

PHYSICIAN AND CONSUMER EDUCATION IN EARLY DETECTION, DIAGNOSIS, AND TREATMENT

# 2021 ALZHEIMER'S DISEASE & RELATED DISORDERS VIRTUAL FORUM

04 NOVEMBER 2021



# DETECTION, DIAGNOSIS, AND TREATMENT OF ALZHEIMER'S DISEASE AND RELATED DISORDERS



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**GEISINGER HEALTH**

04 NOVEMBER 2021



**pennsylvania**  
DEPARTMENT OF AGING



# Detection, Diagnosis, & Treatment of Alzheimer's Disease and Related Disorders

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- Board Member, Pennsylvania Neurological Society

# Disclosure

Disclosure – I have no conflicts to disclose

There will be some mention of off label use for some medications as there are some things in dementia that have no FDA approved treatments

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# Learning Objectives

## DETECTION, DIAGNOSIS, & TREATMENT OF ALZHEIMER'S DISEASE AND RELATED DISORDERS

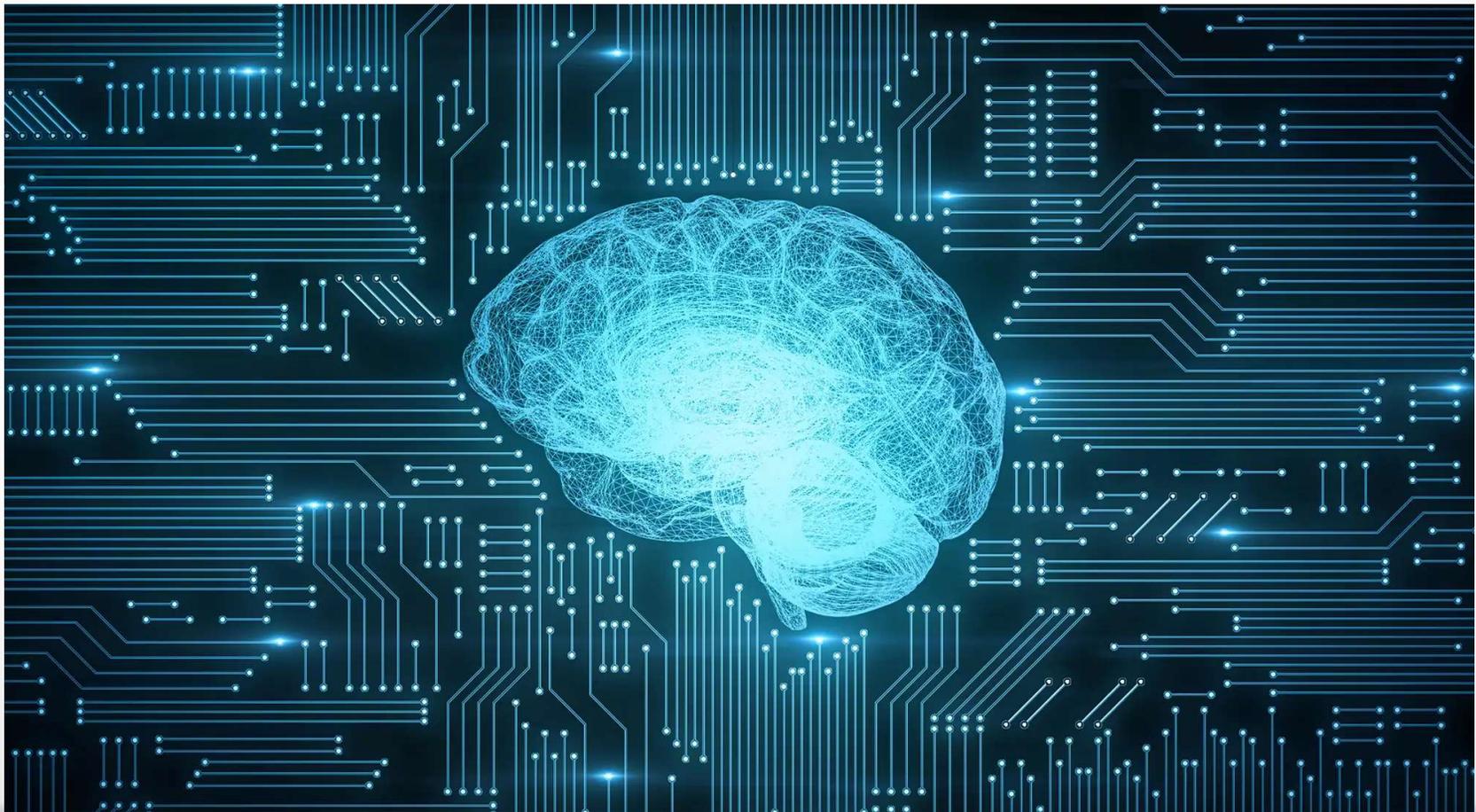
**How to better provide prevention advice, regularly screen for dementia risk, and get to accurate diagnosis. And once a diagnosis of dementia is present, how to manage to optimize quality of life and safety.**

**There are incredible demands on time in everyday clinical practice and dementias are both insidious and challenging to address when the disease itself often takes away the clinician's best ally. Most practices are not structured to be able to handle the detailed and time-consuming evaluation and counseling needed to provide best practice care. There is also in the community a lot of fatalism and lack of knowledge regarding dementias and related disorders that makes it harder to address.**

**Regularly educate on brain health techniques to patients, regularly screen for decline in memory and cognition at its earlier presentation, aggressively investigate and correct possible reversible causes and contributors, and empower providers, patients, and families to keep things as safe as possible in a debilitating disease.**

# Vision of the Memory and Cognition Program

Develop and provide leading edge  
comprehensive collaborative cognitive care



# Mission of Memory and Cognition Program

Develop and promote best practices in cognitive care throughout the time course of neurocognitive disorders.

