

Approach to Neurocognitive Disorders with Acute/Subacute and Insidious Onset

Neuropsychiatry: A Comprehensive Update 2026

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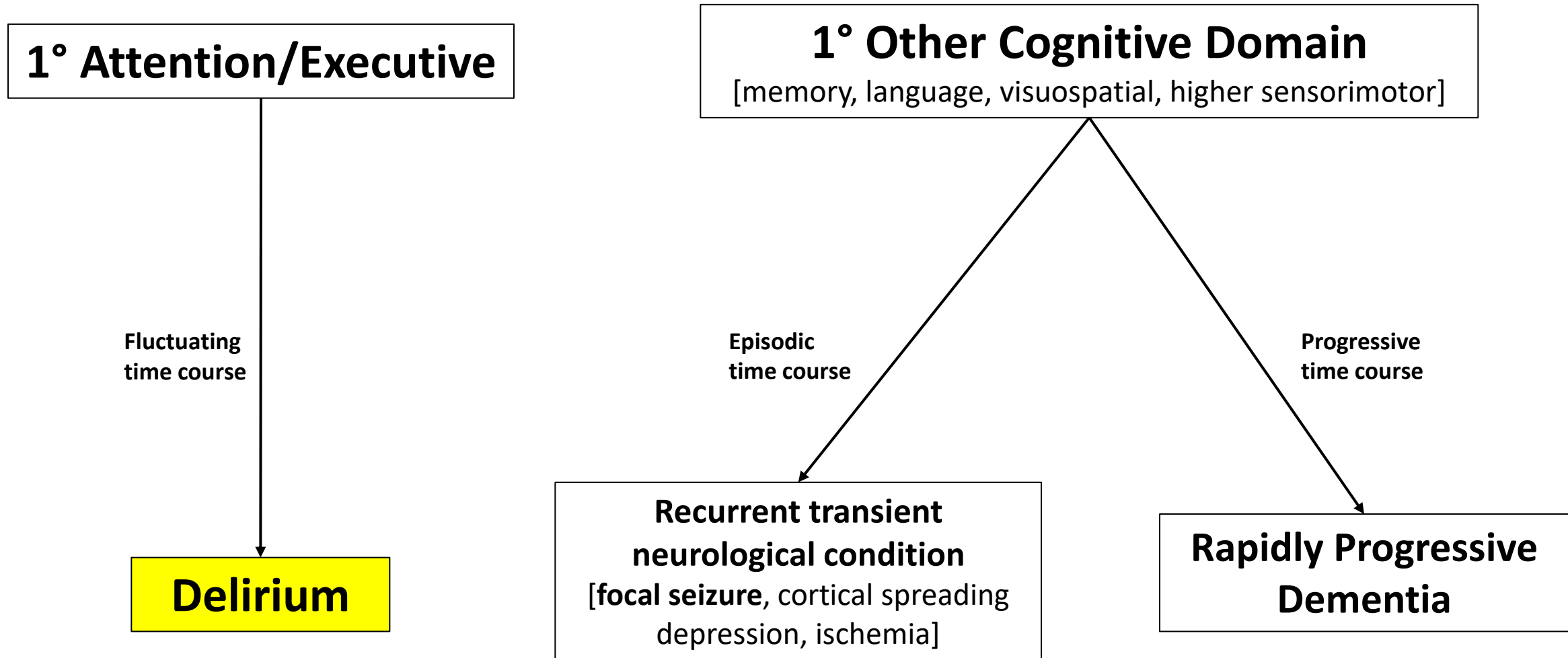
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Neither I nor my spouse has a relevant financial relationship with a commercial interest to disclose.

Goals

- To recognize the value of localization and time course in the diagnostic process of neurocognitive disorders, specifically:
 - As applied to disorders with acute/subacute onset, distinguishing delirium from rapidly progressive dementia and recurrent transient neurological conditions.
 - As applied to disorders with insidious onset, distinguishing neurodegenerative from non-degenerative conditions.
- To review the value of differentiating between syndrome, severity, and neuropathology in neurodegenerative cognitive disorders.
- To understand the key features of and workup for common conditions [i.e., typical Alzheimer disease (AD), Lewy body disease] and identify features that should prompt consideration of and workup for uncommon conditions [i.e., atypical AD, frontotemporal lobar degeneration].

Acute/Subacute Onset Cognitive Dysfunction



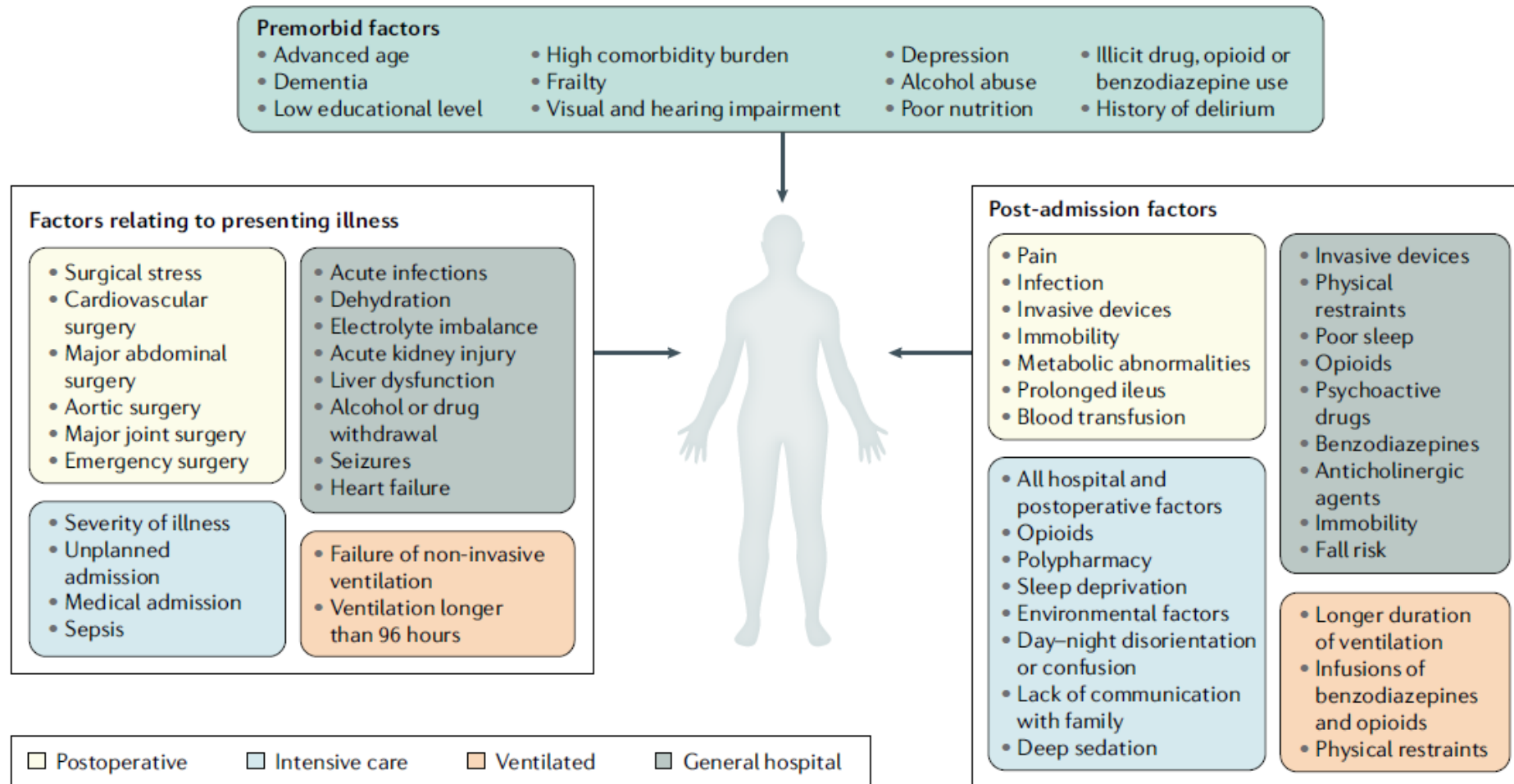
Delirium

- **Primary disturbance in attention: inability to maintain a coherent stream of thought.**
- **Fluctuating course with respect to attention and alertness; disrupted circadian rhythm.**
- **Absence of other localizing neurological signs.**
- **Tremulousness, myoclonus, asterixis, visual hallucinations (variably).**
- **Can be “hypoactive,” “hyperactive,” (with psychomotor agitation and sympathetic hyperactivity), or mixed.**

