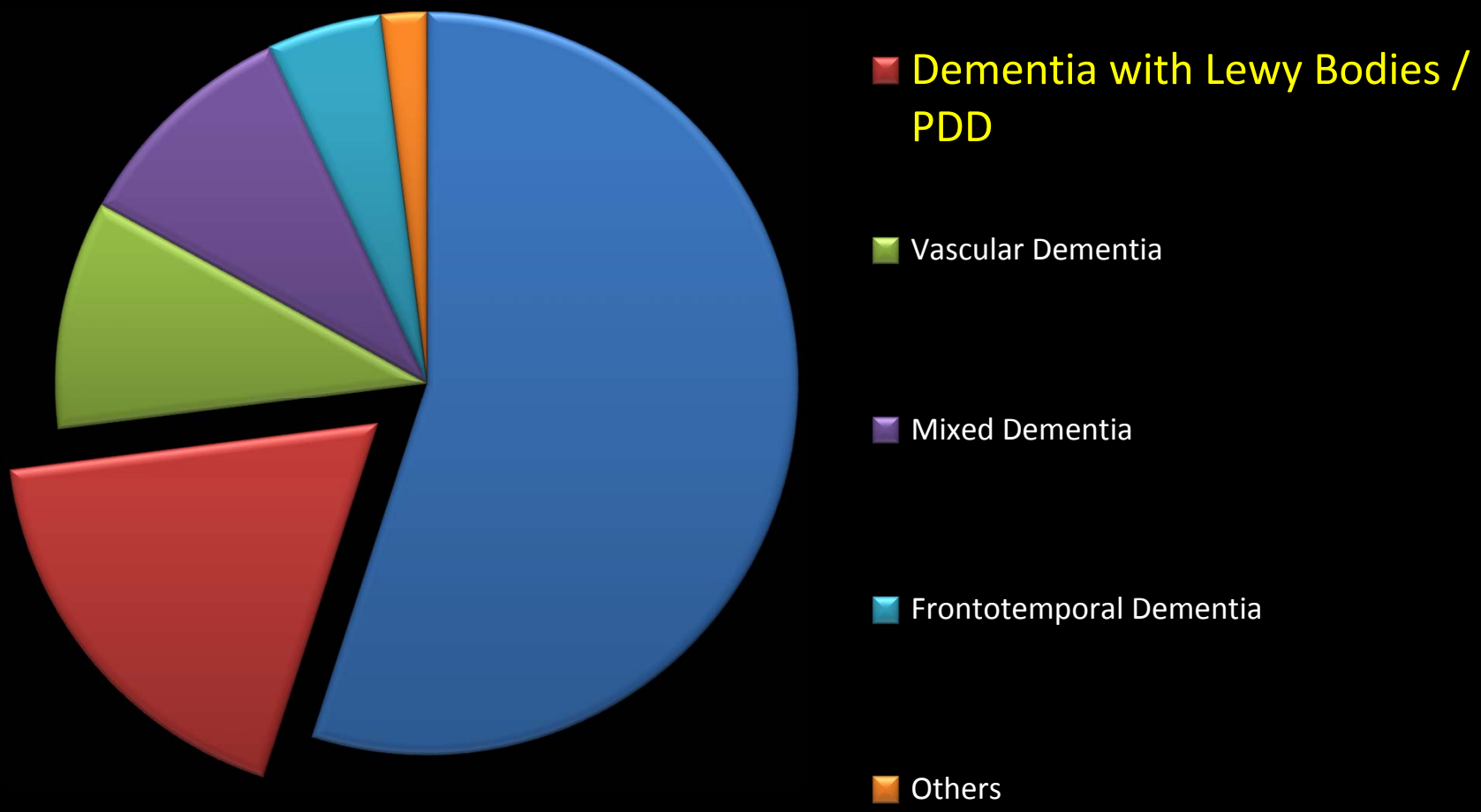
Hippocampal volume
Total brain volume




Sulcus
Gyrus
Ventricle
Language
Memory
Sulcus
Gyrus
Language
Memory
Normal
Alzheimer's

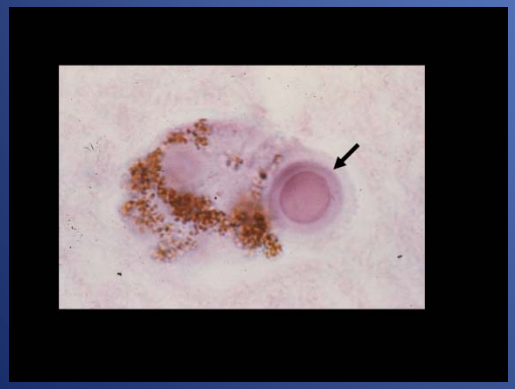

Healthy Brain
Severe AD

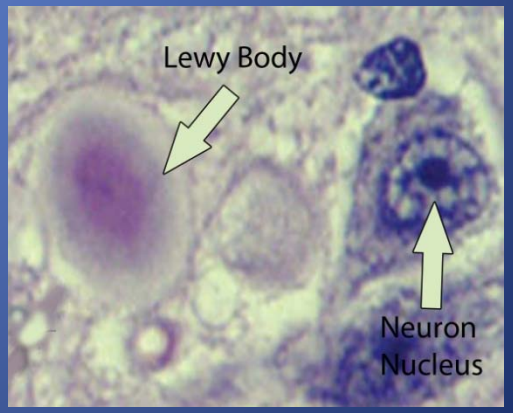
All Age Groups



Dementia with Lewy Bodies

- Progressive neurodegenerative disease
- Proteins called Lewy Bodies (alpha synuclein) are deposited in nerve cells
- Prominent memory impairment may not be evident in early stages





Dementia with Lewy bodies

Early impairment of visual-spatial skills and attention

Core Clinical Features

- Fluctuations
  - Cognition and levels of alertness
  - Subtle or dramatic
- Visual Hallucinations
  - Occur early in the disease (occurs later in other forms of dementia)
- Parkinsonism
  - Bradykinesia, gait disorder, limb rigidity
  - Usually more symmetric than PD and often without tremor

Dementia with Lewy Bodies

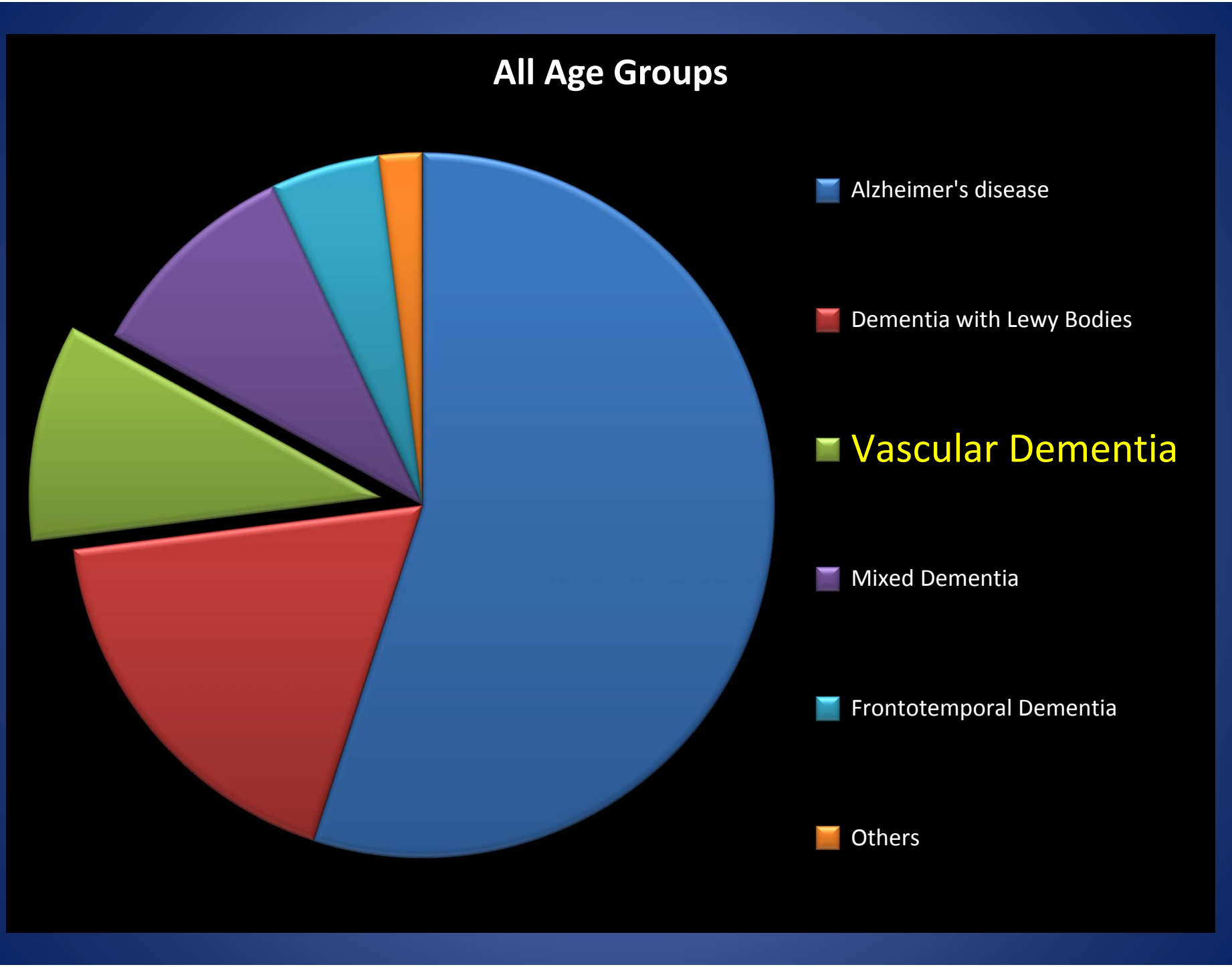
- REM sleep behavior disorder
  - Dream reenactment and vocalizations
  - Usually occurs early in the course of the disease
- Neuroleptic Sensitivity
- Autonomic dysfunction
  - Repeated falls
  - Syncopal episodes

Dementia with Lewy Bodies

- “1 year rule” (to distinguish from idiopathic Parkinson’s
  - Cognitive impairment will start before, concurrently, or within 1 year of Parkinsonism
- Parkinson’s medications can help reduce symptoms but can also cause increased confusion and hallucinations
- Usually good response to cholinesterase inhibitors

Parkinson's Disease Dementia (PDD)

- Dementia occurs in the setting of established parkinsonism
- "1 year rule"
  - parkinsonian motor features are present for more than 1 year before the onset of cognitive decline
- Symptoms similar to DLB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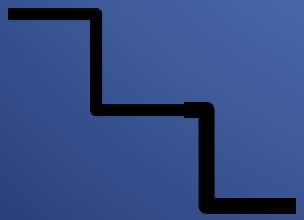
Vascular Dementia (VaD)

- Often overdiagnosed
- Not a single disease but a group of syndromes
  - Underlying cause is cerebrovascular disease in some form
  - Different pathophysiologic mechanisms
  - Variety of clinical manifestations
- Classified in many different ways

Vascular Dementia

Post stroke dementia due to strategic infarcts

- Often present with a clinical stroke/abrupt onset
- Deficits are specific to the areas affected
- Stepwise decline

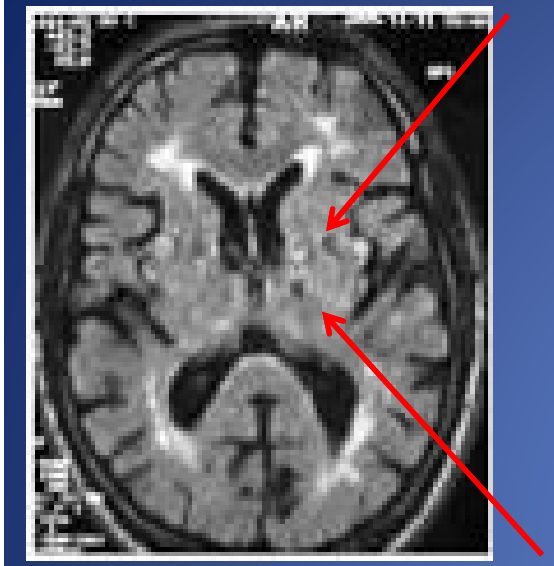


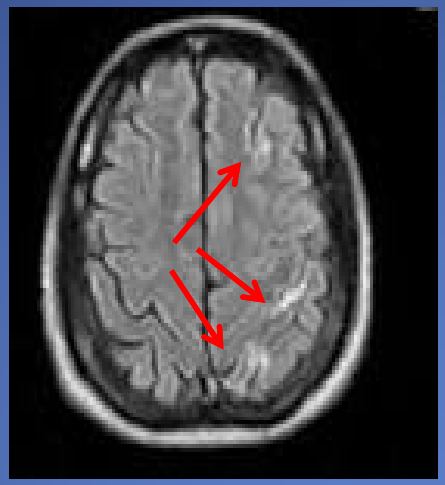
White Matter Disease and Lacunar Infarctions