- Brain Health and Prevention
- Screening and Early Detection
- Evaluation and Diagnosis
- Careplanning and Counseling
- Treatment and Management
- Longitudinal Monitoring
- End of Life

Guide and participate in clinical and quality research, new clinical capacities, and professional education

- Research collaborations and independent research
- Quality improvement
- Innovating new clinical care
- Educating
  - students
  - residents
  - fellows
  - post-docs
  - Providers (non-cognitive)
  - Providers (cognitive)

**Beware  
Brain  
Impairing  
Medications!  
Classes of  
Brain  
Impairing  
Medications**

Anti-Cholinergics

Anti-Dopaminergics

Strong Anti-Histamines

Hypnotics

Sedatives

Narcotics & Tramadol

Most Anti-Epileptics

# Alzheimer's Association 10 Ways to Love Your Brain

Break a Sweat – regular cardiovascular activity – I suggest 30 minutes of moderate intensity cardiovascular exercise 5 days per week

Hit the Books – formal education recommended at any stage in life

Butt Out – don't smoke, stop if you do

Follow your Heart – control vascular risks: obesity, hypertension, diabetes

Heads Up! – practice safety steps to reduce risk of head injuries

Fuel up Right – healthy and balanced diet such as Mediterranean & Mediterranean-DASH

Catch some ZZZ's – improve sleep quality including treating sleep apnea and avoiding insomnia

Take Care of your Mental Health – manage stress, treat anxiety and depression

Buddy Up – social activity is important

Stump Yourself – engage in challenging cognitive activities such as learning a new language or taking up a new art

# Regular Screening in our Elder Population

Screening works best in patient populations with diseases with high incidence and prevalence

Incidence and prevalence of dementia rises with age  
doubles every 5 years after the age of 65  
in the mid 80s some estimate almost 50% of the population have dementia!

Medicare offers some form of screening as part of yearly health assessments (65 years old and older)

## When to Check?

Anytime a person or someone who knows them is concerned about a change in memory, thinking, and/or behavior.

Screening in elders – no perfect answer but reasonable to start at 65 and older.

# The problem of screening tools

Many 'brief' cognitive tools have been developed over the past 50+ years – they vary but several do 'okay' at distinguishing demented from non-demented patients.

None do very well as screening tools distinguishing normal from pre-dementia mild cognitive impairment.

# Examples of Screening Instruments

## Tests:

MMSE

MoCA (registration required)

Mini-Cog (best bet for quick  
screen demented vs not  
demented)

SLUMS (public domain)

Qmci

6CIT

ACE-III

GPCOG

## Informant Questions:

GPCOG

IQCODE

AD8

# Check Labs for Reversible Causes

Check:

CMP

CBC with Differential

TSH with free T4

Vitamin B1 (thiamine) –  
WHOLE BLOOD ONLY!

Vitamin B12

Vitamin D

Magnesium

Consider Checking in select cases:

Syphilis screen (more than RPR as likely looking for tertiary)

HIV

Lyme

Ethanol

Toxicology Screen

ESR

# Additional Referrals to Consider

Options for referral if appropriate to

Sleep Clinic

Audiology (if major concern of hearing)

Eye Clinic (if major concern of seeing)

Physical therapy (if there is a fall concern)

Psychiatry (only if refractory to PCP management of depression)

## **If any risks identified, correct then rescreen**

If scores return to normal go back into surveillance – no further action needed

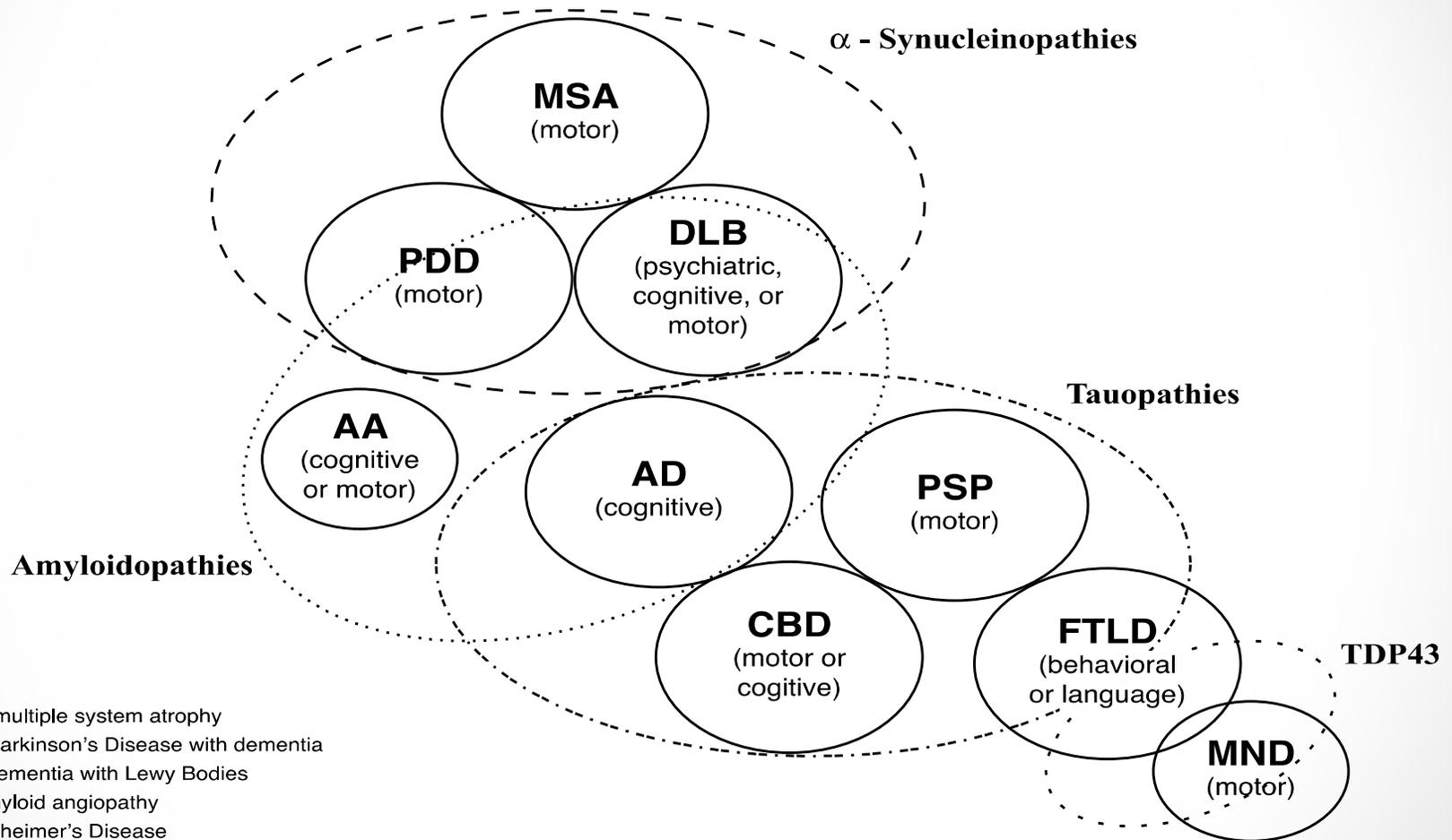
If scores do not return to normal, needs more evaluation!