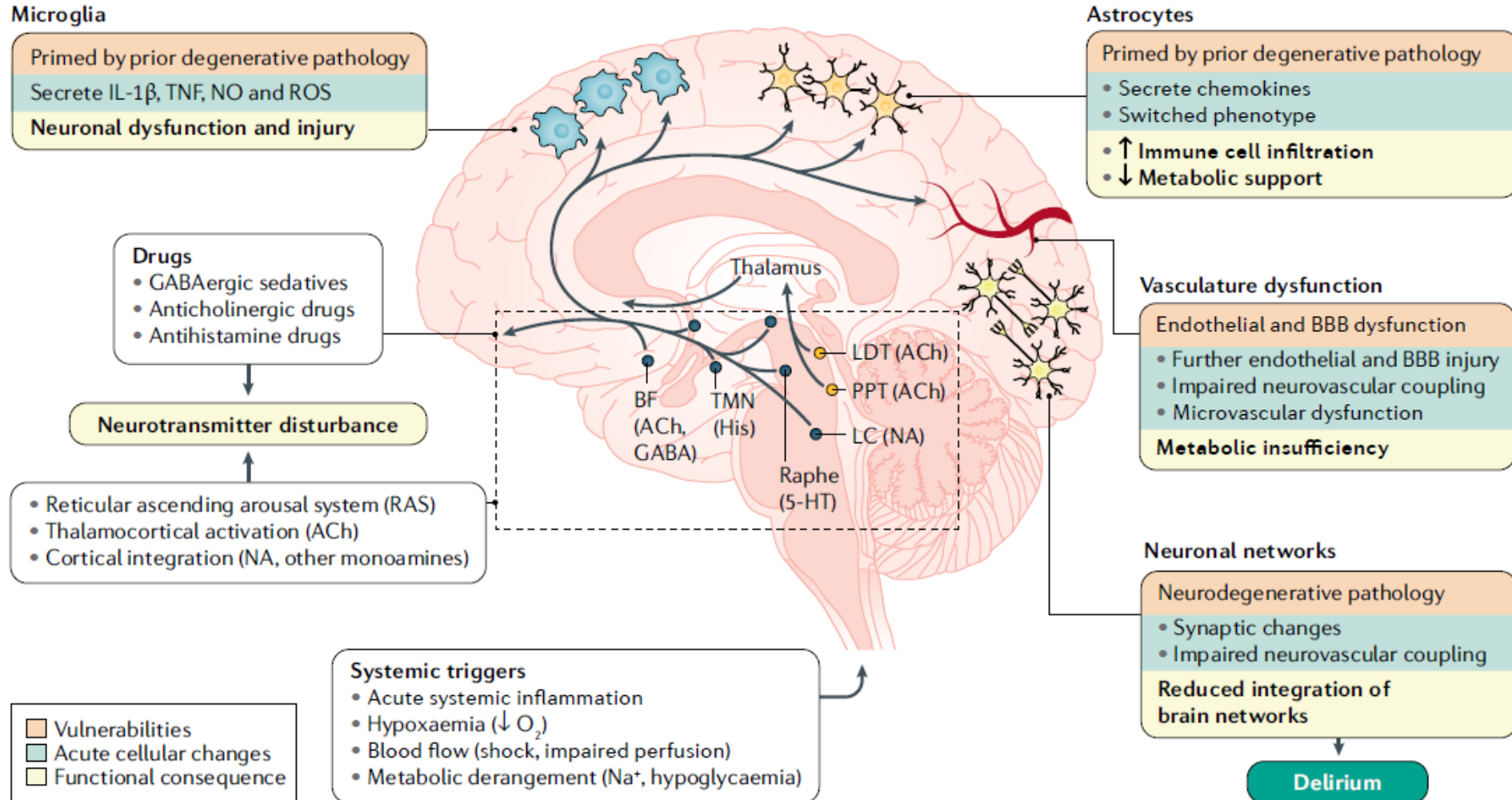


Delirium

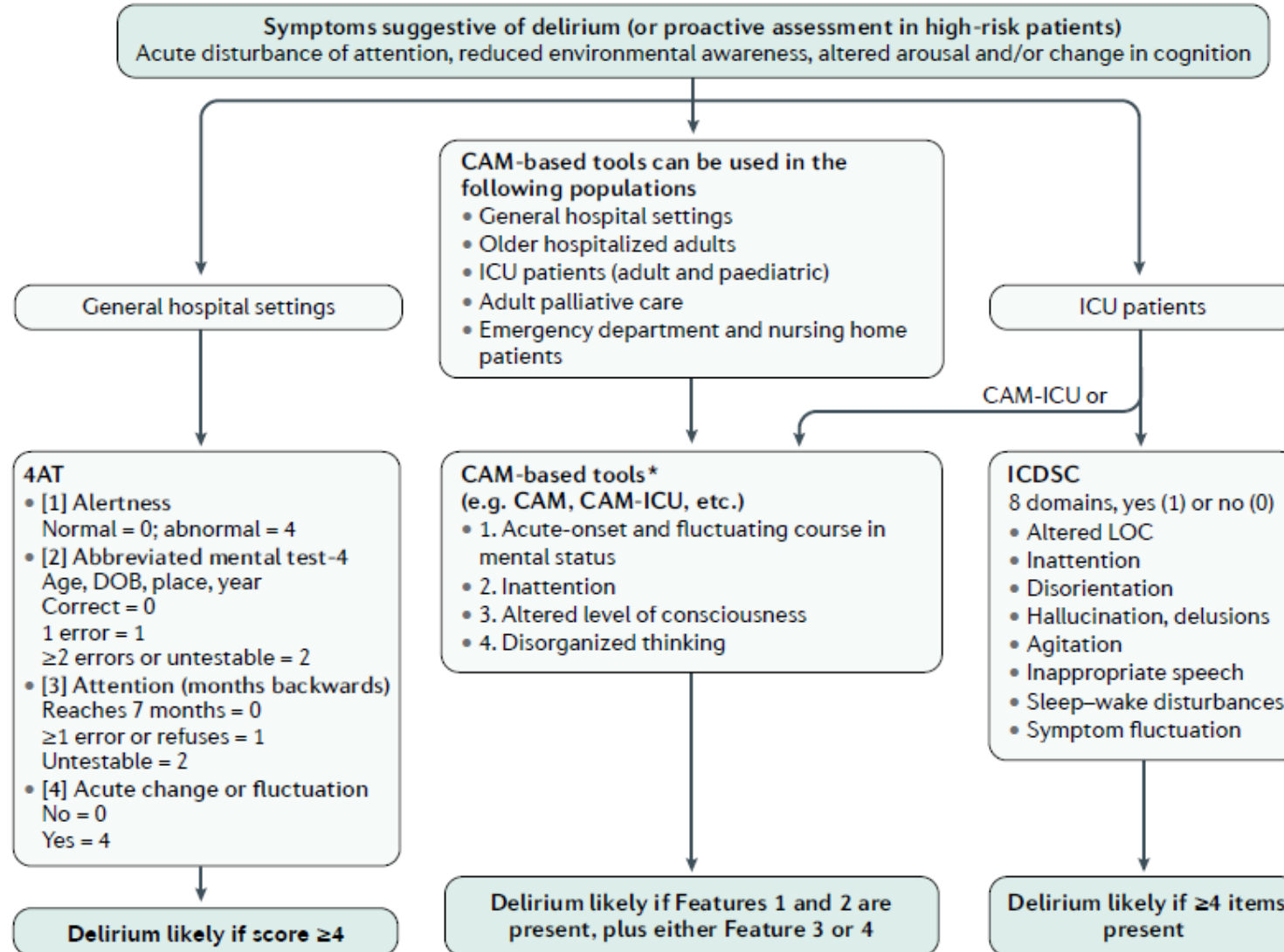
- **Very common:** ~10-15% medical/surgical inpatients, ~25-45% hospitalized cancer patients, ~60% ICU patients, ~80% patients receiving mechanical ventilation, ~80-90% terminally ill palliative care patients.



Delirium: Pathophysiology



Delirium: Screening



Delirium: Evaluation

- **Basic evaluation: metabolic, toxic, infectious causes**
 - Physical and neurological examinations; vitals, pulse oximetry
 - CBC, electrolytes, glucose, BUN, creatinine, LFTs, urinalysis and culture; drug levels; blood, urine toxicology
 - Chest x-ray, ECG
- **Evaluation dictated by preliminary findings**
 - Lumbar puncture for unexplained fever, nuchal rigidity, or suspected infection with unknown source
 - Neuroimaging (CT or MRI for focal findings or evidence of trauma)
- **Evaluation for persistent unexplained delirium**
 - Expanded lab workup (e.g., thyroid function, B12, ammonia, blood gas)
 - EEG
 - MRI brain
 - Lumbar puncture



Delirium: Prevention and Treatment

Box 2 | Delirium prevention in different health-care settings

Consensus guidelines^{241,262} make a number of recommendations for delirium prevention in various health-care settings.

General settings

Multicomponent interventions

- Early recognition of high-risk factors (age >65 years, dementia, hip surgery and high acuity)
- Daily screening for delirium
- Environmental orientation (sensory, auditory, dentures, time, events, family visits and music)
- Maintain normal hydration
- Regulation of bladder and bowel function
- Early establishment of normal diet
- Correction of metabolic disorders
- Cardiorespiratory optimization (with provision of oxygen if appropriate)
- Early identification of infection
- Effective treatment of pain
- Daily mobilization
- Avoidance of antipsychotic drugs
- Avoidance of benzodiazepines
- Reduced nocturnal disturbances to promote sleep

- Early removal of devices (intravascular and airway devices)
- Avoidance of physical restraints
- Sleep promotion (eye mask and earplugs)

Pharmacological interventions

- None with high-level evidence

Intensive care and high acuity units, intubated and non-intubated patients

Non-pharmacological interventions (as above, consider also)

- Early recognition of high-risk patients (age >65 years, high acuity, sepsis, shock, dementia and ventilation)
- Light sedation
- No benzodiazepines
- Early mobilization
- Promotion of day–night routine
- Environmental awareness and orientation
- Removal of devices (intravascular and airway devices)

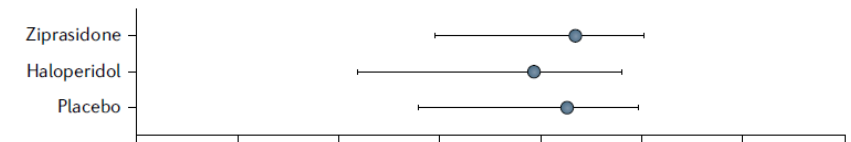
Pharmacological interventions

- Some suggestive evidence but not recommended by consensus guidelines

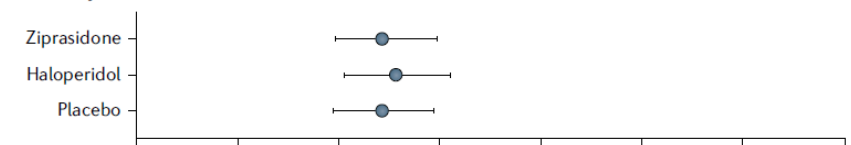
Treat the underlying etiology

- Discontinue non-essential potential contributing medications
- Thiamine supplementation for patients with evidence of nutritional deficiency
- Identify and manage withdrawal states
- Consider low-dose, short-term antipsychotic (haloperidol, risperidone, ziprasidone, olanzapine, quetiapine) for patients with potential to harm self or others despite non-pharmacological interventions.

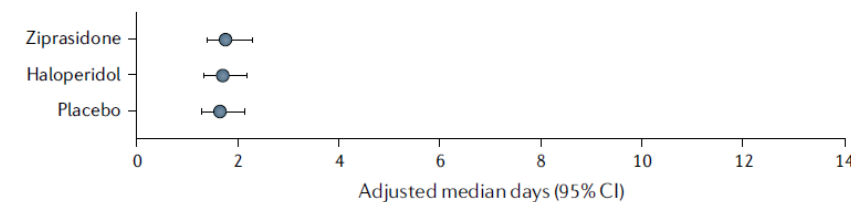
a Days alive without delirium or coma



b Days with delirium



c Days with coma

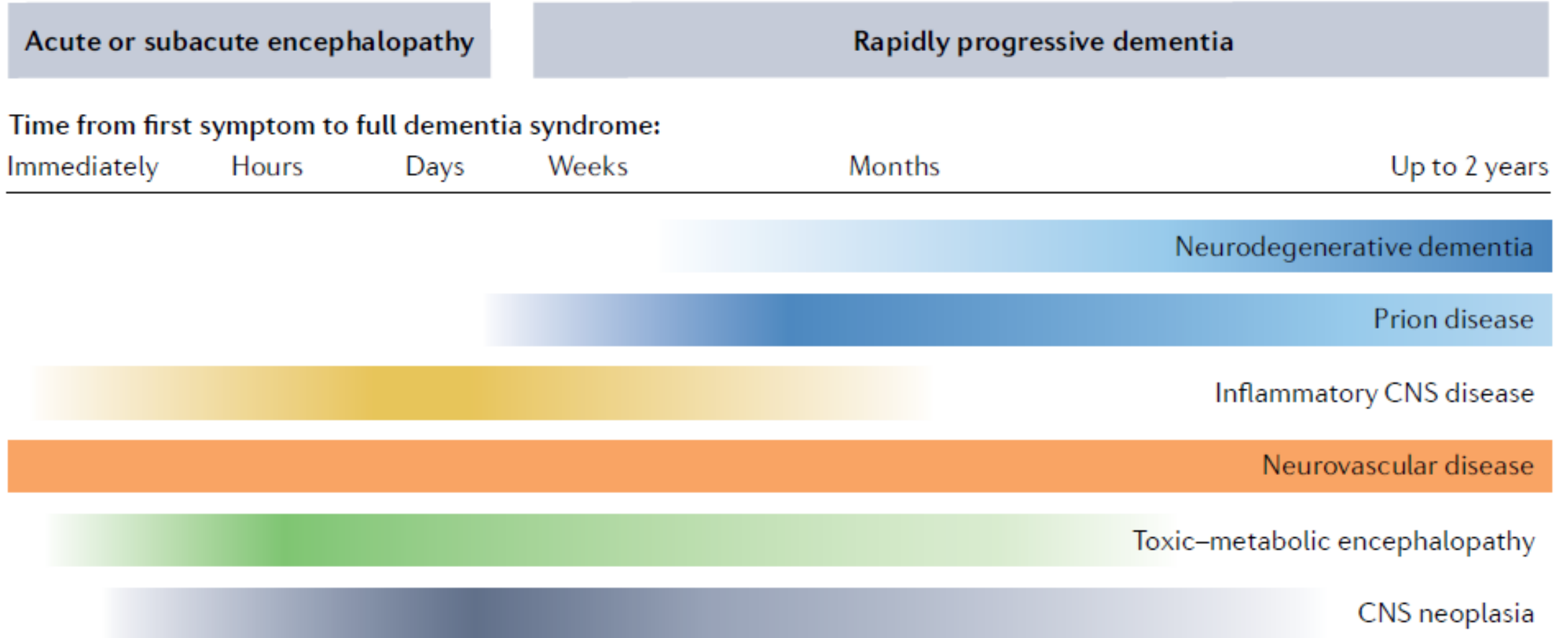


Clues of a primary neurological etiology (subacute presentations)