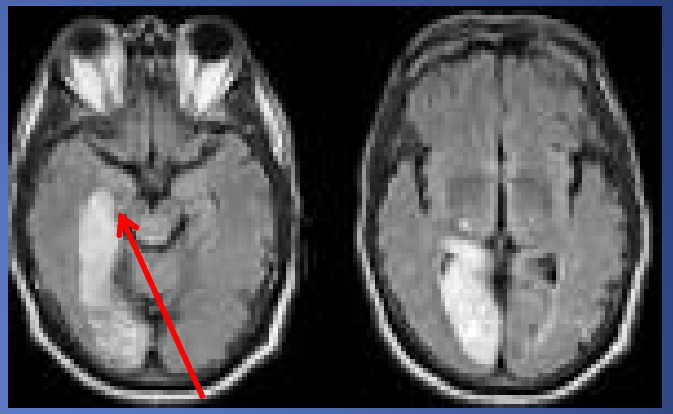
- Usually need “severe” “extensive” or “confluent” white matter changes to affect cognition
- Need “well placed” or multiple small lacunar strokes to affect cognition

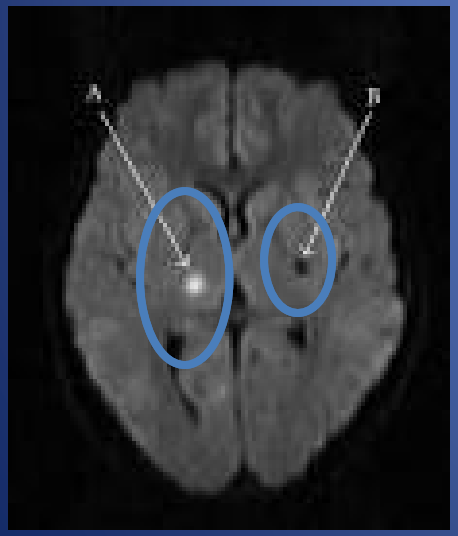
Cerebral Infarcts

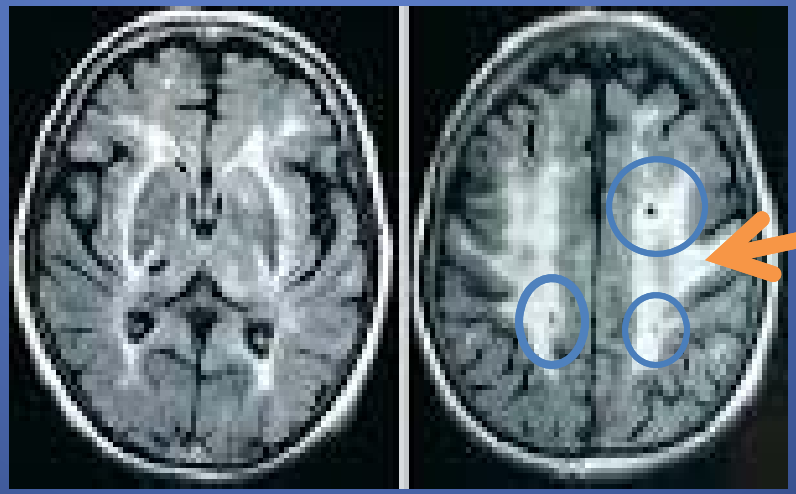
Different strokes (in different folks)





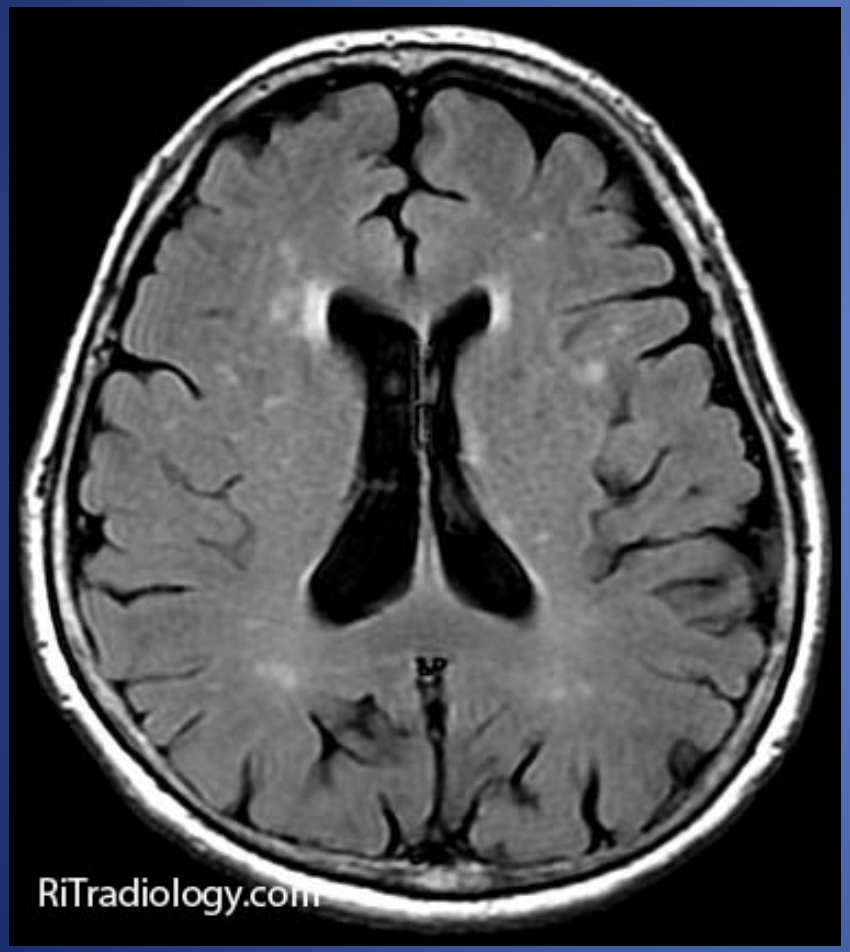


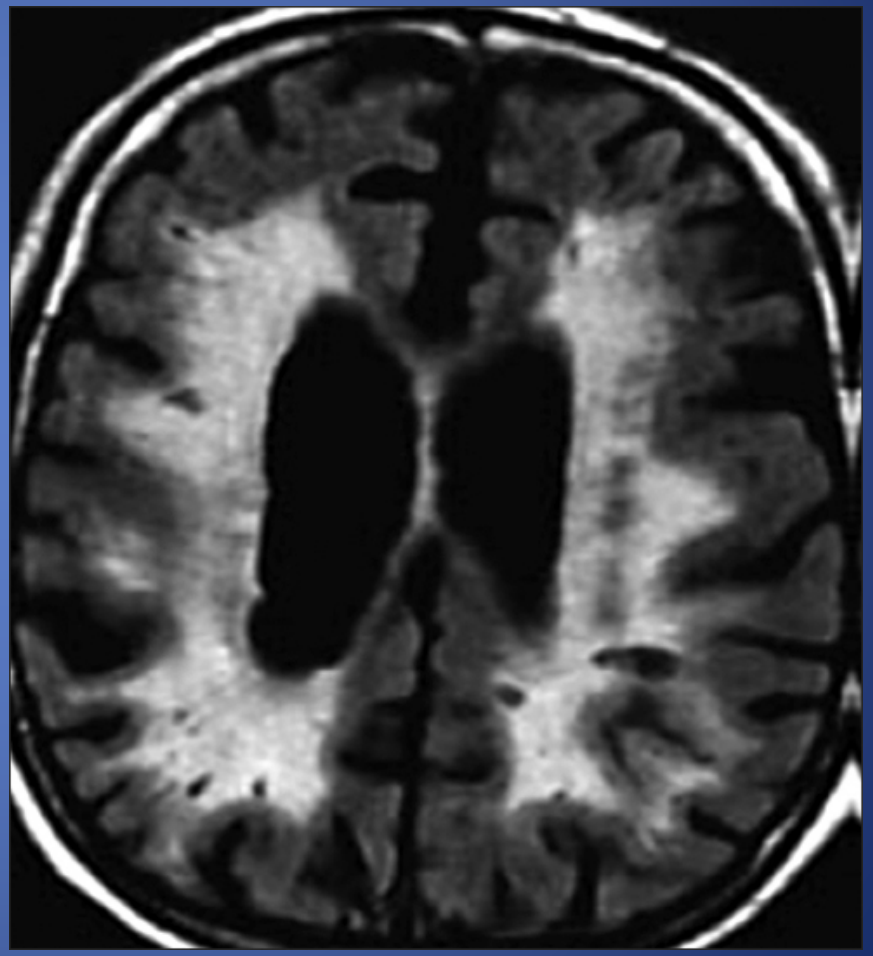




Extensive white matter change

White Matter Disease





Clinical Features Consistent with a diagnosis of VaD

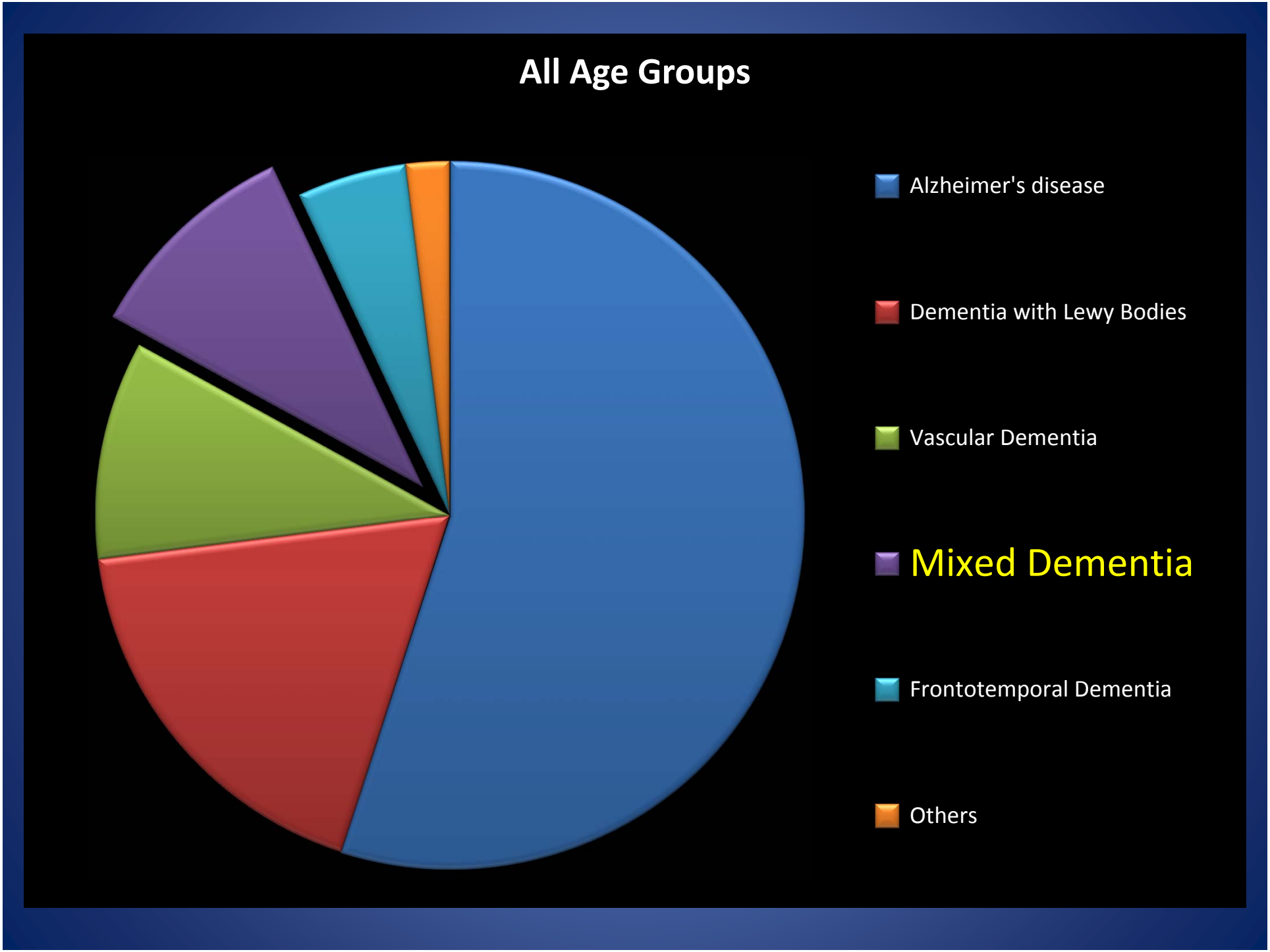
- Early gait disturbance
- Early urinary symptoms not explained by urologic disease
- Abnormal executive functioning (planning, sequencing, etc)
- Personality and mood changes
- Pseudobulbar palsy
- Psychomotor retardation