- MRI of Brain (CT Head if MRI brain can't be obtained, but counsel MRI is the preferred study)
- Consultation to Cognitive Specialists

# Thoughts for Cognitive Consultation

- Consultation first to Neuropsychologist
  - Younger than 55 or younger than 65 with history of bipolar or with mild TBI
  - Subjective cognitive complaint (normal on screening)
- Consultation to both Neuropsychologist and Memory Physician
  - If over 55 (or 65 if bipolar or mild TBI) and early moderate dementia or better (e.g. MMSE over 15 and no recent neuropsychology testing)
- Consultation to a Memory Physician (e.g. Behavioral Neurologist)
  - Rapid progression (completely normal to demented in less than 6 months)
  - Late moderate dementia or worse (e.g. MMSE below 15)
  - Recent neuropsychology testing
- Consult Behavioral Neurologist if mixed cognitive and other neurologic symptoms
- Send to hospital if acute decline (from normal to demented in days to weeks rather than months)

# Overlap of Pathologies in Neurodegenerative Disease



- MSA** - multiple system atrophy
- PDD**- Parkinson's Disease with dementia
- DLB**- dementia with Lewy Bodies
- AA**- amyloid angiopathy
- AD**- Alzheimer's Disease
- PSP**- progressive supranuclear palsy
- CBD**- corticobasal degeneration
- FTLD**- frontotemporal lobar degeneration
- MND**- motor neuron disease

# Major Recommendations for Mild Cognitive Impairment from American Academy of Neurology Guidelines

Assess for MCI with validated tools in appropriate scenarios (Level B).

Evaluate patients with MCI for modifiable risk factors, assess for functional impairment, and assess for and treat behavioral/neuropsychiatric symptoms (Level B).

Monitor cognitive status of patients with MCI over time (Level B).

Cognitively impairing medications should be discontinued where possible and behavioral symptoms treated (Level B).

Clinicians may choose not to offer cholinesterase inhibitors (Level B); if offering, they must first discuss lack of evidence (Level A).

Recommend regular exercise (Level B).

Clinicians may recommend cognitive training (Level C).

Discuss diagnosis, prognosis, long-term planning, and the lack of effective medicine options (Level B).

Clinicians may discuss biomarker research with patients with MCI and families (Level C).

# Levels of Certainty in Alzheimer's Disease Diagnosis

## Three Evidence Types

Clinical Syndrome

Neurodegeneration (e.g. atrophy on structural imaging, decreased metabolism on FDG PET)

Biomarkers (e.g. Amyloid to Tau Index and Phosphorylated Tau in cerebrospinal fluid, Amyloid PET imaging if available)

Possible: 1 of 3 types of evidence support AD

Probable: 2 of 3 types of evidence support AD

Definite: 3 of 3 types of evidence support AD

# Most Common Clinical Presentation of Alzheimer's Disease Dementia

Short Term Memory (anterograde memory) first and worst.

Has multiple domains impacted enough to cause real world problems

Commonly will be worse at things like generating a list of animals than a list of words that start with a letter (semantic generation worse than phonemic generation)

Often will have more problems with things like naming and visuospatial processing

# List of Alzheimer's disease cognitive domains

## Impaired ability to acquire and remember new information

- repetitive questions or conversations
- misplacing personal belongings
- forgetting events or appointments
- getting lost on a familiar route

## Impaired reasoning and handling of complex tasks, poor judgment

- poor understanding of safety risks
- inability to manage finances
- poor decision-making ability
- inability to plan complex or sequential activities

## Impaired visuospatial abilities

- inability to recognize faces
- inability to recognize common objects
- inability to find objects in direct view despite good acuity
- inability to operate simple implements
- inability to orient clothing to the body

# List of Alzheimer's disease cognitive domains continued

## Impaired language functions (speaking, reading, writing)

difficulty thinking of common words while speaking, hesitations  
speech, spelling, or writing errors

## Changes in personality, behavior, or comporment

uncharacteristic mood fluctuations  
agitation  
impaired motivation  
impaired initiative  
apathy  
loss of drive  
social withdrawal  
decreased interest in previous activities  
loss of empathy  
compulsive or obsessive behaviors  
socially unacceptable behaviors