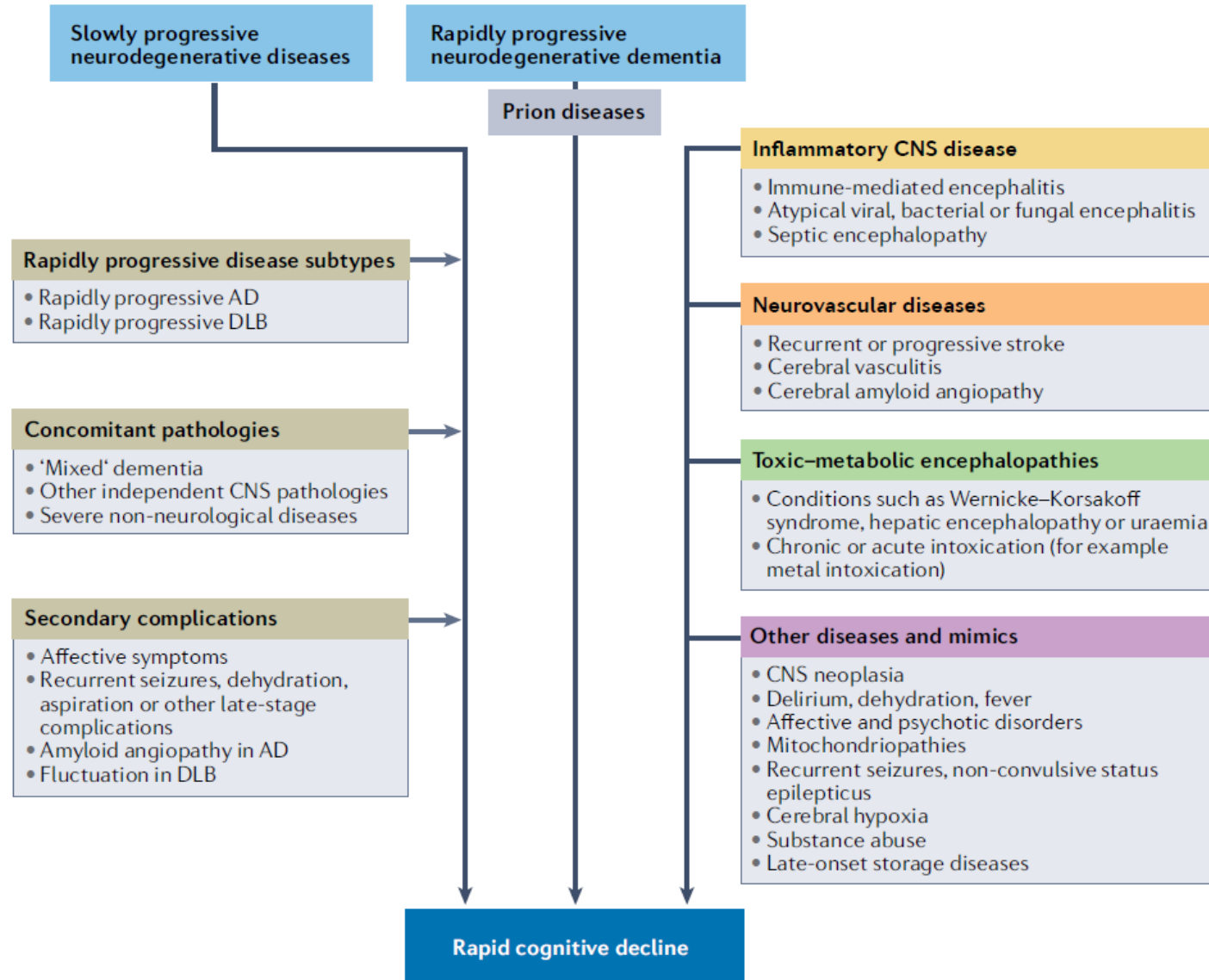
- **Primary domain of impairment in a cognitive domain other than attention.**
- **Neurological signs (other than asterixis/myoclonus).**
- **Steadily progressive time course.**
- **Pertinent abnormalities on neuroimaging, EEG, CSF.**

There is great value in developing and employing a time-efficient bedside mental status and general neurological examination to gain comfort with what is normal vs abnormal.

Rapidly Progressive Dementia

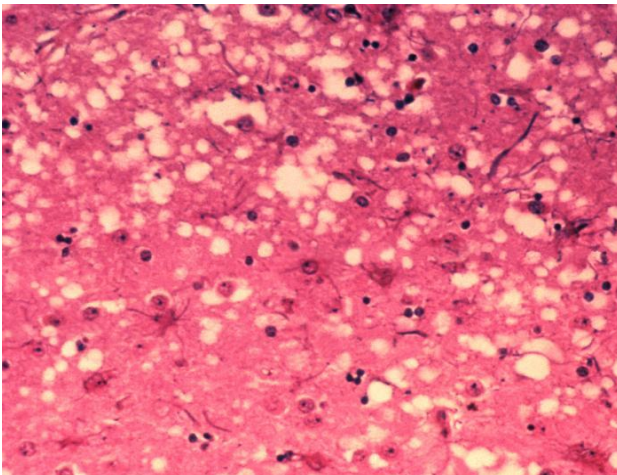


Rapidly Progressive Dementia



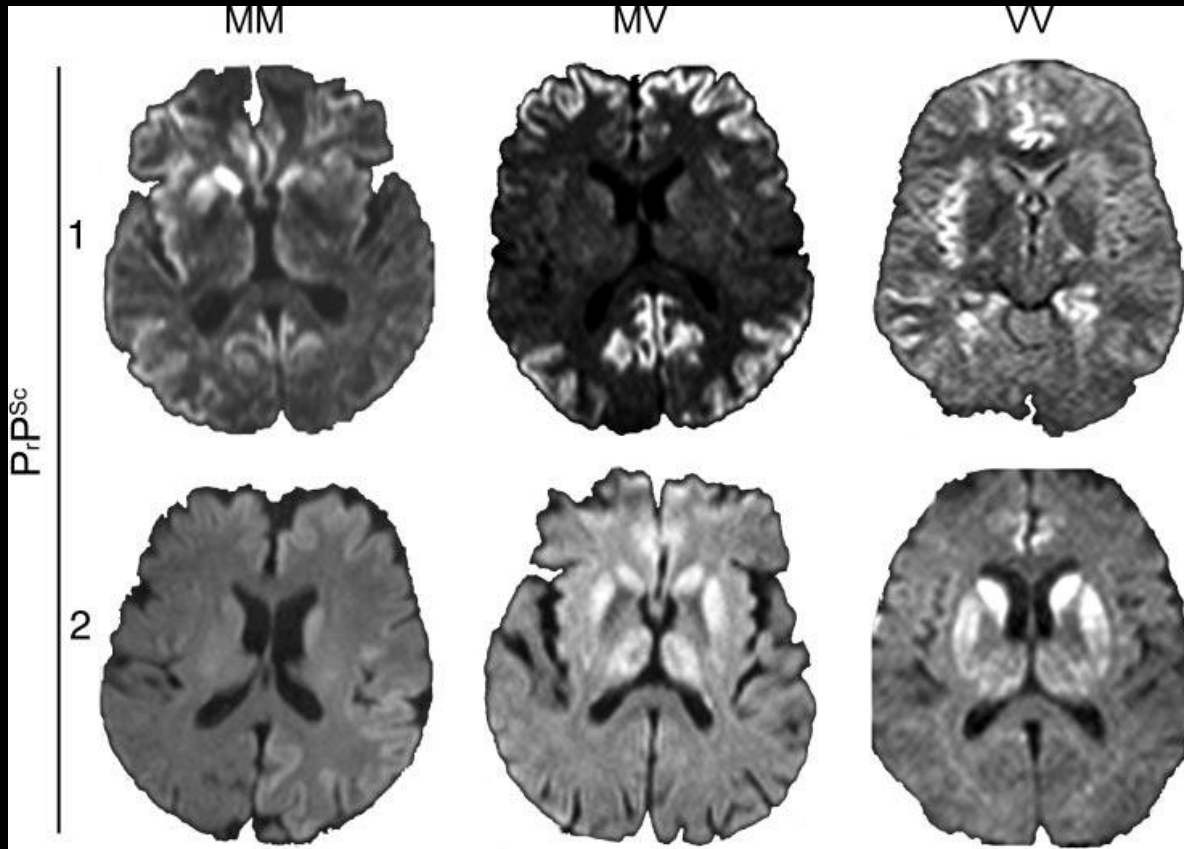
Prion disease

- Sporadic Creutzfeldt-Jakob disease (sJCD) is the most common; incidence 1-2 per 1,000,000 per year.
- Rapidly progressive deterioration in cognition and behavior with higher cortical signs; myoclonus occurs in >90% of cases; pyramidal or extrapyramidal motor signs (>50%), cerebellar signs (>50%) are also common.
- Neuropsychiatric features: apathy → akinetic mutism, depression; less commonly euphoria, emotional lability, anxiety, psychotic features.
- Molecular variants defined by amino acid at codon 129 of prion protein gene (M or V) and type of pathological prion (1 or 2) influence clinical presentation.



CJD biomarkers

DWI restriction ~90-95% sensitive, ~90-99% specific

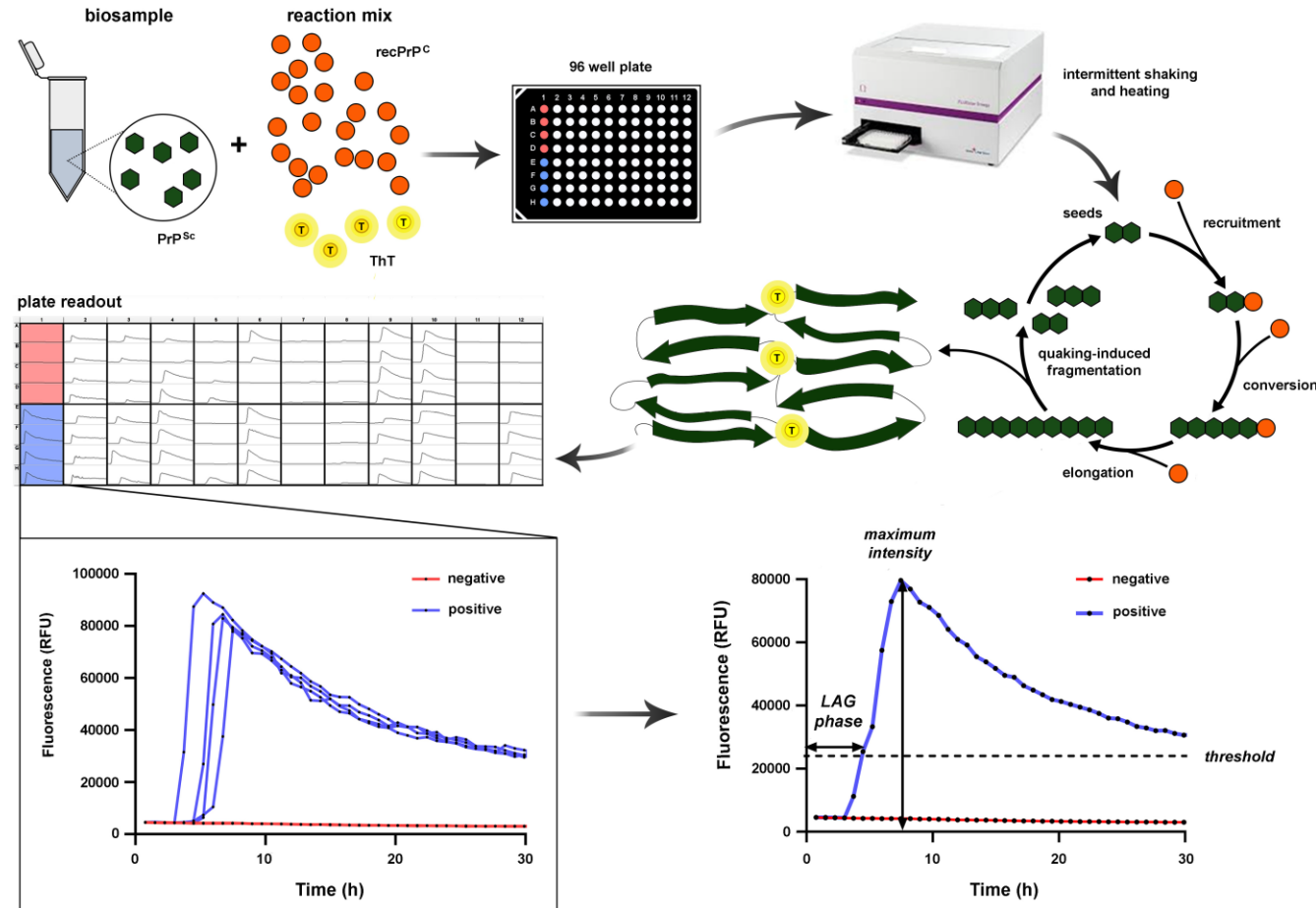


Periodic sharp wave complexes ~65% sensitive, ~90% specific



- Supportive but non-specific: elevated CSF 14-3-3 protein; highly elevated CSF total tau