Hachinski's Ischemic Score

Abrupt onset	2
Stepwise deterioration	1
Fluctuating course	2
Nocturnal confusion	1
Relative preservation of personality	1
Depression	1
Somatic complaints	1
Emotional lability	1
Hypertension	1
History of stroke	2
Evidence of atherosclerosis	1
Focal symptoms	1
Focal signs	2

- Score ≤ 4 – AD

Score ≥ 7 - VaD


All Age Groups
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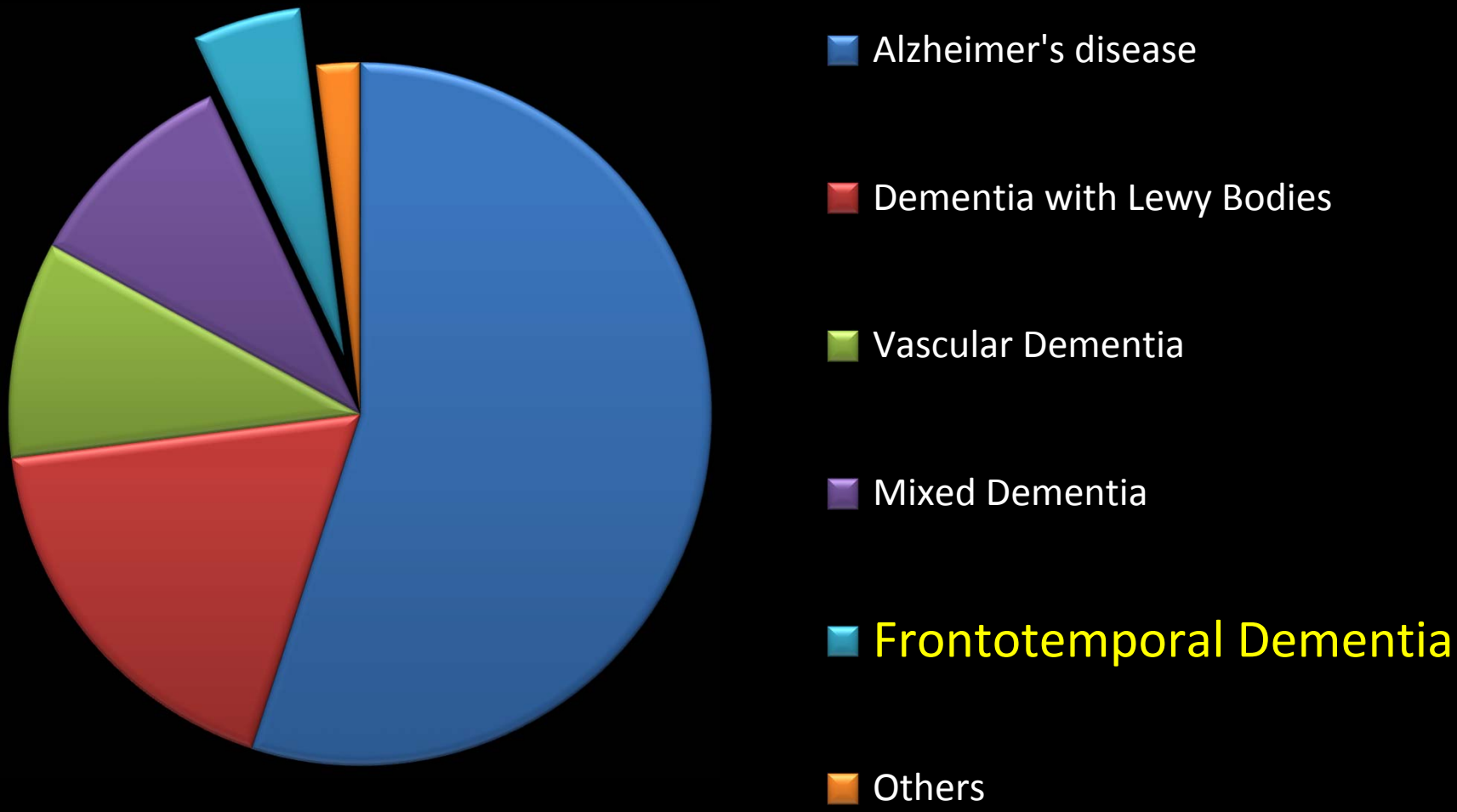
Others

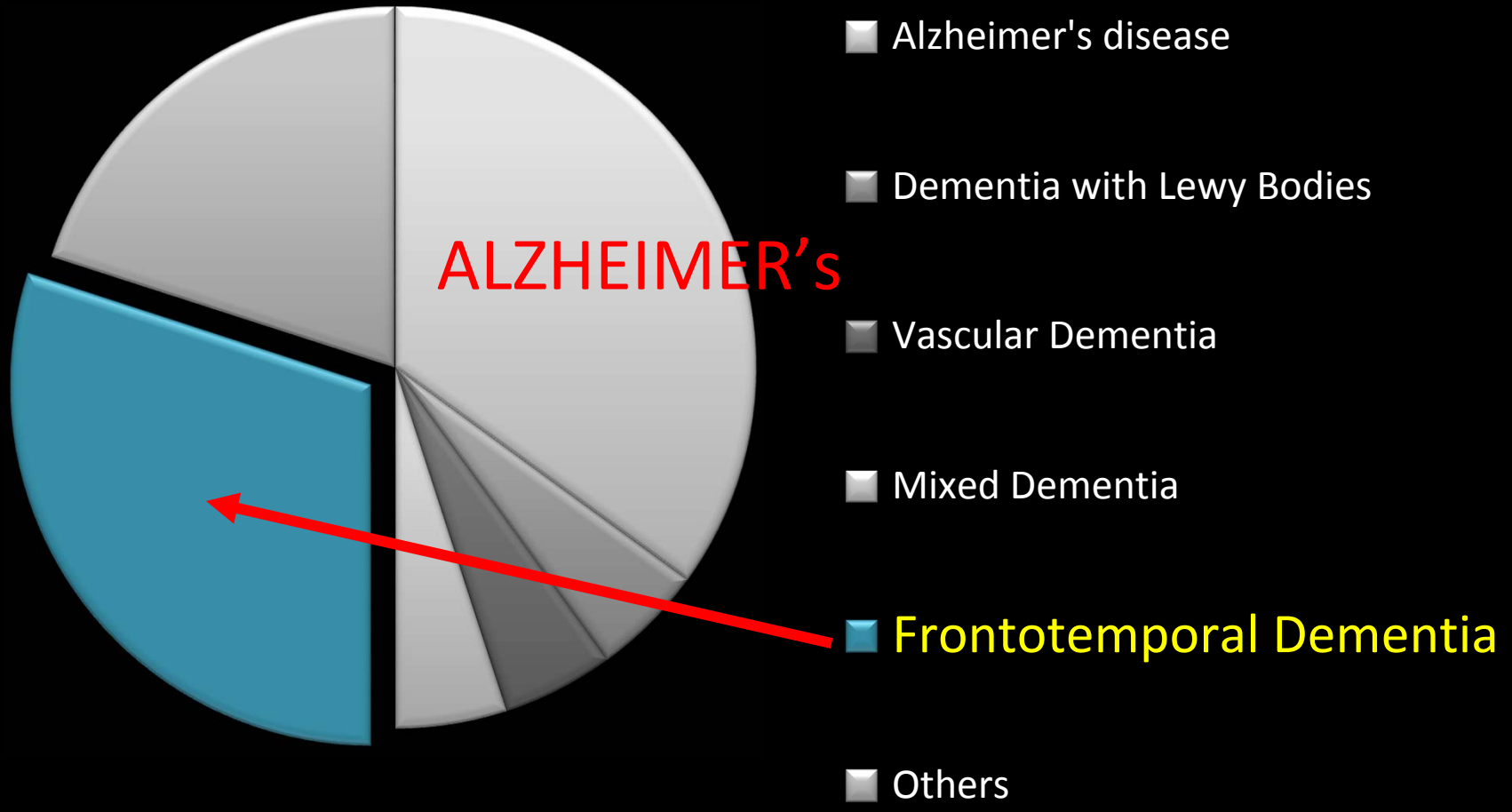
Mixed Dementia

- Refers to the co-occurence of 2 diseases (usually AD and VaD pathology)
- Can be difficult to distinguish which process is "more important"
- 1/3 of patients diagnosed with VaD will have AD pathology at autopsy (Alzheimer Dis Assoc Disord. 1999)

All Age Groups



Under Age 65


ALZHEIMER’s
Alzheimer's disease
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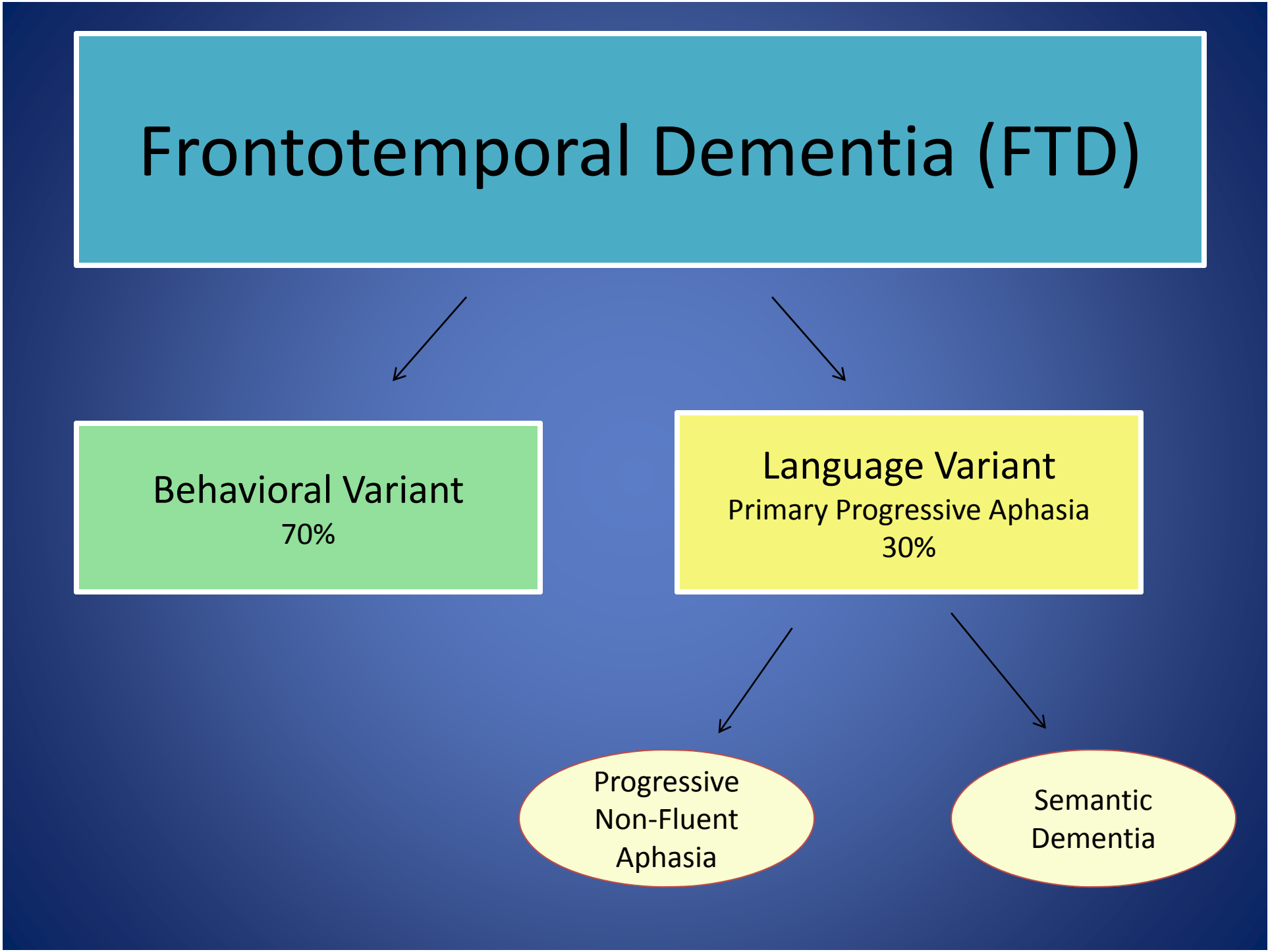
Others