# Alzheimer's Disease: disease "mimicry" variants

## "Frontal"

- Behavioral
- Dysexecutive

## Language

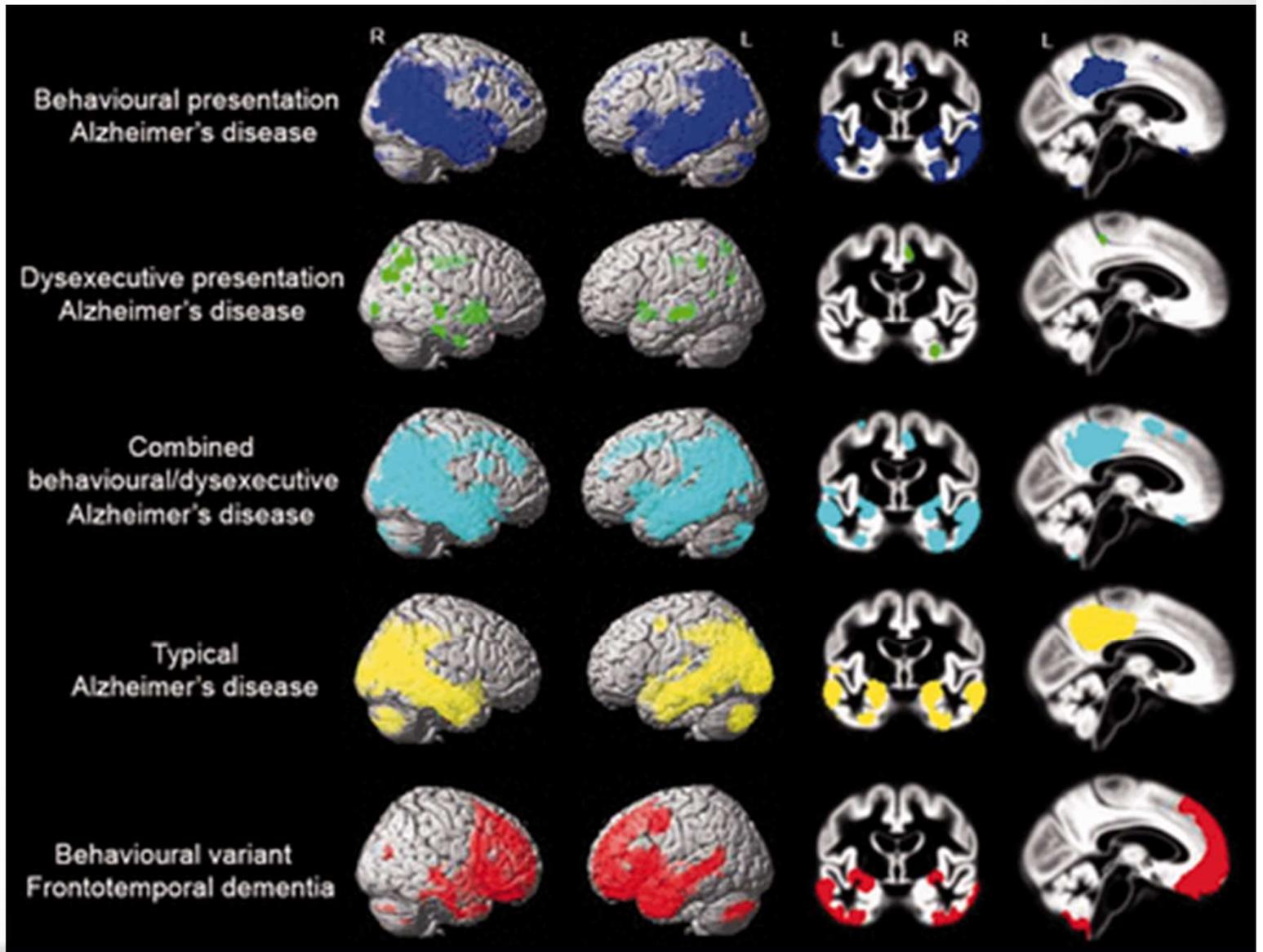
- Logopenic Primary Progressive Aphasia
- Other/mixed aphasias

## Visuospatial

- Posterior Cortical Atrophy

# “Frontal” Variant Alzheimer’s Disease – NOT!!!

Voxel-wise comparisons of grey matter volumes between healthy controls and the different diagnostic groups.



# Logopenic-variant Primary Progressive Aphasia

**Table 4** Diagnostic criteria for logopenic variant PPA

I. Clinical diagnosis of logopenic variant PPA

Both of the following core features must be present:

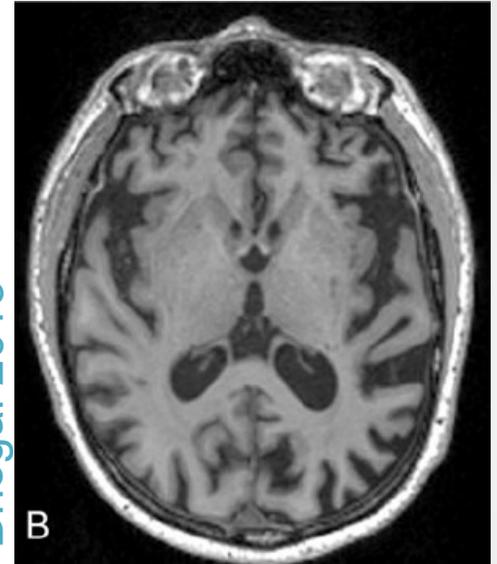
1. Impaired single-word retrieval in spontaneous speech and naming
2. Impaired repetition of sentences and phrases

At least 3 of the following other features must be present:

1. Speech (phonologic) errors in spontaneous speech and naming
2. Spared single-word comprehension and object knowledge
3. Spared motor speech
4. Absence of frank agrammatism