CJD biomarkers: RT-QuIC

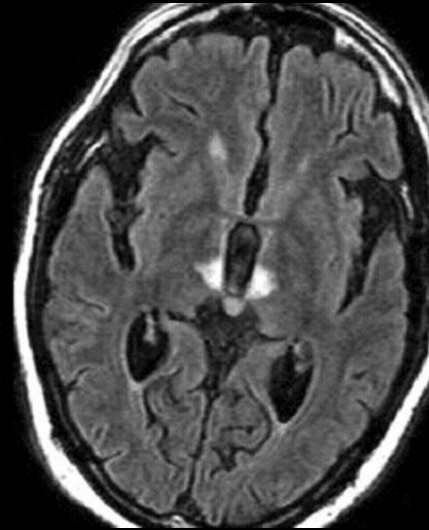


- Real-time quaking-induced conversion (RT-QuIC) assay amplifies detection of $\text{PrP}(\text{CJD})$; CSF or olfactory mucosa brushings; sensitivity ~90-96%, specificity ~99-100%

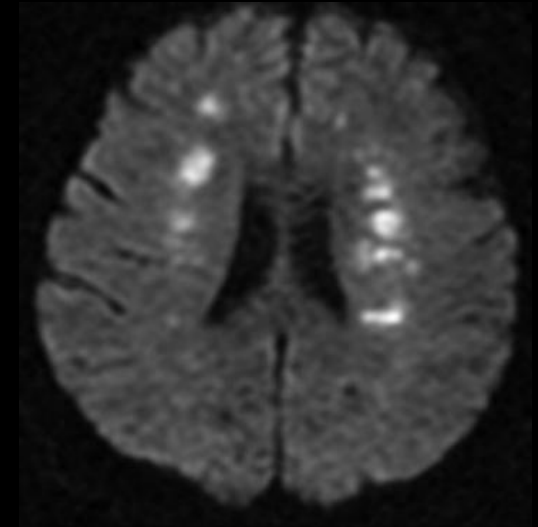
Vascular cognitive impairment



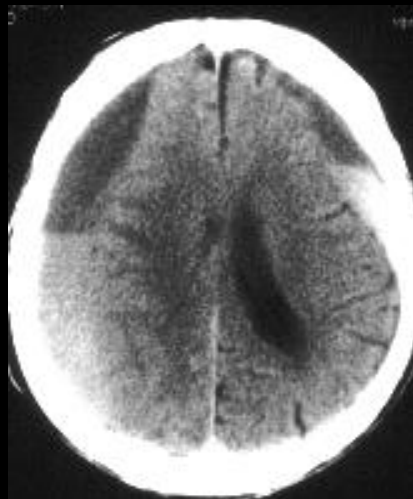
Microvascular disease
"Subcortical" dementia



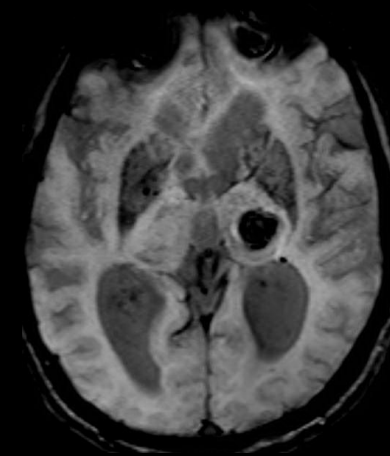
Strategic infarcts



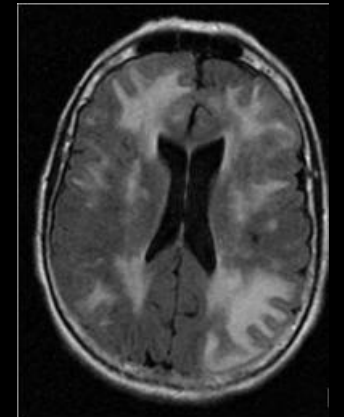
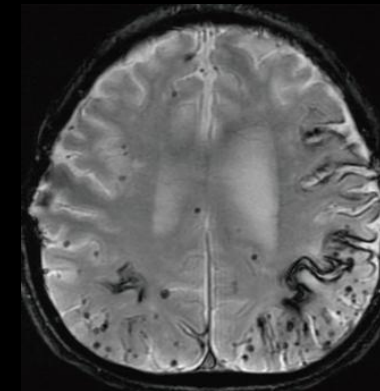
Multifocal/diffuse emboli
(May present as delirium)



Subdural hematomas

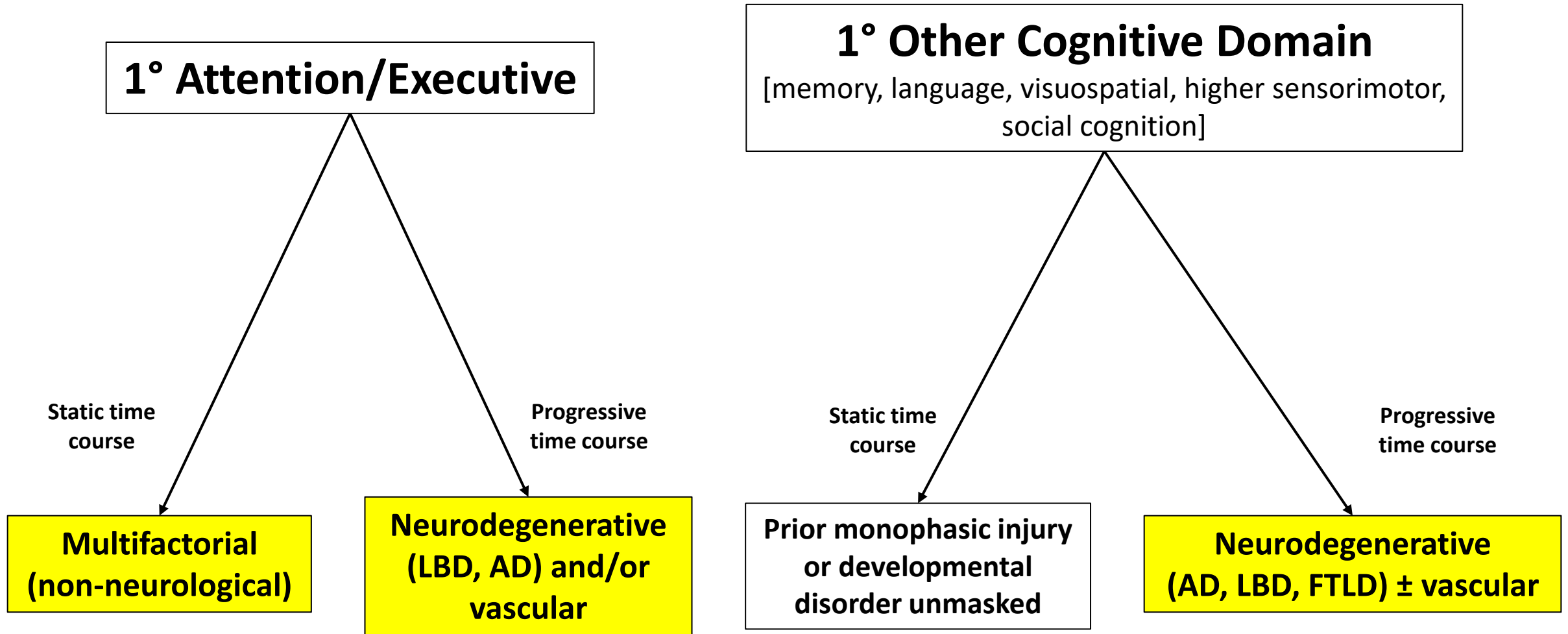


Intraparenchymal hemorrhage (IPH)

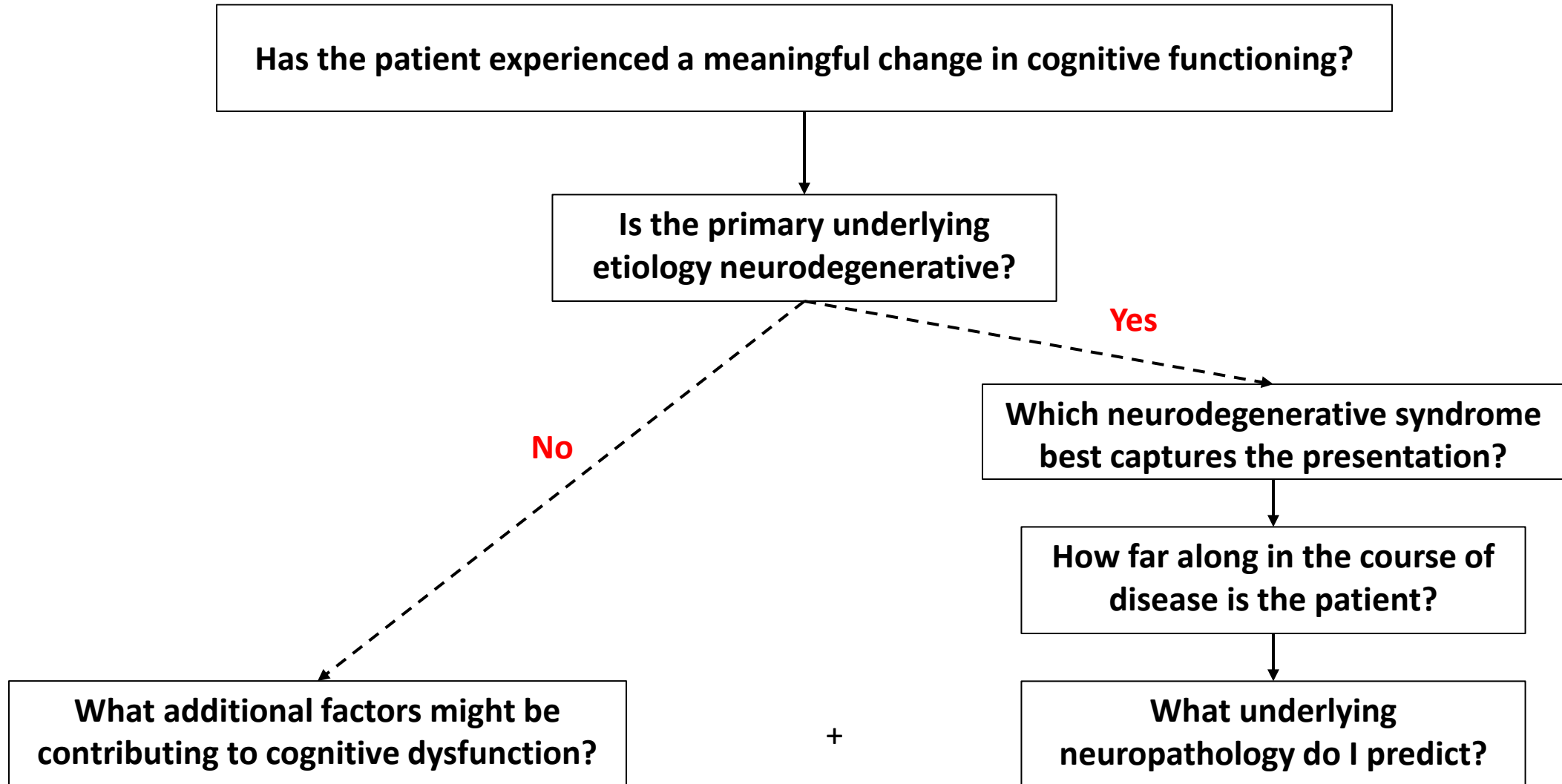


Cerebral amyloid angiopathy (CAA)