Frontotemporal Dementia (FTD)

- Typical onset is < 65 years
- May be the most common cause of dementia in those younger than 65
- Behavior variant (bvFTD) used to be referred to as Pick's disease—Pick's only a subset
  - Specific inclusions called Pick bodies


Frontotemporal Dementia (FTD)
Behavioral Variant
70%
Language Variant
Primary Progressive Aphasia
30%
Progressive
Non-Fluent
Aphasia
Semantic
Dementia

Behavioral Variant FTD

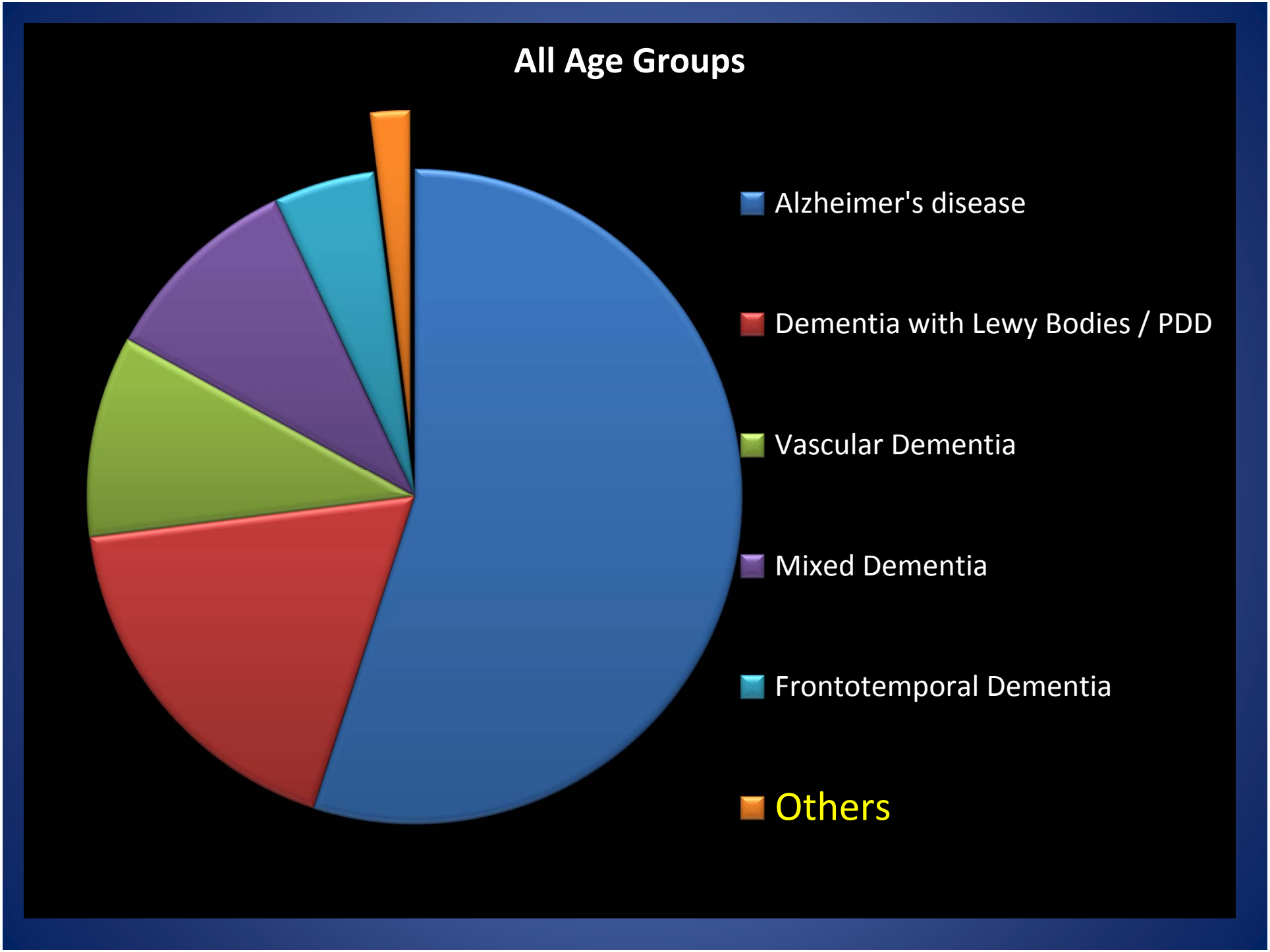
- Insidious onset and slow progression
- Personality change and disordered social conduct are the dominant features at onset
- Memory intact initially
- Lack of insight and empathy
- Decline in personal hygiene

Behavioral Variant FTD

- Mental rigidity and inflexibility
- Hyperorality
- Executive dysfunction OR
- Disinhibition
  - Antisocial and Compulsive behaviors
  - Hoarding
  - Food compulsions

FTD - Language Variant
Primary Progressive Aphasia

- Progressive non-fluent aphasia
  - Expressive aphasia, word finding difficulty
  - Understand but cannot speak
  - Loss of grammar, speech apraxia
- Semantic Dementia
  - Speak fluently but cannot understand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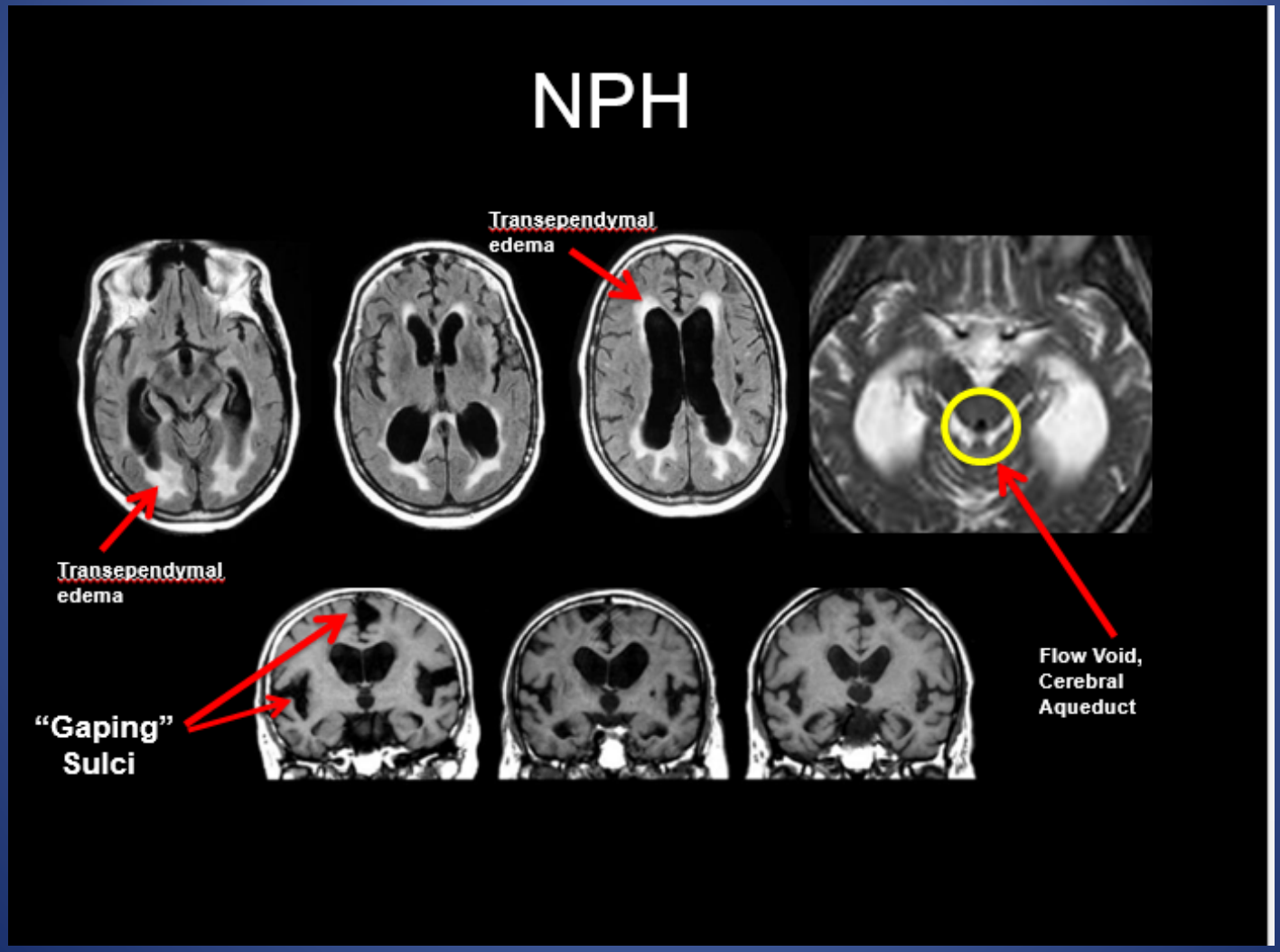
“Other” Dementias

- Reversible causes
- Cortical Basal Degeneration
- Progressive Supranuclear Palsy
- **Normal Pressure Hydrocephalus**
- Chronic Traumatic Encephalopathy
- Creutzfeldt-Jacob disease
- Huntington’s disease
- Alcohol
- HIV
- Anoxia
- ETC

NPH:

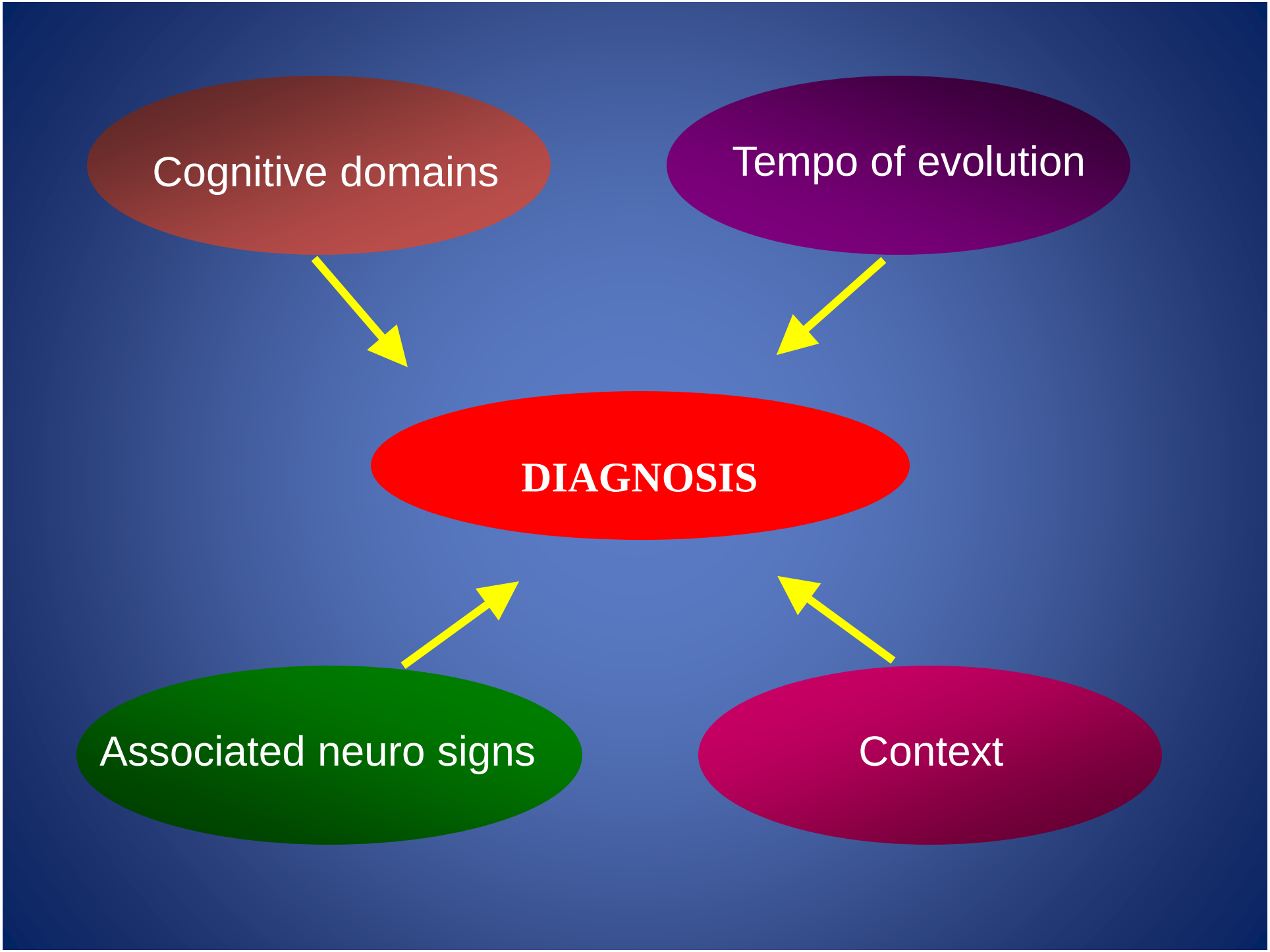
Triad of Gait disturbance, urinary incontinence and mild dementia (attention, working memory, exec function impairments, with good memory)