Gorno-Tempini *et al* 2011

Bhagal 2013

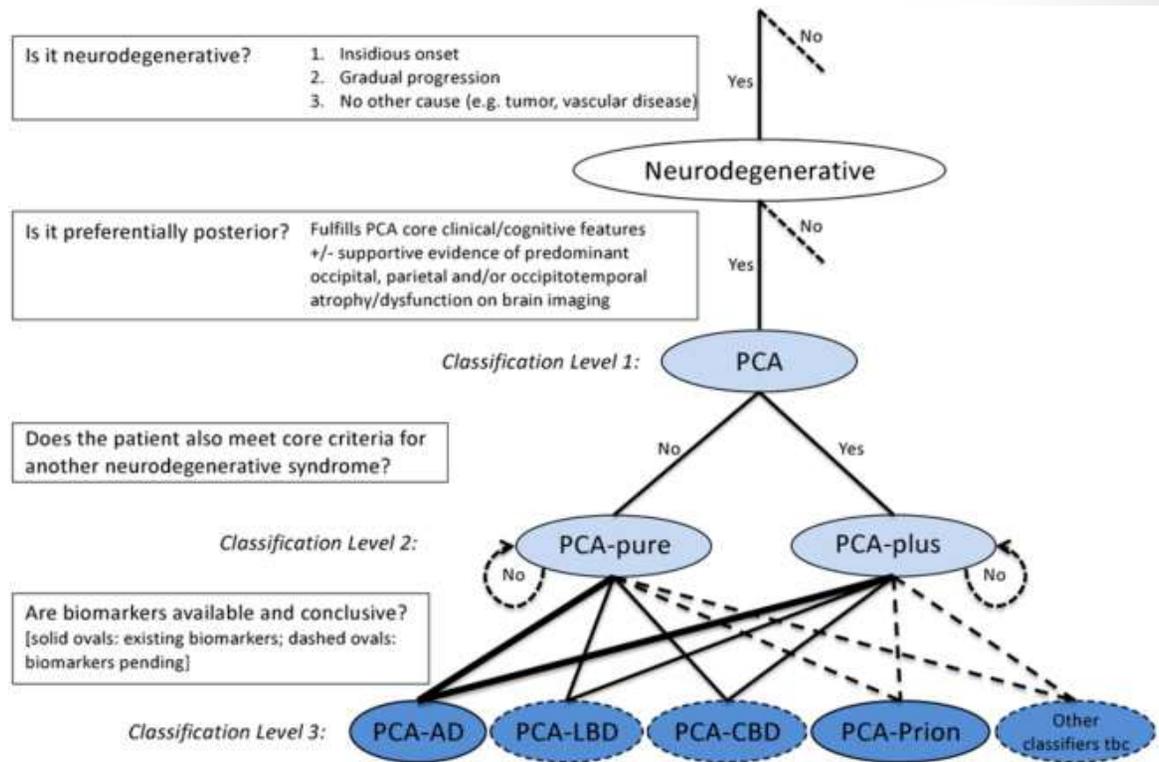


Logopenic variant PPA: note asymmetric involvement of the left angular gyrus and posterior temporal and occipital lobe

# Posterior Cortical Atrophy Consensus Classification Crutch 2017

**PCA-pure:** Fulfill the criteria for the core clinico-radiological PCA syndrome and not fulfill core clinical criteria for any other neurodegenerative syndrome.

**PCA-plus:** Fulfill the criteria for the core clinico-radiological PCA syndrome and also fulfill core clinical criteria for at least one other neurodegenerative syndrome. PCA-AD and PCA-prion (solid ovals) are distinguished from PCA-LBD and PCA-CBD (dashed ovals) owing to the current availability of in vivo pathophysiological biomarkers. Other disease-level classifications may be appropriate (e.g., a patient with PCA plus visual hallucinations may have LBD-variant of AD) or anticipated (e.g., PCA attributable to GRN mutations). The thickness of lines connecting classification levels 2 and 3 is intended to reflect the status of AD as the most common cause of PCA.



## **Don't Forget that Vascular Dementia is #2 Cause of Dementia and largely preventable!**

Multiple Infarct Dementia fairly clear-cut in etiology and diagnosis but needs the same sort of care as neurodegenerative dementias.

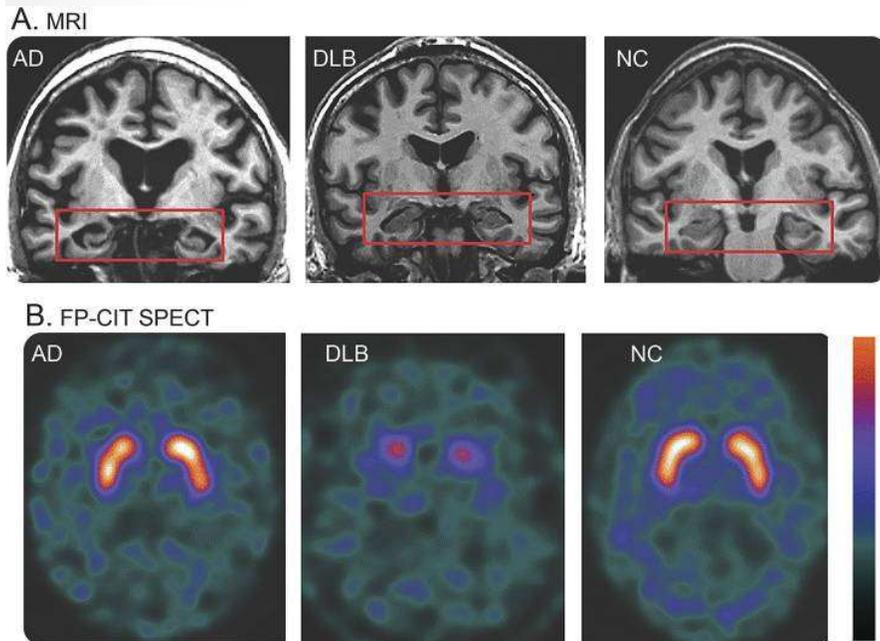
Subcortical Vascular Dementia harder to diagnose.