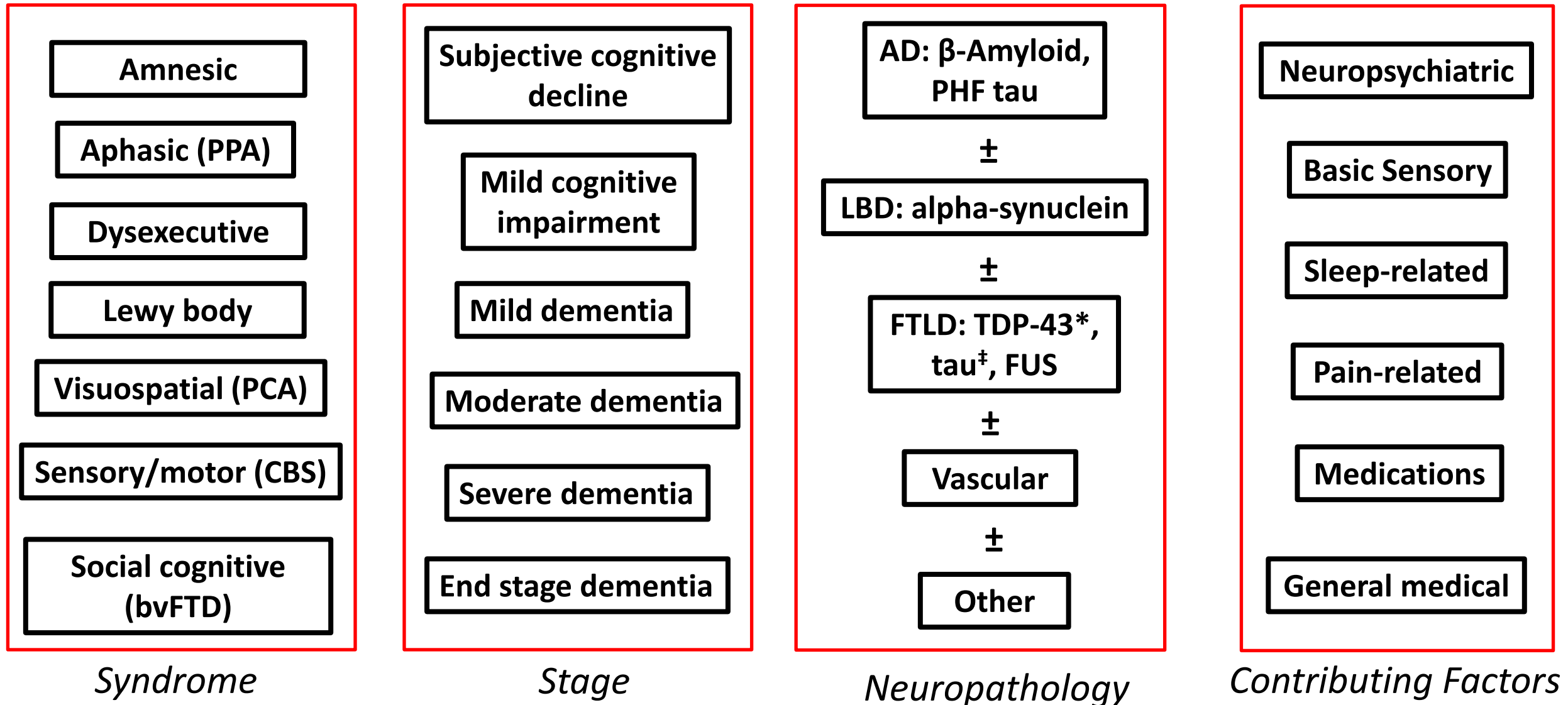
Insidious Onset Cognitive Dysfunction



Initial Assessment: Questions



Formulation: Degenerative Cognitive Disorders

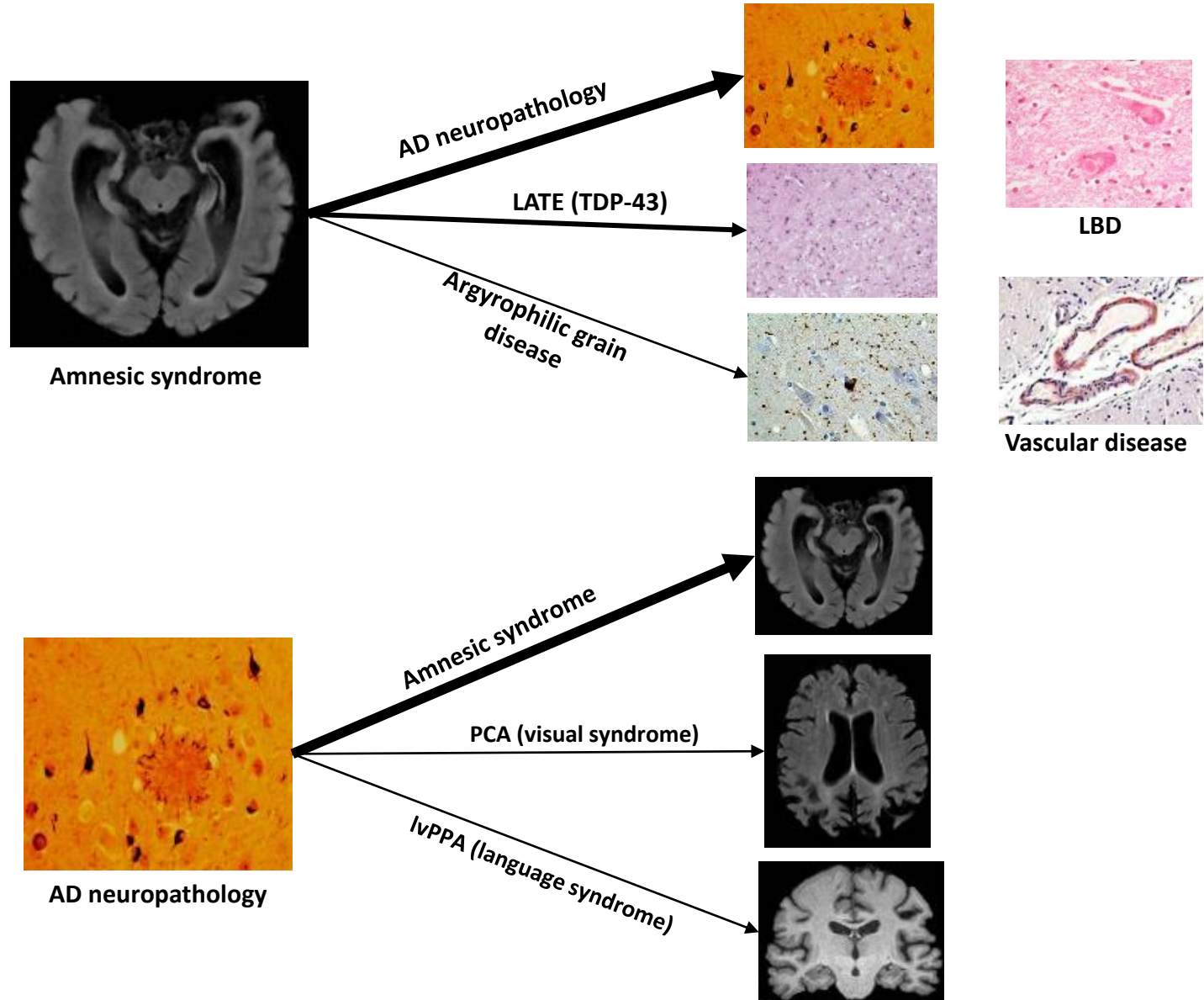


*‡Additional forms include LATE-NC, CTE

Rationale for a 4-tiered formulation

Syndrome ≠ Neuropathology

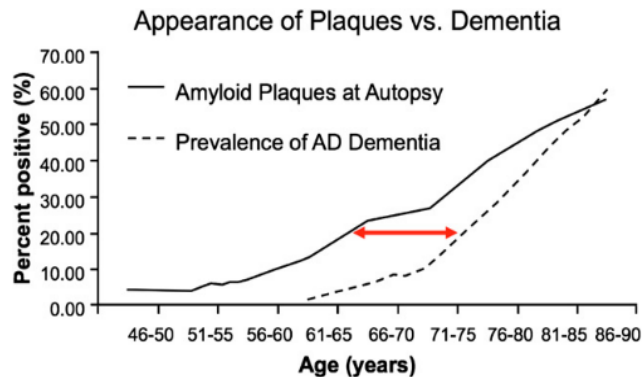
- Large-scale networks subserve cognitive functions.
- Neurodegenerative conditions start focally in vulnerable populations of cells, then progress along the anatomical lines of large-scale networks.
- There are associations between networks and pathologies, but correspondence between syndromes and pathologies is variable and never 1:1.
- Patients can evolve to develop elements of more than one syndrome, secondary syndromes depending on pathology.
- In later stages of illness, more and more networks are compromised and syndromes less distinguishable.
- Multiple (mixed) neuropathologies is the rule, not the exception.



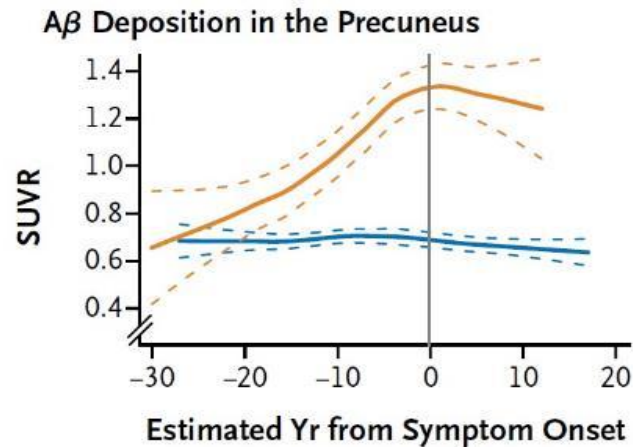
Rationale for a 4-tiered formulation

Syndrome $\neq$ Severity

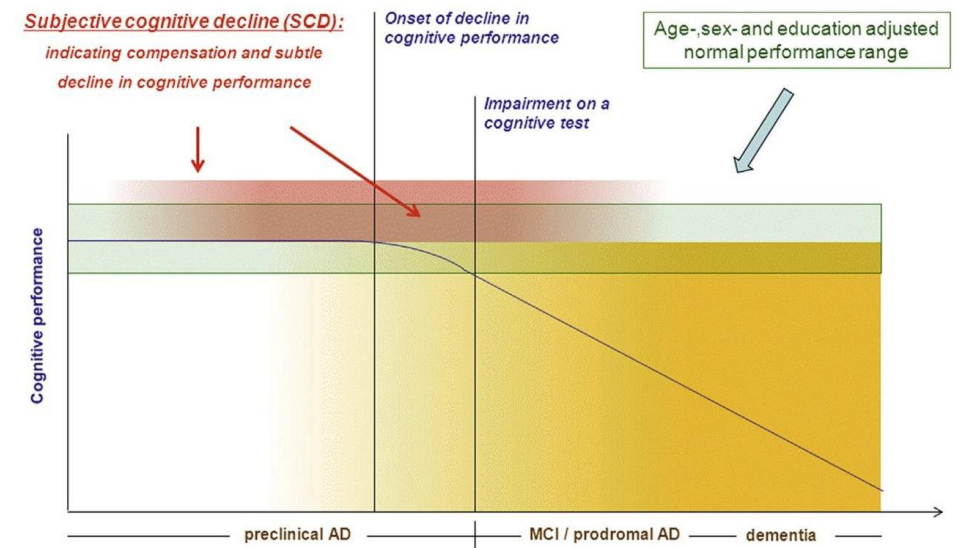
- Dementia and mild cognitive impairment (MCI) are terms denoting stages of illness, not syndromes or etiologies.
- Neurodegenerative conditions are not the only causes of dementia.
- Neurodegenerative processes typically start years prior to onset of symptoms and progress through stages of preclinical illness and MCI prior to dementia.