Disease	Age (y) at diagnosis	Progression	Earlier cognitive symptoms	Visual hallucinations	Parkinsonism	REM sleep behavior disorder	Autonomic insufficiency	Dominant presenting symptoms
Alzheimer dementia	Late (> 65) Early (< 65)	Gradual	Early impairment of memory and attention	Rare	Late stages	Rare	Rare	Memory loss, cognitive impairment
Vascular dementia	≥ 60	Sudden, stepwise	Executive dysfunction, deficits depend on location of stroke or lesion	Rare	Depends upon location of stroke	None	None	Sudden onset of cognitive deficits and impairment
Dementia with Lewy bodies	70s [6]	Gradual with fluctuation in cognition	Early Impairment of visual spatial skills and attention Delayed recall is relatively preserved in the beginning	Typical	Within first year	Common	Occasional	Parkinsonism or cognitive impairment
Progressive supranuclear palsy	60s [8]	Gradual	Frontal behavioral disturbance, deficit in verbal fluency or abstract thoughts	Rare	Symmetric,[9] (1/3 initially asymmetric)	Infrequent[7,8]	Common	Motor symptoms, balance problems, falls
Corticobasal degeneration	Around 60	Gradual	Deficit in frontal-parietal cognitive domains, including attention, concentration, executive function, verbal fluency	Rare	Asymmetric[9]	Rare[10]	Rare	Motor symptoms
Multiple system atrophy	≥ 60	Gradual	Late dementia, with deficits in learning, recognition, memory, and verbal fluency	Rare	Symmetric	Common	Common	Autonomic failure, motor symptoms
Parkinson disease dementia	70s[6]	Gradual	Impairment in attention, memory, executive and visuospatial functions	Occasional at late stage	Asymmetric at onset	Common[7]	Common	Motor symptoms
Frontotemporal dementia	Mostly < 65	Gradual	Difficulty with language and executive function or behavioral change	Rare	Sometimes	Occasional	Infrequent	Behavioral changes
Primary progressive aphasia	Around 60	Gradual	Expressive language impairment	Rare	In late stages	None	None	Expressive language impairment
Normal-pressure hydrocephalus	50s–60s	Gradual	Impairment of attention, working memory, verbal fluency and executive function; recognition memory is preserved	Rare	May present as parkinsonism	None	None	Gait impairment with urinary frequency and/or cognitive impairment

Evaluation of Cognitive Impairment

- Detailed history
  - Should have informant
  - Social and Family histories are important
- Thorough physical examination
- Laboratory testing
- Cognitive Testing
- Imaging

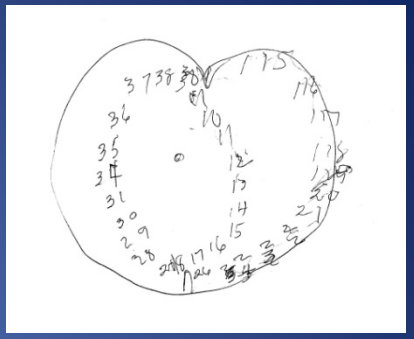

Cognitive domains
Tempo of evolution
DIAGNOSIS
Associated neuro signs
Context

Laboratory Evaluation

Labwork to rule out reversible causes

- CBC (Complete Blood Count)
- CMP (Comprehensive Metabolic Panel)
- TSH (Thyroid)
- Vitamin B12
- ESR (sedimentation rate), in some cases
- RPR (not usually done, only if increased risk)

Cognitive Testing



- MMSE
  - Scores influenced by age and education
- Montreal Cognitive Assessment (MoCA)
- Clock drawing
- Neuropsychiatric evaluation
  - 3-5 hours (simple vs. complex)
  - Provides a baseline or assist with making a diagnosis or relative strengths vs. weaknesses helps understand how to compensate

Evaluation - Imaging

Structural Imaging

- MRI or CT scan
  - Evaluate for structural abnormalities (strokes, tumors, hydrocephalus)
  - Assess for atrophy (global, focal)

Functional Imaging

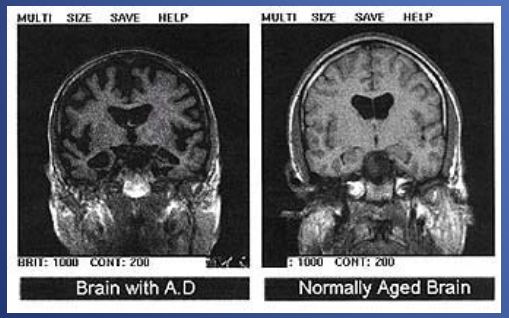
- FDG – PET scan
  - Assess for decreased glucose uptake
  - Look for patterns
- DaT scan
  - Can assist with a diagnosis of DLB /PDD

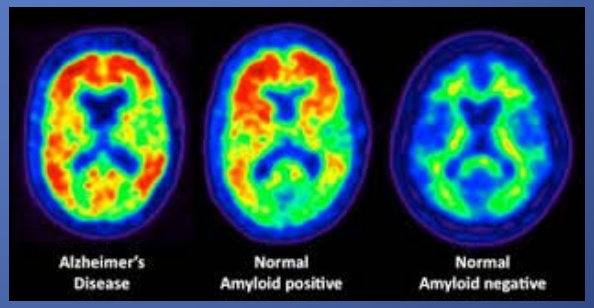
Molecular Imaging

- Amyloid imaging – used primarily in research protocols

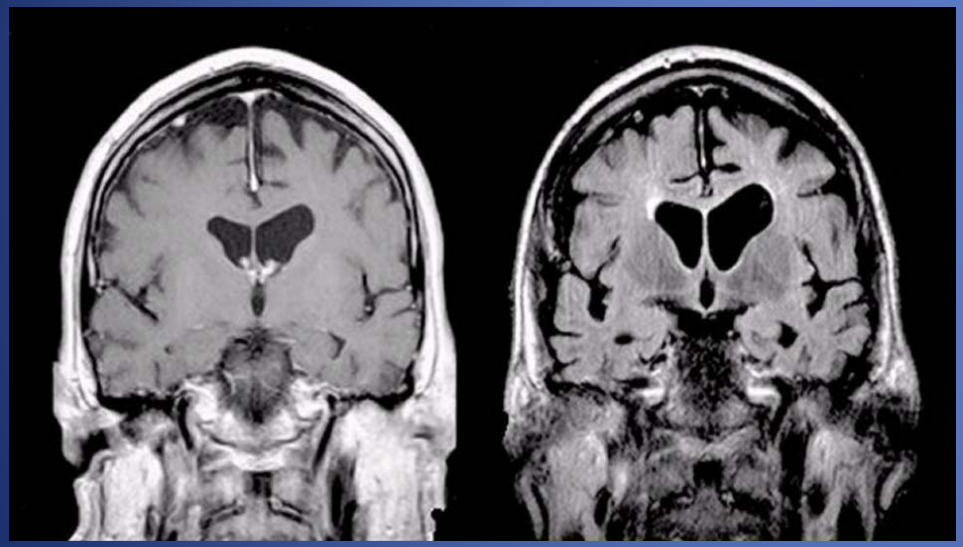
Alzheimer's Disease

- **MRI – normal or global atrophy, medial temporal lobe (hippocampal) atrophy**
  - **Main purpose is exclude a reversible cause, eg, tumor, stroke, bleeding, etc.**
- FDG PET – hypometabolism in parietal lobes
- Amyloid scan – elevated amyloid levels