Libon et al proposed need 25% of white matter involved for subcortical vascular disease to contribute to dementia and 50% for it to be sole cause of a dementia.

# Dementia with Lewy Bodies update!

## McKeith et al. 2017

SPECT DaT distinguishing DLB from AD is well-established, with sensitivity (78%) and specificity (90%).



**Table 1** Revised<sup>1,2</sup> criteria for the clinical diagnosis of probable and possible dementia with Lewy bodies (DLB)

**Essential** for a diagnosis of DLB is dementia, defined as a progressive cognitive decline of sufficient magnitude to interfere with normal social or occupational functions, or with usual daily activities. Prominent or persistent memory impairment may not necessarily occur in the early stages but is usually evident with progression. Deficits on tests of attention, executive function, and visuo-perceptual ability may be especially prominent and occur early.

**Core clinical features** (*The first 3 typically occur early and may persist throughout the course.*)

Fluctuating cognition with pronounced variations in attention and alertness.  
 Recurrent visual hallucinations that are typically well formed and detailed.  
 REM sleep behavior disorder, *which may precede cognitive decline.*  
 One or more spontaneous cardinal features of parkinsonism: these are bradykinesia (defined as slowness of movement and decrement in amplitude or speed), rest tremor, or rigidity.

**Supportive clinical features**

Severe sensitivity to antipsychotic agents; postural instability; repeated falls; syncope or other transient episodes of unresponsiveness; severe autonomic dysfunction, e.g., constipation, orthostatic hypotension, urinary incontinence; hypersomnia; hyposmia; hallucinations in other modalities; systematized delusions; apathy, anxiety, and depression.

**Indicative biomarkers**

Reduced dopamine transporter uptake in basal ganglia demonstrated by SPECT or PET.  
 Abnormal (low uptake) <sup>123</sup>Iodine-MIBG myocardial scintigraphy.  
 Polysomnographic confirmation of REM sleep without atonia.

**Supportive biomarkers**

Relative preservation of medial temporal lobe structures on CT/MRI scan.  
 Generalized low uptake on SPECT/PET perfusion/metabolism scan with reduced occipital activity ± the cingulate island sign on FDG-PET imaging.  
 Prominent posterior slow-wave activity on EEG with periodic fluctuations in the pre-alpha/theta range.

**Probable DLB** can be diagnosed if:

- Two or more core clinical features of DLB are present, with or without the presence of indicative biomarkers, or
- Only one core clinical feature is present, but with one or more indicative biomarkers.

**Probable DLB** should not be diagnosed on the basis of biomarkers alone.

**Possible DLB** can be diagnosed if:

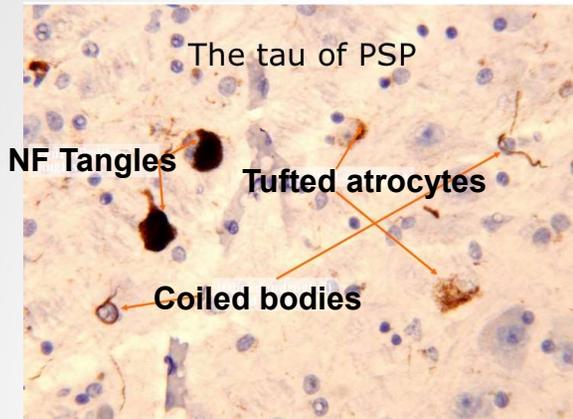
- Only one core clinical feature of DLB is present, with no indicative biomarker evidence, or
- One or more indicative biomarkers is present but there are no core clinical features.

**DLB is less likely:**

- In the presence of any other physical illness or brain disorder including cerebrovascular disease, sufficient to account in part or in total for the clinical picture, although these do not exclude a DLB diagnosis and may serve to indicate mixed or multiple pathologies contributing to the clinical presentation, or
- If parkinsonian features are the only core clinical feature and appear for the first time at a stage of severe dementia.

DLB should be diagnosed when dementia occurs before or concurrently with parkinsonism. The term Parkinson disease dementia (PDD) should be used to describe dementia that occurs in the context of well-established Parkinson disease. In a practice setting the term that is most appropriate to the clinical situation should be used and generic terms such as Lewy body disease are often helpful. In research studies in which distinction needs to be made between DLB and PDD, the existing 1-year rule between the onset of dementia and parkinsonism continues to be recommended.

# Progressive Supranuclear Palsy



## Clinical criteria of probable PSP:

- Gradually progressive disorder
  - Onset at age 40 or later
  - Vertical (upward or downward gaze) supranuclear palsy
- AND**
- Prominent postural instability with tendency to fall in the first year of disease onset
  - No evidence of other diseases that could explain these features

## Supportive features of PSP:

- Symmetric akinesia or rigidity
- Abnormal neck posture
- Poor or absent response of parkinsonism to levodopa therapy
- Early dysphagia and dysarthria

## Early onset of cognitive impairment including at least two of the following:

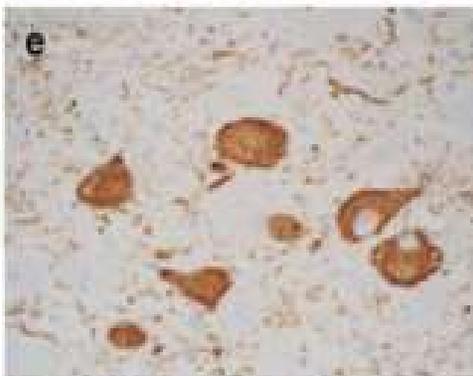
- apathy, impairment in abstract thought, decreased verbal fluency, utilization or imitation behavior, or frontal release signs