Mintun and Morris, Washington University



Bateman NEJM 2012

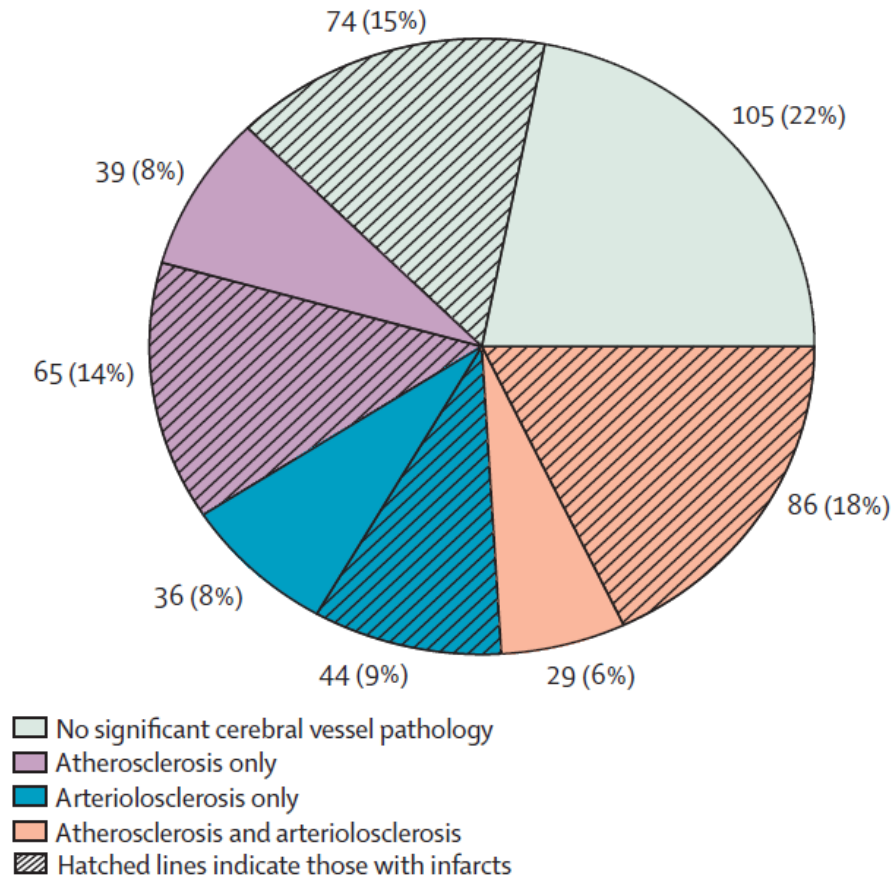


progression of disease pathology and clinical states

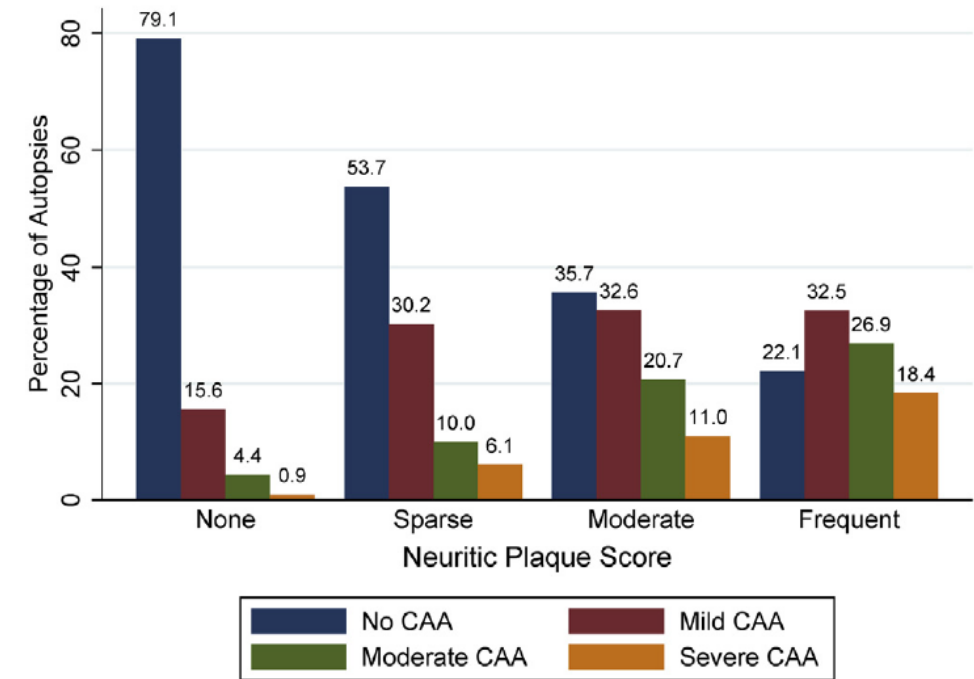
Jessen Alzheimers Dement 2014

Rationale for a 4-tiered formulation

Alzheimer's disease dementia



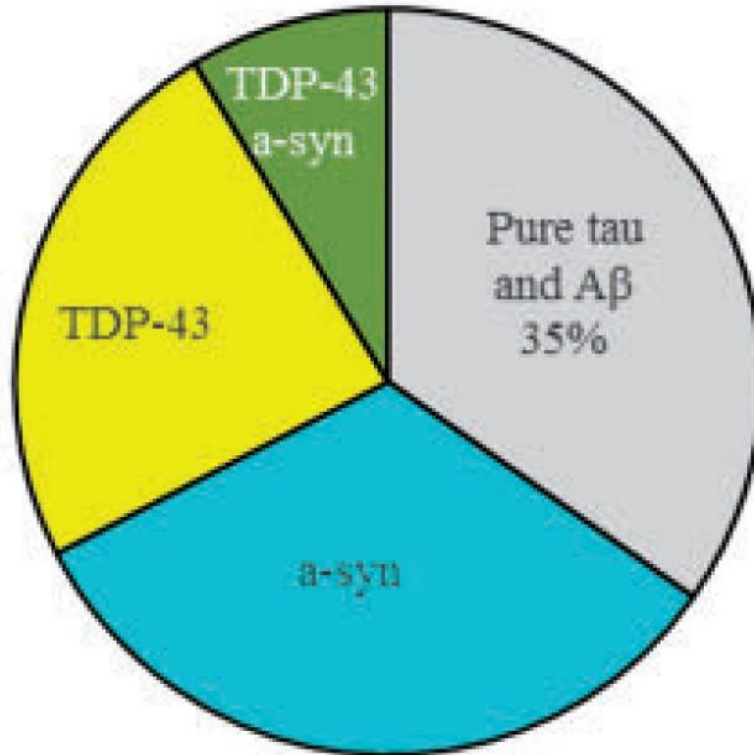
Arvanitakis *Lancet Neurol* 2016;15:934-943



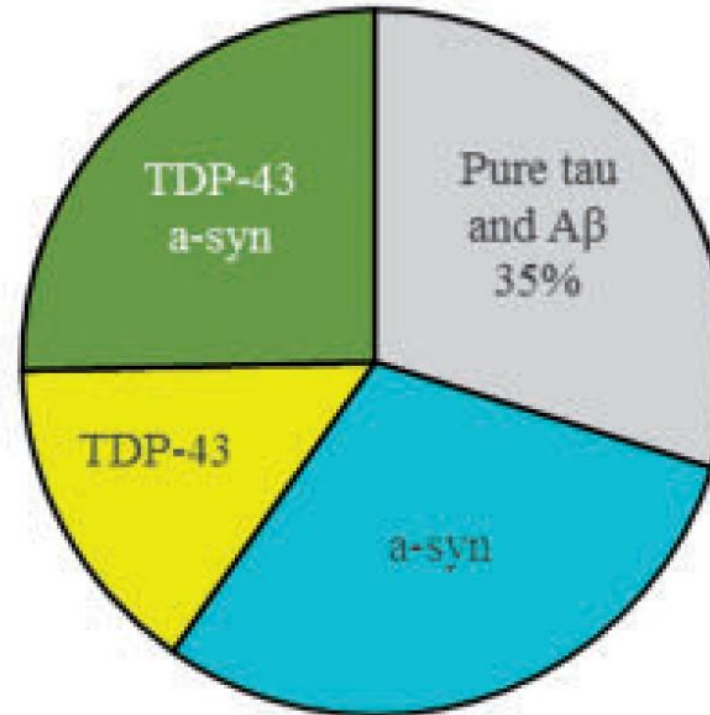
Brenowitz *Neurobiol Aging* 2015;36:2702-8

Rationale for a 4-tiered formulation

***i*AD** (n = 46)



***h*AD** (n = 201)

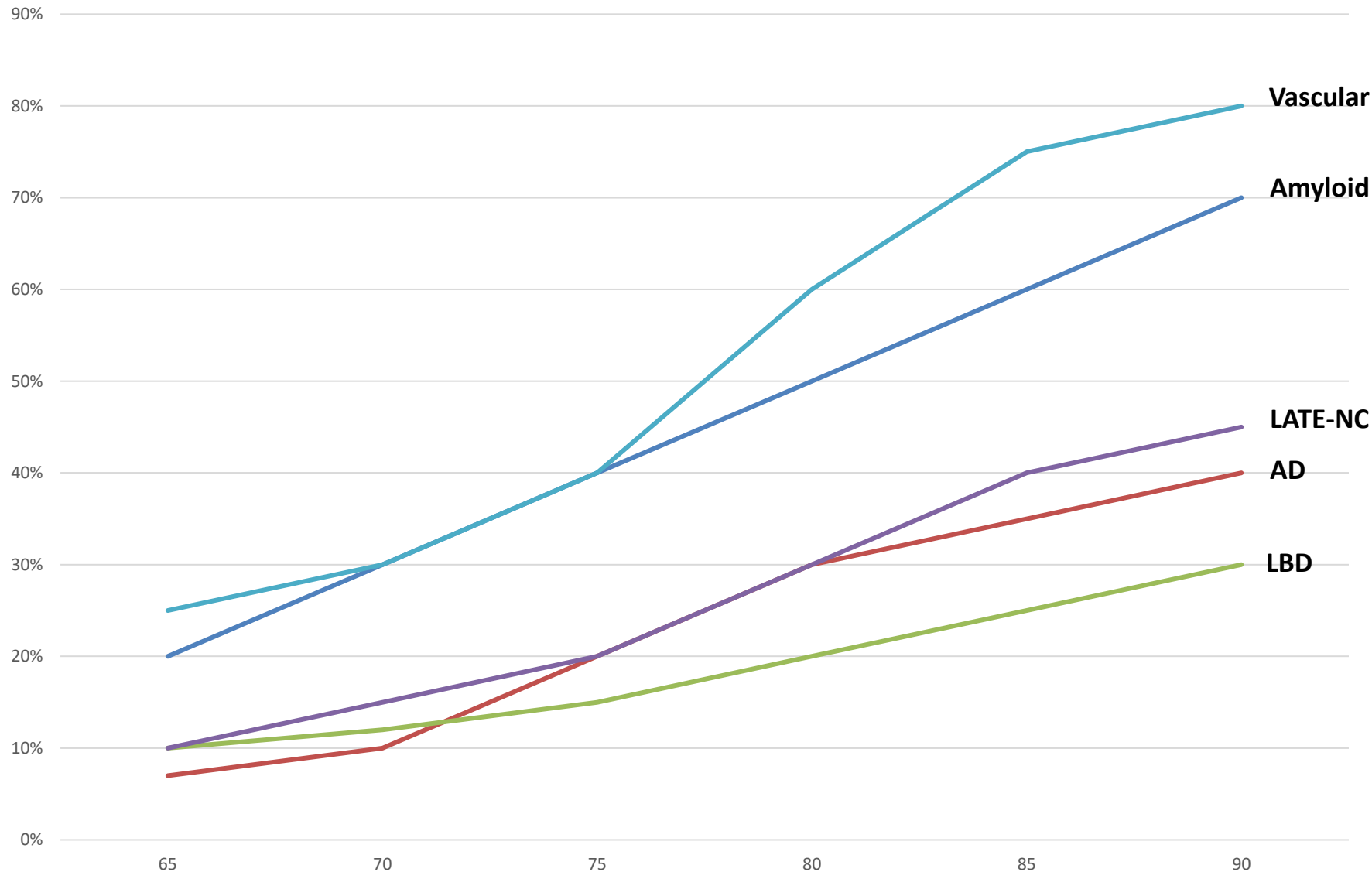


α -synuclein co-pathology: 53%

TDP-43 co-pathology: 39%

Pure AD pathology: 31%

Age-associated prevalence of neuropathologies



Nelson *Acta Neuropathol* 2022;144:27-44.