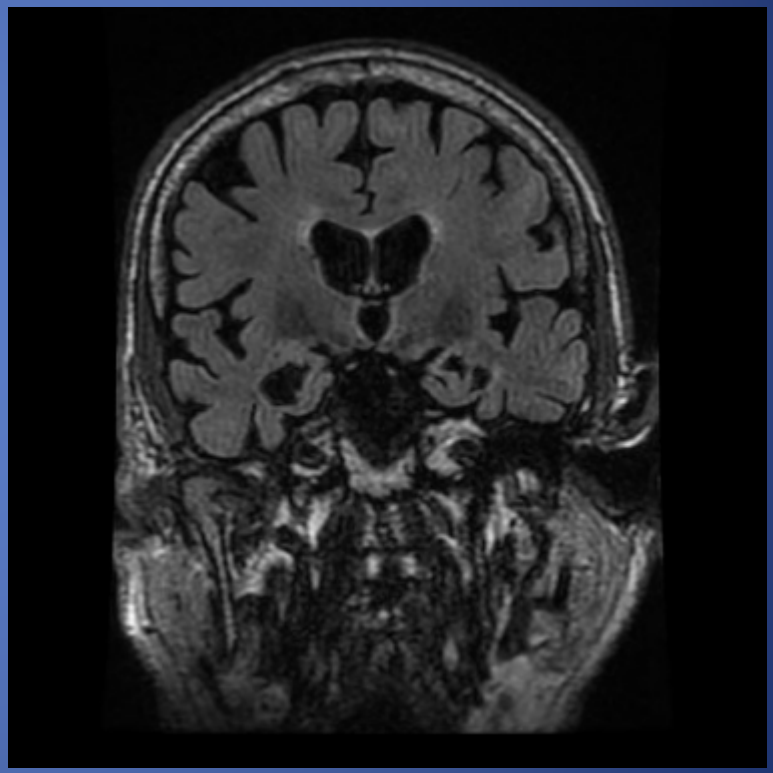




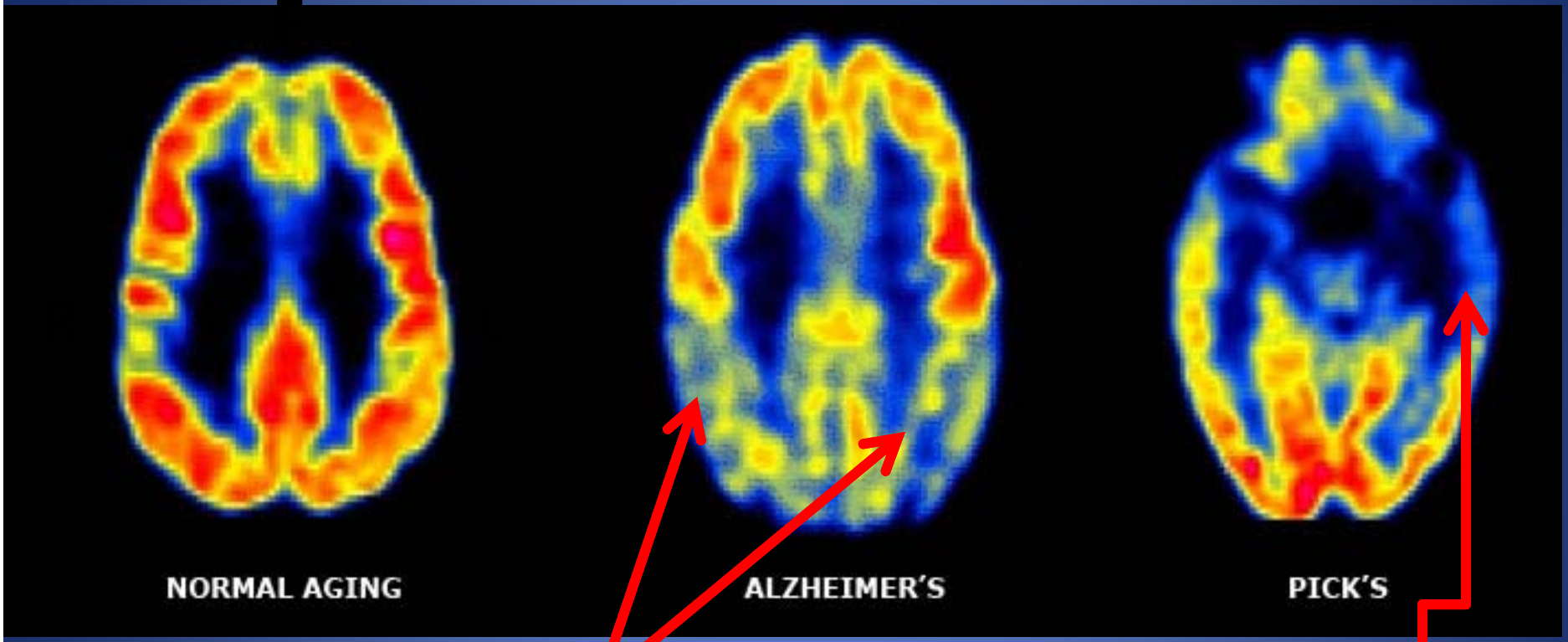
MCI due to Alzheimer's



Two years prior

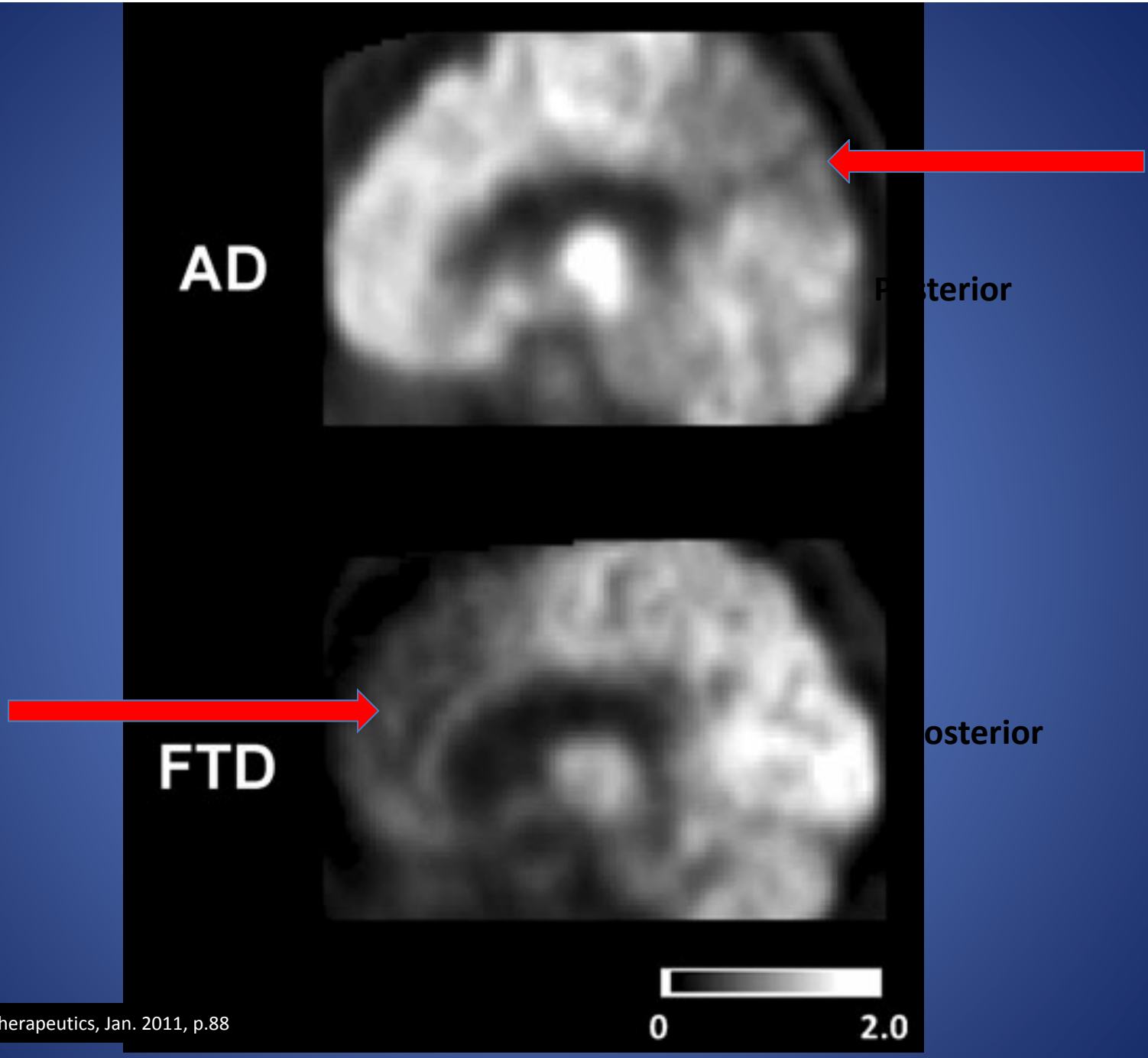


At time of Dx,
2 yrs. later



Lower Metabolism of glucose

FTD



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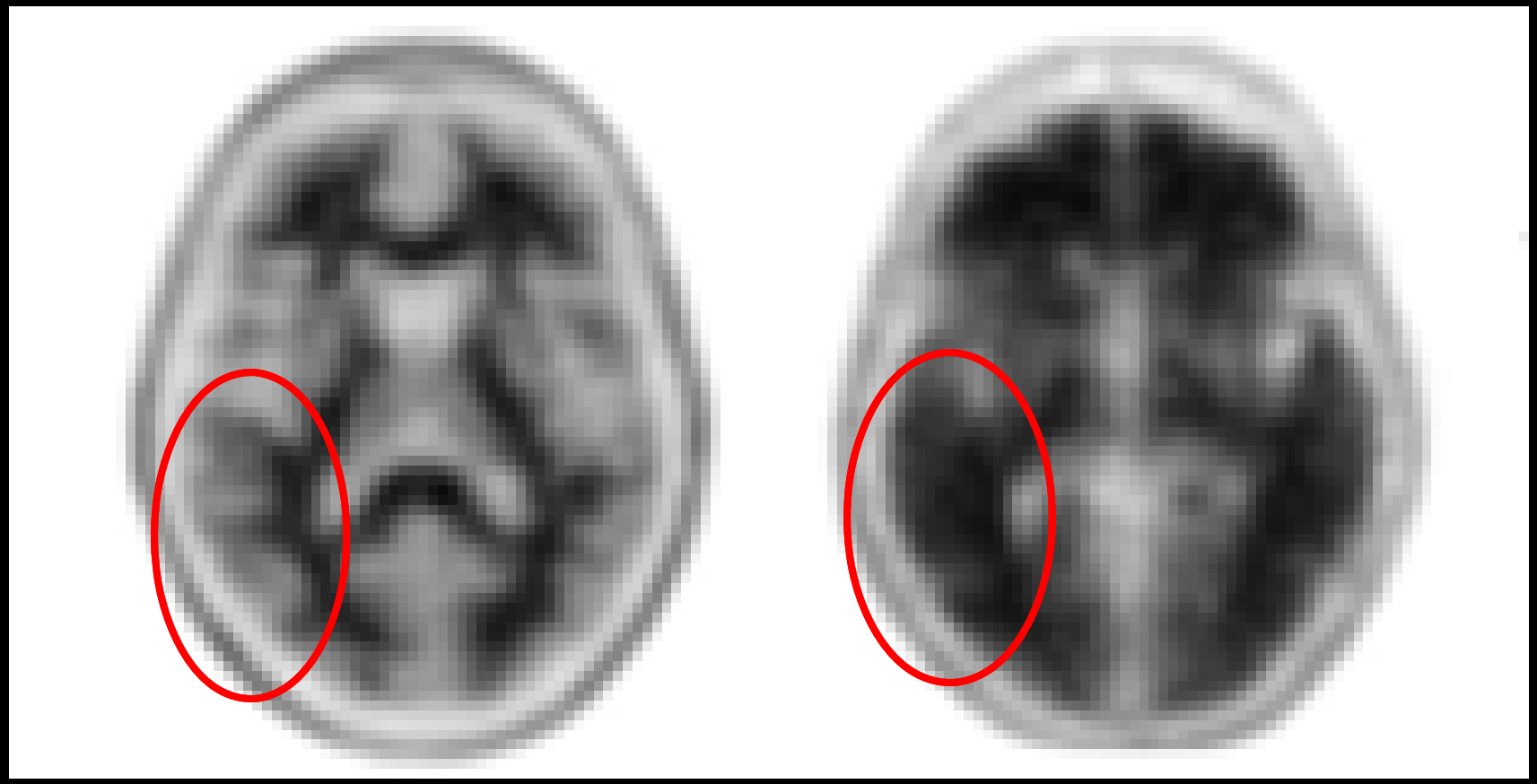
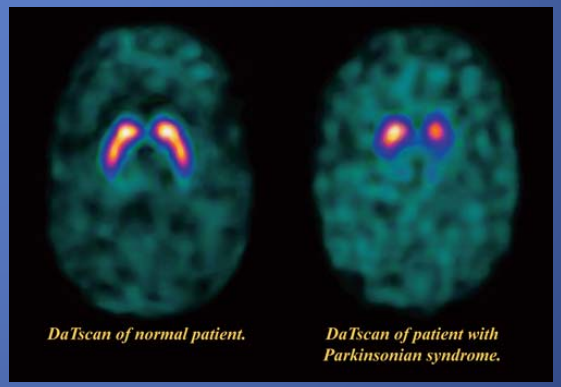


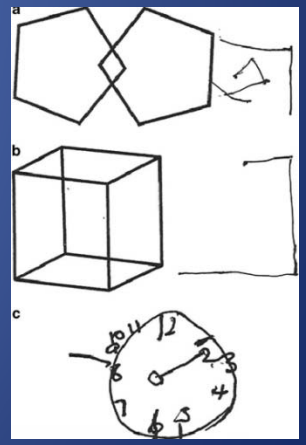
NORMAL GREY-
WHITE MATTER
DIFFERENTIATION

Amyloid in Cortex

DLB/PDD

- History : hallucinations, fluctuations, REM sleep behavior disturbance, motor changes
- Physical Exam: Parkinsonism
- Cognitive Evaluations: visualspatial difficulties,
  - Memory spared initially
- Imaging
  - MRI - normal or atrophy
  - DaT scan – abnormal
  - FDG PET - hypometabolism in occipital areas