# Corticobasal Degeneration

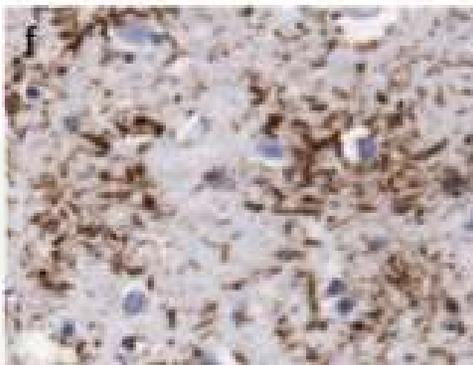
ballooned neurons



pretangles in basal nucleus



astrocytic plaques in the cortex



## Core Features of Corticobasal Syndrome (Boeve *et al* 2003):

Insidious onset and progressive course w/out identifiable cause

- Cortical dysfunction as reflected by one of the following:
  - Focal or asymmetric ideomotor apraxia
  - Alien limb phenomenon
  - Cortical sensory loss
  - Visual or sensory hemineglect
  - Constructional apraxia
  - Focal or asymmetric myoclonus
  - Apraxia of speech/nonfluent aphasia
- Extrapyrarnidal dysfunction as reflected by at least one of the following:
  - Focal or asymmetric appendicular rigidity lacking prominent/sustained levodopa response
  - Focal or asymmetric appendicular dystonia

# OR Should we say Corticobasal Syndrome?

## Pathologic Diagnoses in 36 Consecutive Autopsied Cases at the Mayo Clinic With the Corticobasal Syndrome

Diagnosis	No. of cases
Corticobasal degeneration	18
Progressive supranuclear palsy	6
Alzheimer's disease	4
Creutzfeldt–Jakob disease	3
Nonspecific degenerative changes	3
Pick's disease	1
Combined Alzheimer's disease/Pick's disease	1

# Non-fluent/Agrammatic variant Primary Progressive Aphasia

**Table 2**      **Diagnostic features for the nonfluent/agrammatic variant PPA**

**I. Clinical diagnosis of nonfluent/agrammatic variant PPA**

At least one of the following core features must be present:

1. Agrammatism in language production
2. Effortful, halting speech with inconsistent speech sound errors and distortions (apraxia of speech)

At least 2 of 3 of the following other features must be present:

1. Impaired comprehension of syntactically complex sentences
2. Spared single-word comprehension
3. Spared object knowledge

# Semantic Dementia

**Table 3      Diagnostic criteria for the semantic variant PPA**

**I. Clinical diagnosis of semantic variant PPA**

Both of the following core features must be present:

1. Impaired confrontation naming
2. Impaired single-word comprehension

At least 3 of the following other diagnostic features must be present:

1. Impaired object knowledge, particularly for low-frequency or low-familiarity items
2. Surface dyslexia or dysgraphia
3. Spared repetition
4. Spared speech production (grammar and motor speech)

# Behavioral Variant FTD

**International consensus criteria for behavioural variant FTD** (Raskovsky et al 2011):

Shows progressive deterioration of behaviour and/or cognition by observation or history (as provided by a knowledgeable informant), affecting function, with imagign consistent with FTD

**Three** of the following behavioural/cognitive symptoms (A–F) must be present to meet criteria.

- A. Early behavioural disinhibition [one of the following symptoms (A.1–A.3) must be present]:
  - A.1. Socially inappropriate behaviour
  - A.2. Loss of manners or decorum
  - A.3. Impulsive, rash or careless actions
- B. Early loss of apathy or inertia
- C. Early loss of sympathy or empathy
- D. Early perseverative, stereotyped or compulsive/ritualistic behaviour
  - D.1. Simple repetitive movements
  - D.2. Complex, compulsive or ritualistic behaviours
  - D.3. Stereotypy of speech
- E. Hyperorality and dietary changes [one of the following symptoms (E.1–E.3) must be present]
  - E.1. Altered food preferences
  - E.2. Binge eating, increased consumption of alcohol or cigarettes
  - E.3. Oral exploration or consumption of inedible objects
- F. Neuropsychological profile: executive/generation deficits with relative sparing of memory and visuospatial functions

# New Diagnosis of Dementia

Review Advanced Care/End of Life Care Planning and Goals of Care

Resources

- Alzheimer's Association (it's not just for Alzheimer's disease!)
- Area Agency on Aging in PA
- Elder Law Attorney

# Cognitive Rehabilitation

Some evidence in Mild Cognitive Impairment and can help in Mild Dementia

Typically, by some Speech Language Pathologists less commonly some Occupational Therapists

It is possible to 'retrain' the memory system and with more structured learning important facts and techniques can be learned.

This is true to a degree even in patients with progressive neurodegenerative disorders.

# Aducanumab

- Monoclonal antibody targeting amyloid
  - Monthly infusion over 1 hour
- FDA approved June 2021
- Recommended only for mildest of the mild
  - Practically means mild cognitive impairment, rarely mild dementia
- Should have biomarker evidence for abnormal amyloid in CNS
  - Lumbar Puncture for cerebrospinal fluid AD markers
  - Perhaps Amyloid PET in future
- Controversial as to whether it has significant benefit
  - Generous interpretation of data so far seen would be 22% reduction in rate of decline after 1.5 years.
- Significant side effect of ARIA-E and ARIA-H common
  - Need to be able to have MRI before starting, 6 months, 12 months

# Acetylcholinesterase Inhibitors

## Three commonly used in the United States

- Donepezil:
  - minimal therapeutic dose is 5 mg daily
  - can increase to 10 mg daily after four weeks
  - highest dose is 23 mg daily
- Rivastigmine:
  - start patch at 4.6 mg/24 hours
  - increase in 4 weeks to minimal therapeutic dose of 9.5 mg/24 hr daily
  - highest dose is 13.3 mg/24 hours
  - Minimal therapeutic dose of rivastigmine oral form is 4.5 mg twice daily (but highest risk of gastrointestinal side effects), can go up to 6 mg twice daily
- Galantamine XR:
  - start at 8 mg daily
  - increase in 4 weeks to minimal therapeutic dose of 16 mg daily
  - 4 weeks after that can increase to 24 mg daily

# Acetylcholinesterase Inhibitors – Limiting Side Effects

Main side effects gastrointestinal

- Nausea
- Diarrhea
- Vomiting (rarely)
- other side effects include bradycardia, vivid dreaming/sleep disturbance, and muscle cramps.