Wang *Acta Neuropathol* 2022;144:45-57.

Mikhaelenko *Brain* 2025;148:154-67.

Jack *JAMA Neurol* 2019;76:1174-83.

Polivoski *Neurology* 2001;56:1690-6.

Savica *JAMA Neurol* 2018;75:503-9.

Kok *Ann Neurol* 2024;95:843-8.

Wakisaka *Acta Neuropathol* 2023;106:374-82.

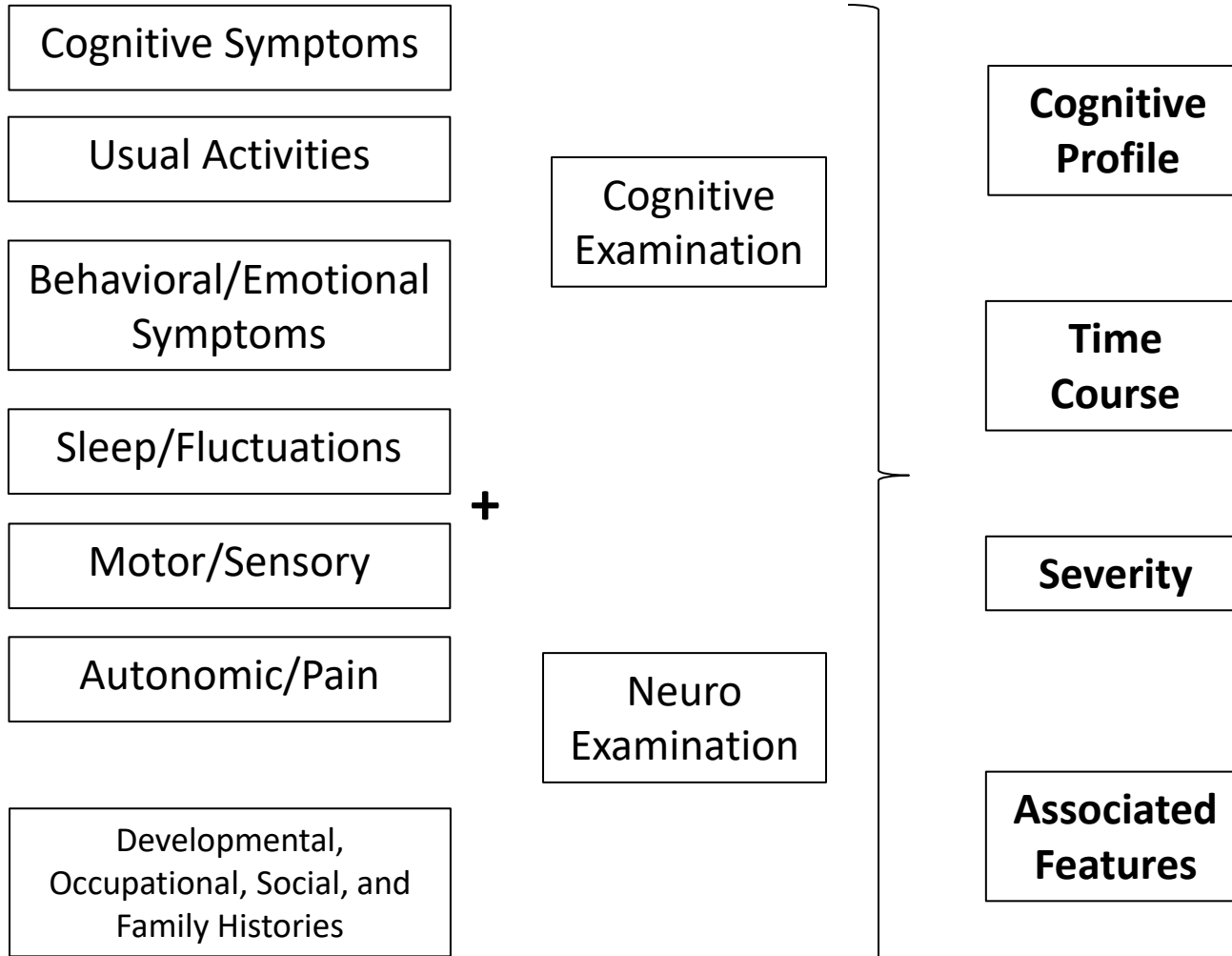
Graff-Radford *Mayo Clin Proc* 2020;95:1195-205.

Nichols *Lancet* 2023;4:e115-25.

Lamar *Stroke* 2022;53:218-27.

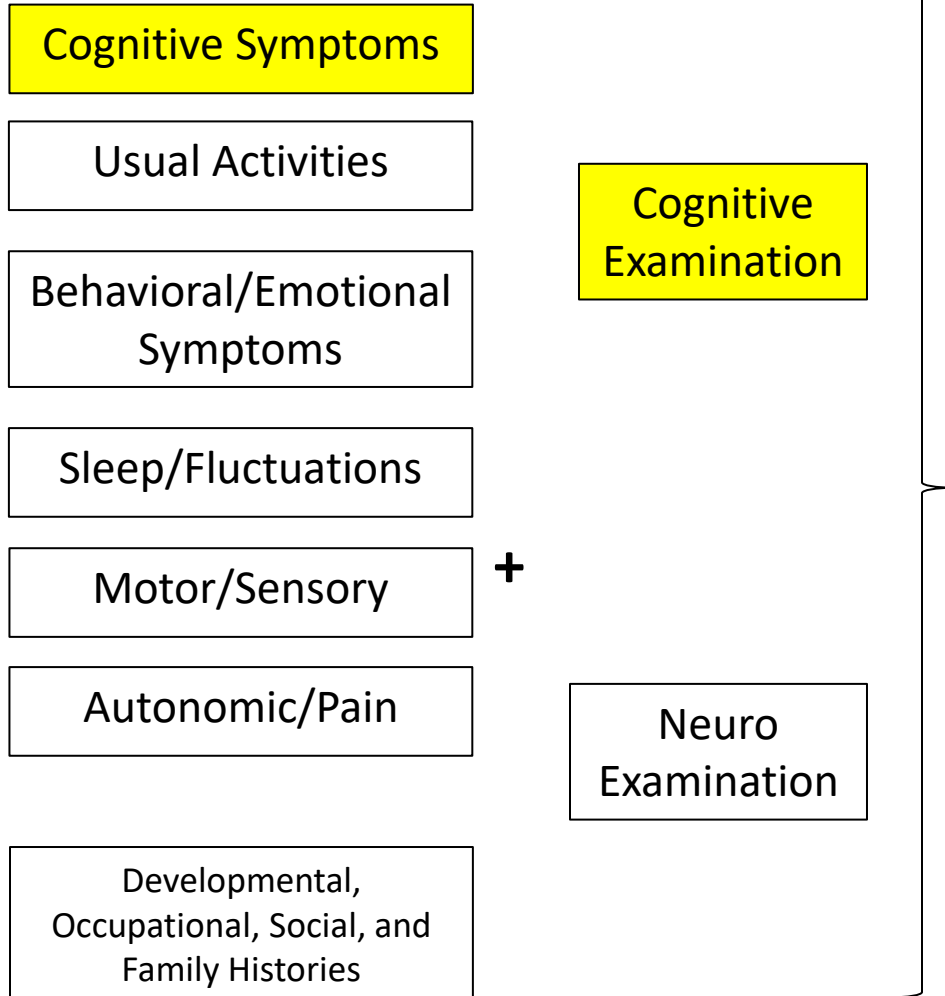
Initial Assessment

History



Initial Assessment

History



Challenges

- **Variable reliability of self-report**
- **Variable reliability of informant report**
- **“Memory” is frequently not (episodic) memory.**
- **Impaired attention can affect processing in other domains.**

Initial Assessment

History

Cognitive Symptoms

Usual Activities

Behavioral/Emotional
Symptoms

Sleep/Fluctuations

Motor/Sensory

Autonomic/Pain

Developmental,
Occupational, Social, and
Family Histories

+

Cognitive
Examination

Neuro
Examination

Cognitive
Profile

Time
Course

Severity

Associated
Features

Challenges

- Evaluations are often cross-sectional.
- Subjective worsening often does not equal objective worsening.
- Standard brief cognitive examination measures may lack sensitivity early in the disease course.

Initial Assessment

History

Cognitive Symptoms

Usual Activities

Behavioral/Emotional
Symptoms

Sleep/Fluctuations

Motor/Sensory

Autonomic/Pain

Developmental,
Occupational, Social, and
Family Histories

+

Cognitive
Examination

Neuro
Examination

Cognitive
Profile

Time
Course

Severity

Associated
Features

Challenges

- **Variable lifestyles/responsibilities at baseline**
- **Imperfect correlation between standard brief cognitive measures and functional severity of illness**

HPI: Usual Activities

Type	Elements/Examples
Advanced ADLs Compromised in MCI	Performance at work Participating in group/community activities Leisure activities (intellectual, social)
Instrumental ADLs Loss of independence in mild dementia	Driving (and other forms of transportation in the community) Taking prescribed medications Managing finances Shopping for groceries and completing errands in the community Completing household chores, preparing meals Using the telephone and computer
Basic ADLs Loss of independence in moderate and severe dementia	Tending to personal hygiene and grooming Bathing and/or showering Dressing Toileting Functional mobility Self-feeding

Initial Assessment

History

Cognitive Symptoms

Usual Activities

Behavioral/Emotional
Symptoms

Sleep/Fluctuations

Motor/Sensory

Autonomic/Pain

+

Developmental,
Occupational, Social, and
Family Histories

Cognitive
Examination

Neuro
Examination

Cognitive
Profile

Time
Course

Severity

Associated
Features

Challenges

- **Variable reliability of self-report**
- **Variable reliability of informant report**
- **Time limitations**

HPI: Other Features

Behavioral/Emotional Symptoms

Neuropsychiatric Inventory-Questionnaire (Kaufer 2000)

Mood; ability to derive pleasure from usual sources; motivation; anxiety; appetite

False beliefs; bipolar-spectrum symptoms; PTSD-spectrum symptoms; disinhibition, obsessive-compulsive or perseverative behaviors, sympathy/empathy, food behaviors

Sleep/Fluctuations

Mayo Sleep Questionnaire (Boeve 2011)
Mayo Fluctuations Scale (Fermin 2004)

Schedule; insomnia (early, middle, late); snoring/breathing; dream enactment; daytime energy level; napping, dozing; episodes of prolonged staring and/or disorganized speech

Restless legs symptoms; other movements in sleep; vivid dreams

Motor/Sensory

Queen Square Visual Hallucinations Inventory (Williams 2008)

Walking, balance; tremors; fine manual dexterity; basic vision, hearing, smell/taste; visual hallucinations, illusions

Speech, swallowing; handwriting; stiffness; eye movements; episodic muscle contractions; fasciculations; extracampine (presence) hallucinations; motion hallucinations; auditory or tactile hallucinations