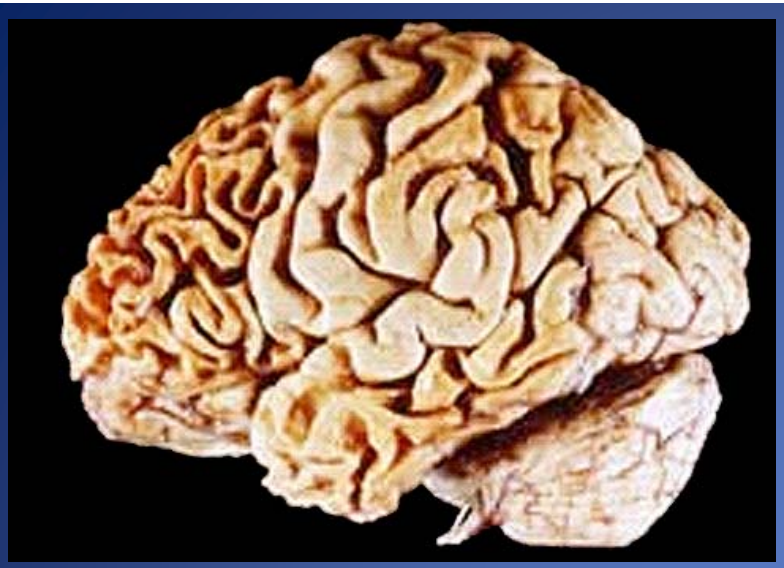
Vascular Dementia

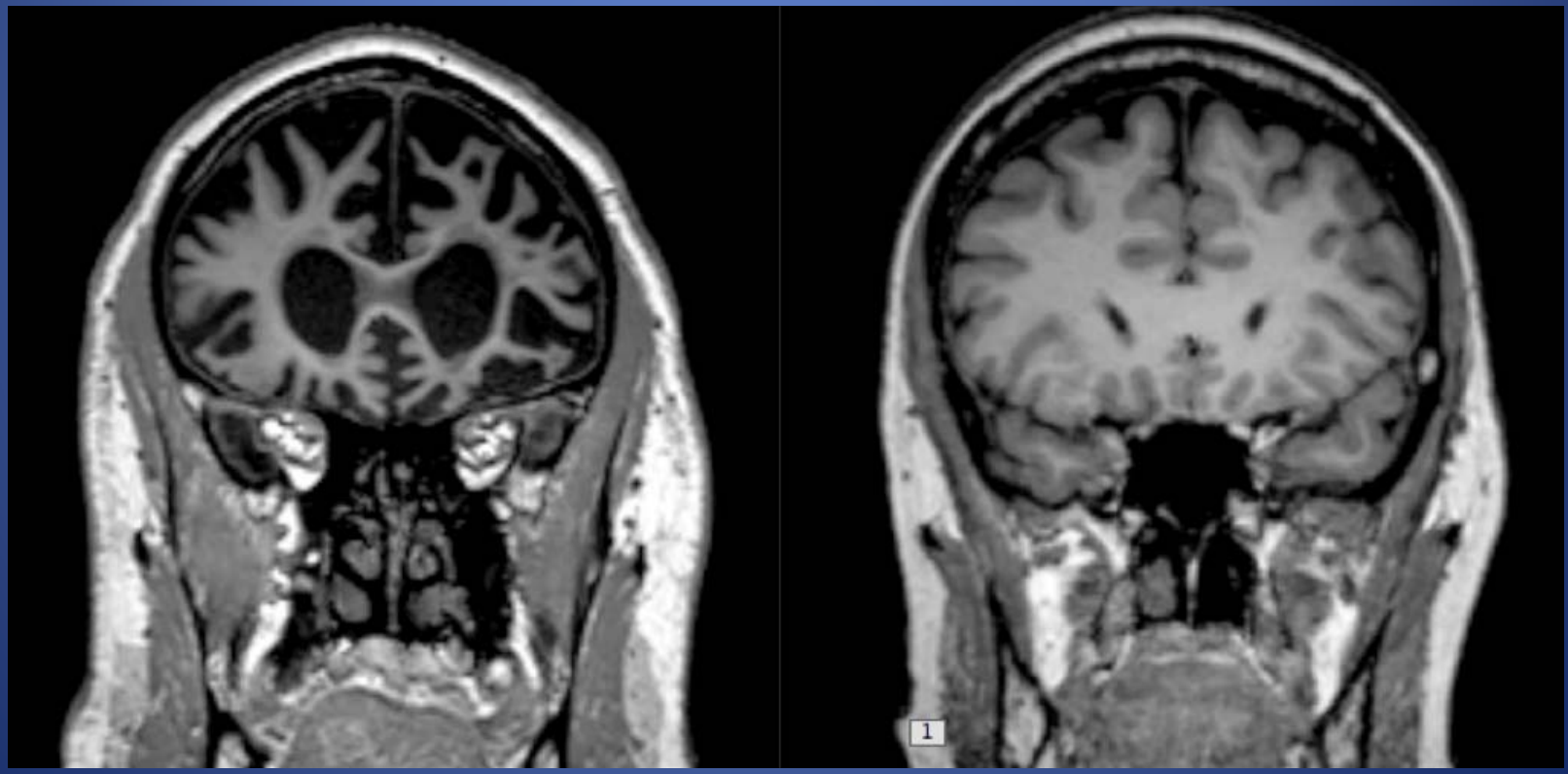
- History: strokes or gait changes, early urinary symptoms
- Physical Exam: consistent with a stroke or gait changes, pseudobulbar palsy, psychomotor slowing
- Cognitive Evaluations: Executive dysfunction
- Imaging:
  - MRI/CT: Evidence of CVA or severe or extensive white matter changes

FTD

- History: Personality/behavior changes or significant language issues
- Physical Exam: fairly normal, language issues
- Cognitive Evaluations:
  - Memory intact initially
  - Executive dysfunction or language impairment
- Imaging
  - MRI/CT scan – normal or frontotemporal atrophy
  - FDG PET – hypometabolism in frontal and/or temporal lobes