Gradual titration helps limit side effects.

Taking oral forms with a large, hearty meal helps lower risk of GI side effects.

Taking medication earlier in the day may help if patients have dream/sleep side effects.

If a patient can not tolerate one AChE-I, try another.

# Memantine (Namenda) (weak NMDA antagonist)

Two forms, immediate and extended release  
(XR)

- Titration of immediate release memantine is increase by 5 mg weekly to target of 10 mg BID.
- Titration of extended release memantine is increasing by 7 mg weekly to target of 28 mg Daily.

Only evidence for a therapeutic dose is at the 10 mg twice daily or 28 mg daily for extended release dose except when dose needs adjustment for decreased renal excretion.

Can be a double edged sword in agitation – transient agitation increase has been noted anecdotally but long term use reduces agitation.

# Agitation Behavioral Advice

Challenging or debating doesn't work with dementia patients - recommendation is for distracting the patient from what is agitating, as well as redirecting them to more constructive activities.

# Sundowning

The literature has very little evidence for effective and safe treatment for sundowning in Dementia.

Behavioral Management is still considered first line - distraction, redirection.

One study recommends using LED lighting bright blue white during the day and dimmer yellow white at night and that this helps reduce behaviors.

Another safe option is the introduction of the supplement melatonin. I would recommend taking it no earlier than two hours before the sundowning behavior typically occurs. Can start with 3 mg and go as high as 16 mg.



# Aggression, Agitation, Delusion, Hallucination Principles

- Environmental and Behavioral Interventions Safest and Most Proven
- If appropriate for diagnosis, use of the acetylcholinesterase inhibitors and memantine can have behavior benefits
- SSRIs and SNRIs can be tried for agitation – avoid paroxetine as it is also anticholinergic
- If needed, can try mirtazapine, trazodone, and/or aripiprazole
- Benzodiazepines should be avoided unless history of good response
- Little evidence but some anecdotal success with lamotrigine
- Atypical antipsychotics only if all else fails and behaviors are dangerous or very distressing
  - quetiapine may be safest
  - risperidone slightly more evidence
  - pimavanserin and clozapine (with caution) can be considered in Parkinson's disease dementia/Lewy body dementia if too much movement side effect

# Things that Kill in Dementia - Driving

Driving while demented!

AAN Driving & Dementia guidelines of when to be concerned:

CDR score 0.5 or worse

MMSE score 24 or worse

Self Restriction of Driving

Others concerned about Driving

Patients are NOT reliable evaluators of their own safety!

PA is a mandatory reporting state (but vague as to when you should report for cognitive concerns)

Gold standard is Driving Rehabilitation Evaluation (usually through occupational therapy) where patient taken out on road.

Driving Rehabilitation Evals usually NOT covered by insurance!!

# Things that Kill in Dementia - Fire

Burning down the house!

Some patients are risk for starting fires.

Leave things burning (many forget, many can't smell smoke)

Some even play with matches.

# Things that Kill in Dementia - Wandering

## Wandering!

Patients who are not found within the first 24 hours after wandering away from home are usually found dead.

Wandering behavior not inevitable, but becomes more likely as the dementia progresses.

Consider using home alarm systems to help guard against leaving home.

Patients who wander need 24/7 supervision

Can be a challenge to get in home help.

Safe return programs exist and can help save lives!

Finding them can be challenging however.

# Things that Kill in Dementia – Temperature Extremes

## Extremes of Heat and Cold

Dementia patients may not always recognize or seek help for early symptoms of heat stroke or hypothermia.

Need to make sure that homes have warmth in winter, and cooler options in a heat wave.

May need to intervene with utilities to guarantee power to elders with dementia when financially strapped (or they've forgotten to pay).

# Things that Kill in Dementia

Falls!

Many dementia patients also have problems with falls.

Physical therapy for gait assessment, retraining, assistive devices.

Occupational therapy for home safety assessments.

# Things that Kill in Dementia

Caregiver Stress and Burnout!!

Caregivers have as much, maybe more, morbidity and mortality than dementia patients themselves.

Need to educate to take care of themselves, need to educate regarding respite care.

Need access to respite care options!

# Recap

## DETECTION, DIAGNOSIS, & TREATMENT OF ALZHEIMER'S DISEASE AND RELATED DISORDERS

**How to better provide prevention advice, regularly screen for dementia risk, and get to accurate diagnosis. And once a diagnosis of dementia is present, how to manage to optimize quality of life and safety.**

**There are incredible demands on time in everyday clinical practice and dementias are both insidious and challenging to address when the disease itself often takes away the clinician's best ally. Most practices are not structured to be able to handle the detailed and time-consuming evaluation and counseling needed to provide best practice care. There is also in the community a lot of fatalism and lack of knowledge regarding dementias and related disorders that makes it harder to address.**

**Regularly educate on brain health techniques to patients, regularly screen for decline in memory and cognition at its earlier presentation, aggressively investigate and correct possible reversible causes and contributors, and empower providers, patients, and families to keep things as safe as possible in a debilitating disease.**



# QUESTION TIME!