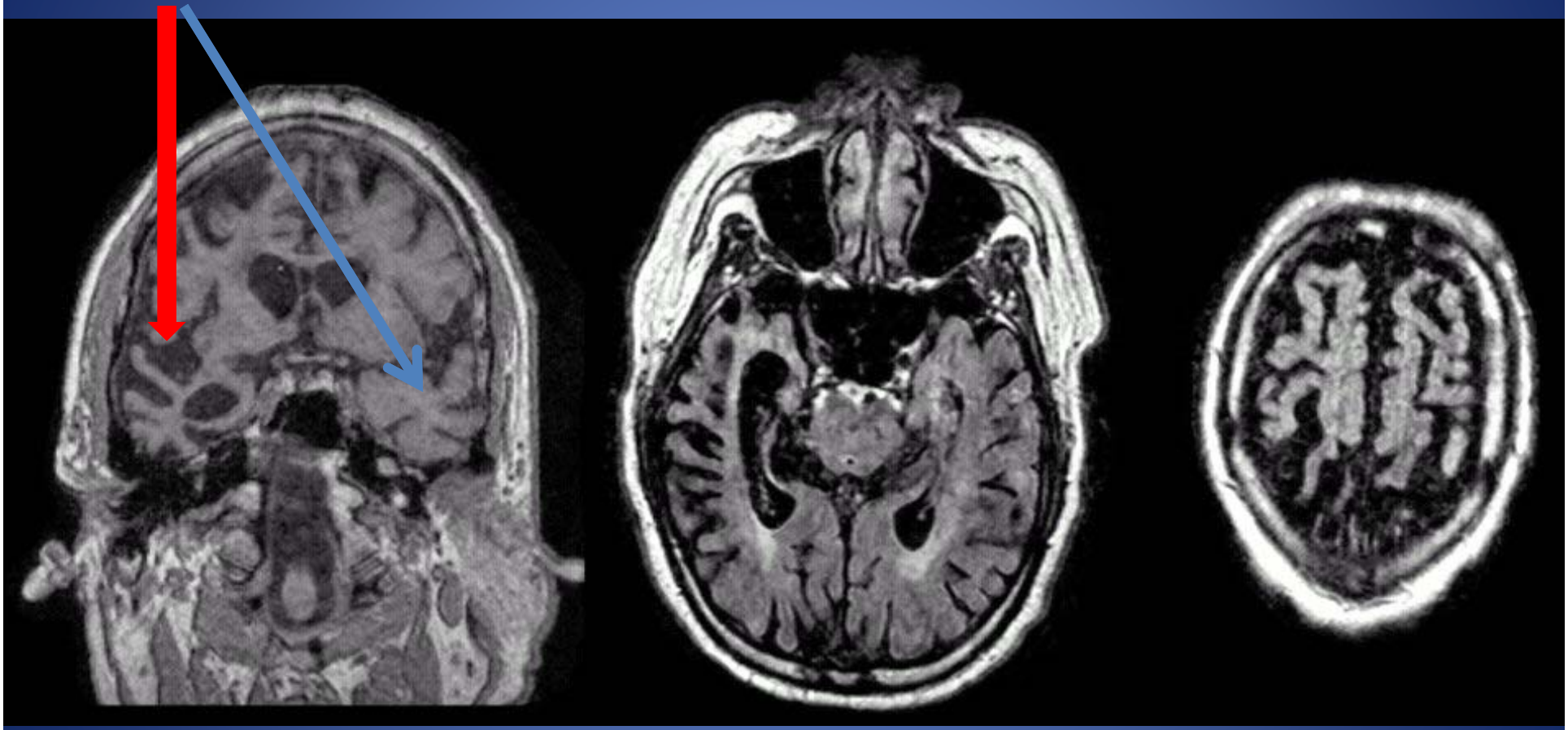
FTD

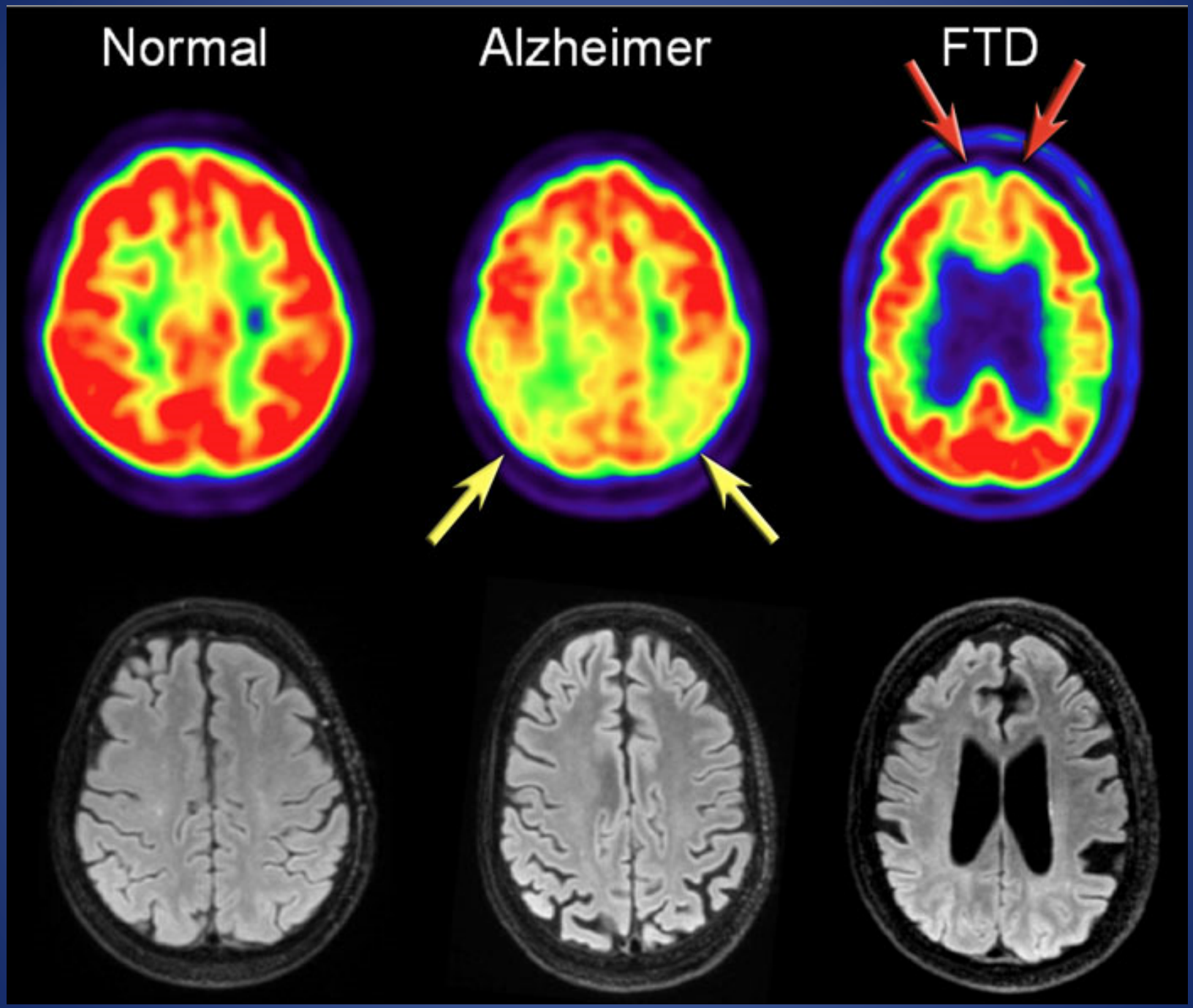




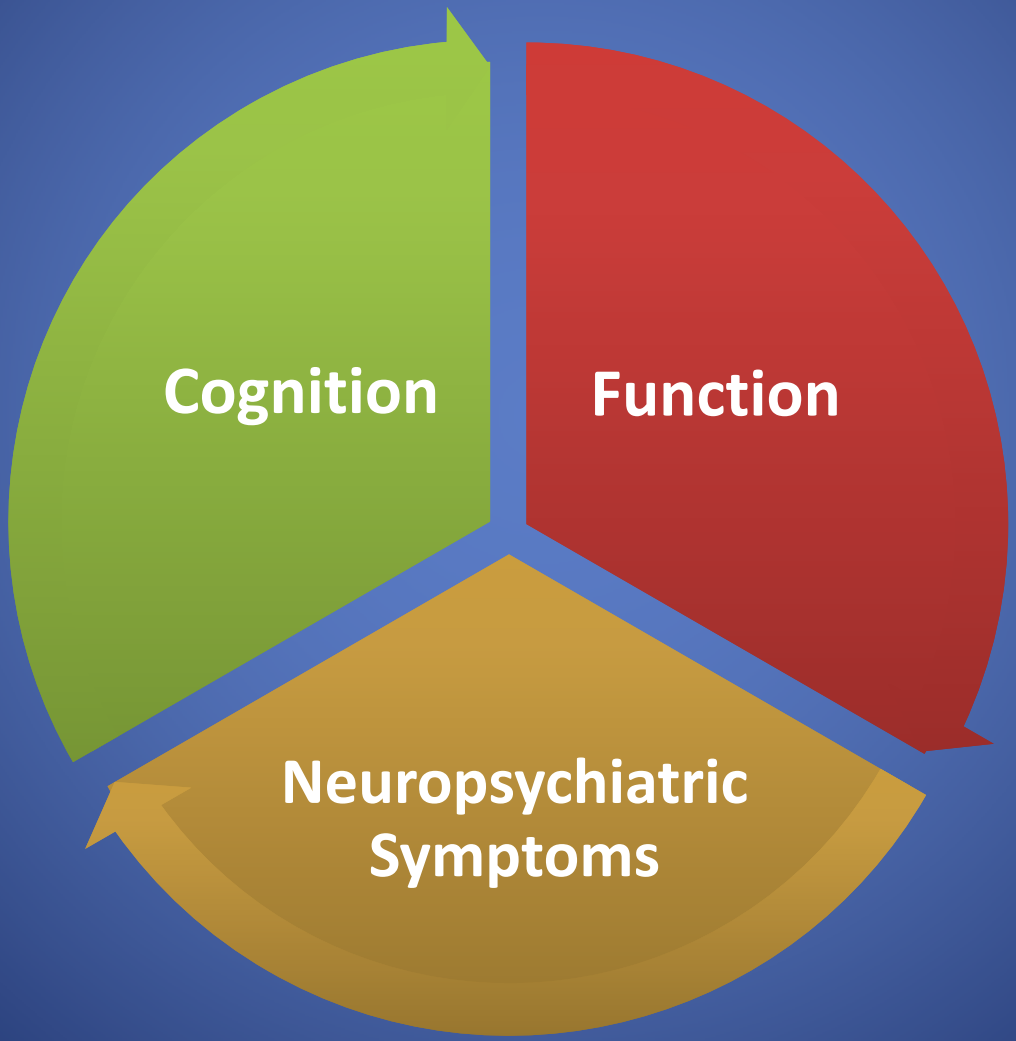
Occasionally can diagnose a degenerative dementia

FTD




Normal
Alzheimer
FTD

Dementia - Treatment



Treatments - Medications

Acetylcholinesterase Inhibitors

Donepezil (Aricept™)

Rivastigmine (Exelon™)

Galantamine (Razadyne™)

NMDA Antagonist

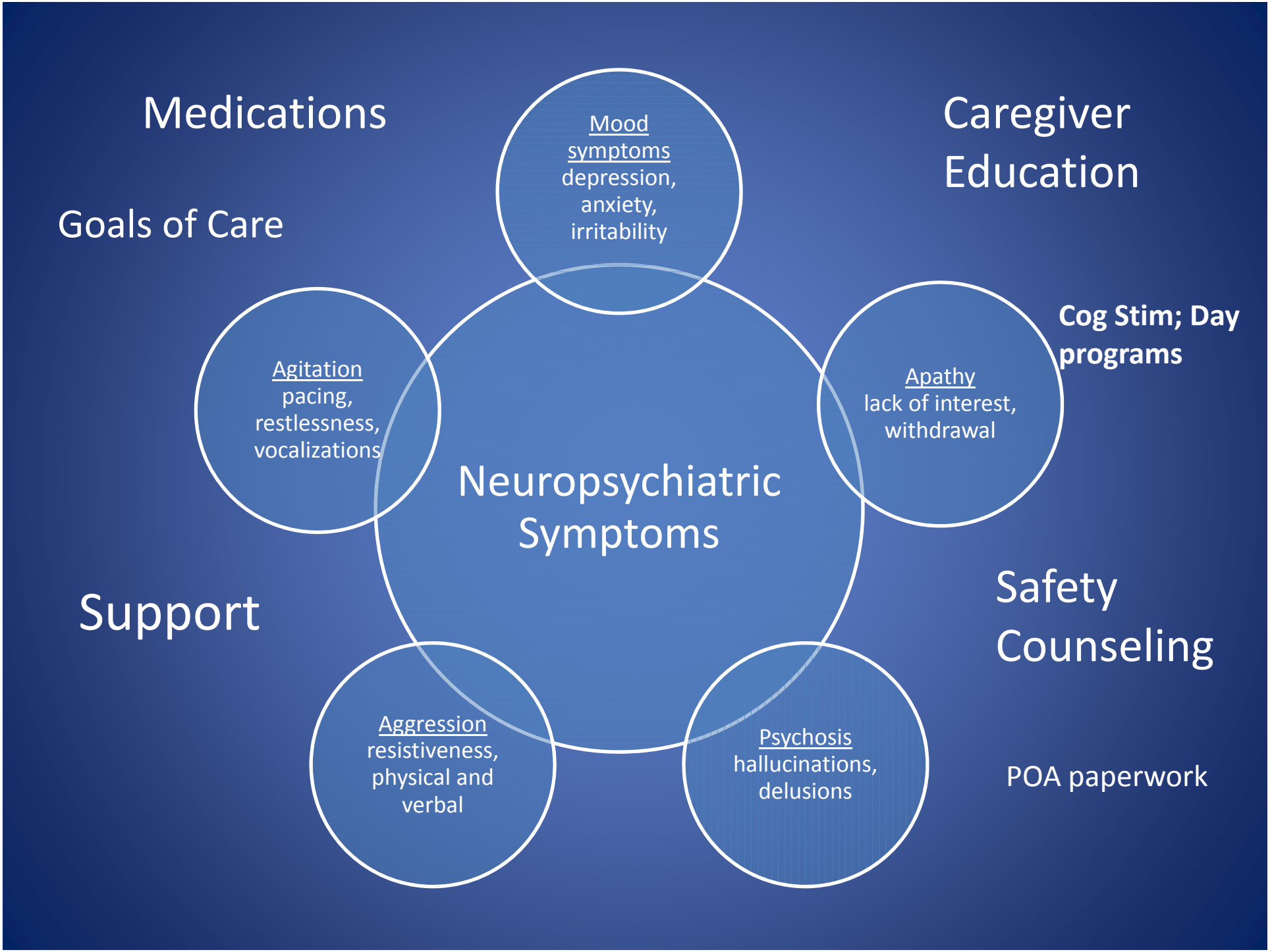
Memantine (Namenda™)

Cholinesterase Inhibitors

- Mild to advanced AD
  - also used for vascular, DLB, PDD
- Modest benefits
  - Cognition
  - Activities of daily living
  - Behavioral symptoms
- Does not stop or reverse disease
- GI side effects, vivid dreams, bradycardia
- Take with food in the morning

memantine

- NMDA antagonist
- Moderate to severe AD (little help in mild disease)
- Modest benefits
  - Cognition
  - Activities of daily living
  - Behavior
- Immediate release (twice daily) and XR (once daily) forms
  - IR generic in summer of 2015
- Dose needs to be slowly titrated for the first month
- Well tolerated
  - Headache, dizziness, confusion


Medications
Goals of Care
Support
Caregiver Education
Cog Stim; Day programs
Safety Counseling
POA paperwork
Mood symptoms depression, anxiety, irritability
Agitation pacing, restlessness, vocalizations
Apathy lack of interest, withdrawal
Aggression resistiveness, physical and verbal
Psychosis hallucinations, delusions
Neuropsychiatric Symptoms

Key Points

1. Delirium is an ***acute, fluctuating*** alteration in mental status characterized by altered level of *consciousness*, fluctuating *attention*, and globally clouded ***cognition***.
2. Perceptual disturbances, eg, illusion, hallucinations
3. Constructional disturbances, dysgraphia
4. Agitated (hyperactive) vs. hypoactive (quiet) vs. both
5. Altered sleep-wake cycle

Delirium Evaluation

- Primary problem is usually a ***medical*** condition
  - Toxic-metabolic encephalopathy
  - Infection
- ICU or post-op psychosis
  - risk factor for dementia months later
- Decompensated dementia; dementia is NOT the cause, but predisposes to more obvious symptoms/signs

Risk Factors

- MCI, Dementia
- Immobility, dependence on others
- Sensory impairment
  - visual, auditory
- Dehydration, malnutrition
- Drugs
  - Psychoactive ones
  - Polypharmacy

Preventing Delirium

1. Orientation & therapeutic activities
2. Early mobilization
3. Minimize psychoactive drugs
    a. Orientating stimuli
    b. Normal sleep-wake cycles
4. Adaptive equipment
    a. Glasses, hearing aids
5. Early intervention for volume depletion

Preventing Delirium*

- In addition to the above…

6. Optimize oxygen delivery to brain
7. Monitor fluid & electrolyte balance
8. Pain management
9. *Attend to bowel & bladder function*

*Marcantonio et al. J Am Geriatr Soc 2001;49:516-22

Treating Delirium

- Acute stabilization
  - Airway
  - Hydration/volume status
  - Close nursing supervision (ICU?)
  - Positioning (prevent decubiti)
  - DVT prophylaxis

Treating Delirium

- Environmental cues
  - Calendars, clocks, familiar home objects
  - Reorienting by staff
- Limit staff & room changes
- Allow for uninterrupted sleep at night
  - Coordinate VS measures, meds, etc.
- Low noise, low light at night
- Up and about during the day

Table 4. Pharmacologic Treatment of Delirium.

Class and Drug	Dose	Adverse Effects	Comments
Antipsychotic			
Haloperidol	0.5–1.0 mg twice daily orally, with additional doses every 4 hr as needed (peak effect, 4–6 hr) 0.5–1.0 mg intramuscularly; observe after 30–60 min and repeat if needed (peak effect, 20–40 min)	Extrapyramidal symptoms, especially if dose is >3 mg per day Prolonged corrected QT interval on electrocardiogram Avoid in patients with withdrawal syndrome, hepatic insufficiency, neuroleptic malignant syndrome	Usually agent of choice Effectiveness demonstrated in randomized, controlled trials[20,37] Avoid intravenous use because of short duration of action
Atypical antipsychotic			
Risperidone Olanzapine Quetiapine	0.5 mg twice daily 2.5–5.0 mg once daily 25 mg twice daily	Extrapyramidal effects equivalent to or slightly less than those with haloperidol Prolonged corrected QT interval on electrocardiogram	Tested only in small uncontrolled studies Associated with increased mortality rate among older patients with dementia
Benzodiazepine			
Lorazepam	0.5–1.0 mg orally, with additional doses every 4 hr as needed*	Paradoxical excitation, respiratory depression, oversedation	Second-line agent Associated with prolongation and worsening of delirium symptoms demonstrated in clinical trial[37] Reserve for use in patients undergoing sedative and alcohol withdrawal, ~~those with Parkinson's~~ disease, and those with neuroleptic malignant syndrome
Antidepressant			
Trazodone	25–150 mg orally at bedtime	Oversedation	Tested only in uncontrolled studies

X

?

Statistically significant, clinical significance unclear

* Intravenous use of lorazepam should be reserved for emergencies.

Epidemiology

- 20-50% ***older*** patients (esp. postoperatively)
- 70-90% of patients in ***ICU***
- 80% of patients at ***end of life***
- Community prevalence: < 1-2%

- Mortality 25-75% (***comparable to MI or sepsis***)
- One-year mortality: 35-40%

- Up to 20% of 12.5 M patients > 65 years
- Cost ≈ $2,500/pt ($6.9 billion) per year

DHHS 2004 CMS Statistics (pub. #03445)
Inouye, Clin Geriatr Med 199;14:745-764
Pisani et al, Clin Chest Med 2003;24:727-737
Casarett & Inouye, Ann Intern Med 2001;135:32-40

Key Points

1. Delirium is an ***acute, fluctuating*** alteration in mental status characterized by altered level of *consciousness*, fluctuating *attention*, and globally clouded ***cognition***.
2. Risk increases with age, presence of dementia, polypharmacy, severity of illness, and metabolic derangement.
3. Delirium increases ***risk of death***.
4. Delirium increases is costly and increases length of hospital stay.
5. The best treatment for delirium is ***prevention***, and directed interventions can prevent delirium.
6. Other treatments exist, but prevention is key.

Statistics

- An estimated 5.2 million Americans have Alzheimer's disease in 2014
- 6th leading cause of death (more than breast and prostate cancer combined) and the fifth leading cause for people aged 65 years and over
- By 2050, the number of people with Alzheimer's disease in the US may nearly triple, from 5 million to as many as 16 million
- In 2010 the estimated global societal economic cost of dementia was $ 604 billion per year (World Health Organization)

Alzheimer's Association 2015