Autonomic/Pain

NACC/UDS Autonomic Symptoms Checklist

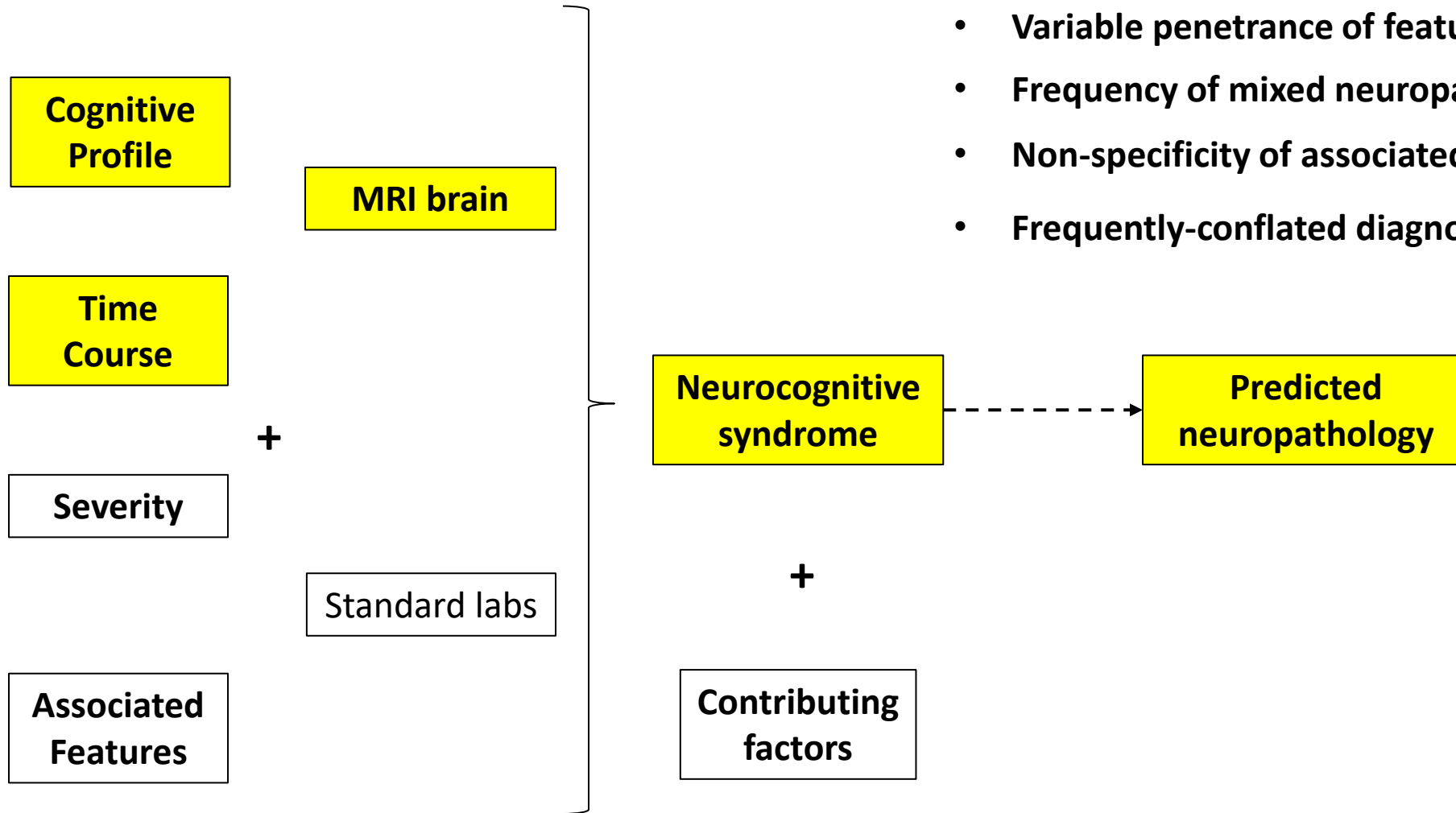
Orthostatic lightheadedness; constipation, loose stools; urinary frequency, urgency; pain

Rhinorrhea; excessive salivation, sweating; temperature regulation; erectile function

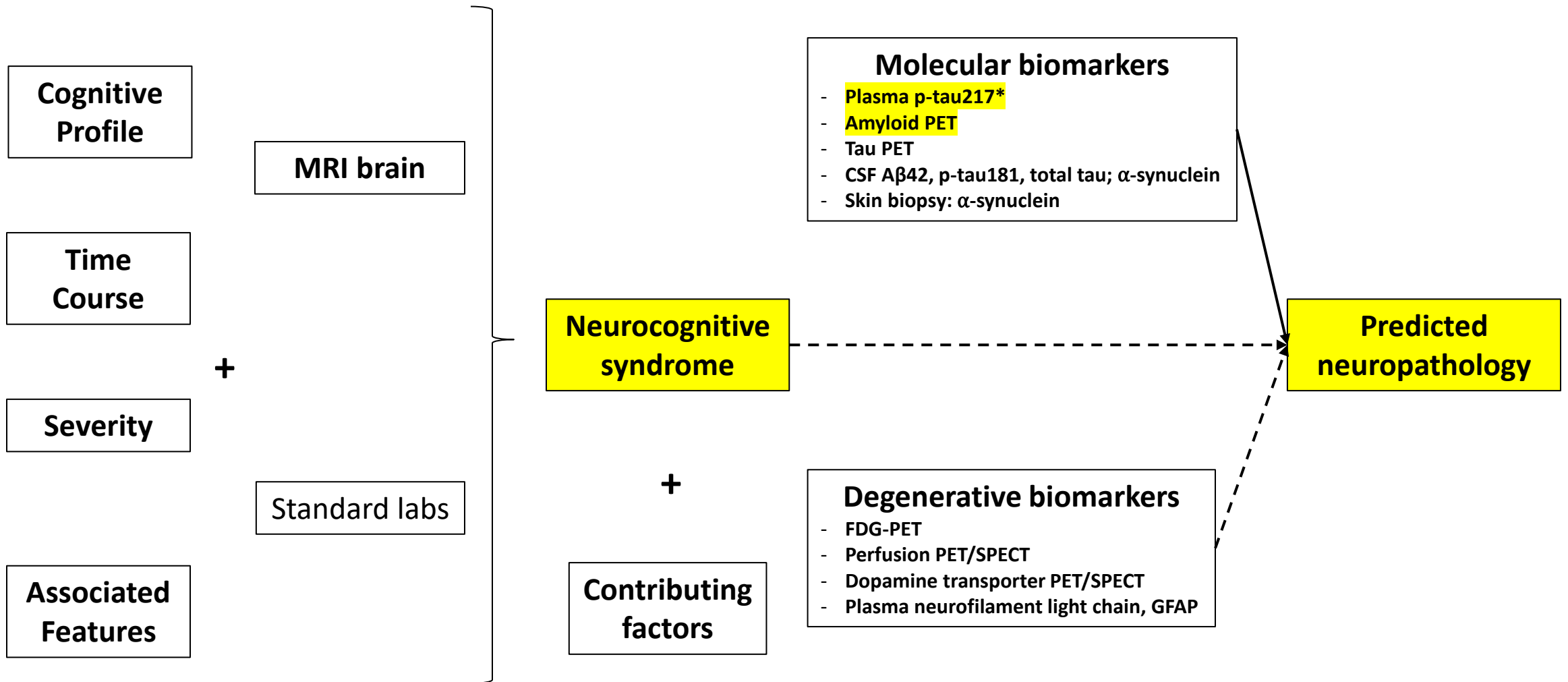
Initial Assessment

Challenges

- Imperfect correlation between syndrome and pathology
- Variable penetrance of features early in the disease course
- Frequency of mixed neuropathology
- Non-specificity of associated vs. contributing factors
- Frequently-conflated diagnostic terms



Initial Assessment



How will the information impact the care of the patient?

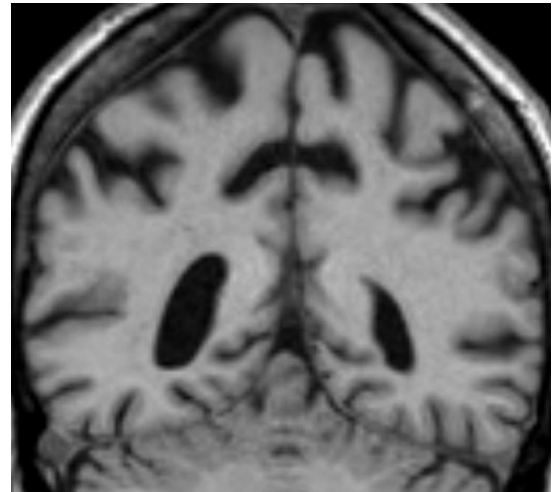
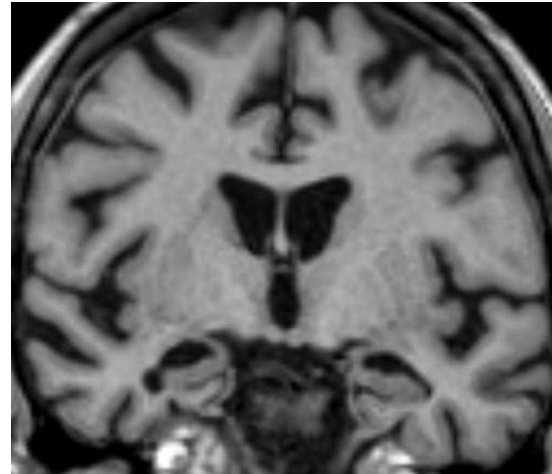
Amnesic MCI/Dementia

History + Examination

Progressive amnesia
± attention/executive,
language, visuospatial

±

**Irritability, anxiety,
apathy, depression,
paranoia, poor insight**

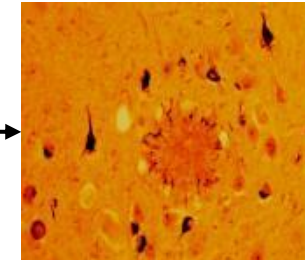


Vascular disease



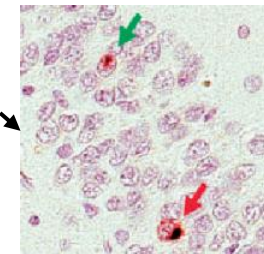
±

Alzheimer disease



±

Limbic-predominant Age-related
TDP-43 Encephalopathy (LATE-NC)



PPV ~80-90% in
late-onset amnesic
dementia

Lewy body MCI/Dementia

History + Examination

**Primary attention/dysexecutive, visuospatial
± memory, language**

+

**Visual hallucinations and/or dream
enactment and/or fluctuations in
attention/alertness and/or parkinsonism**

±

**Anxiety, depression, apathy, delusions,
dysautonomia, anosmia**

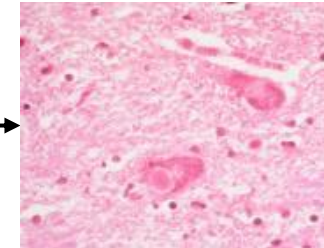
PPV ~80-90% for
diffuse neocortical
Lewy bodies

Vascular Disease



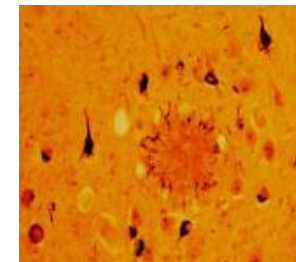
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Lewy body disease



AD co-pathology in
~60-70% of cases

Alzheimer Disease



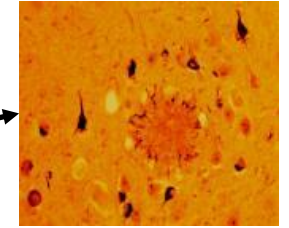
Non-progressive attention/executive dysfunction



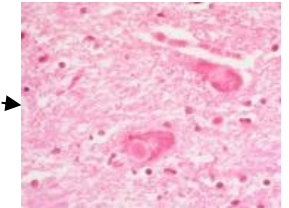
Less Common Neurocognitive Syndromes

AD-spectrum:

- Posterior cortical atrophy
- Logopenic variant PPA
- Dysexecutive/behavioral AD



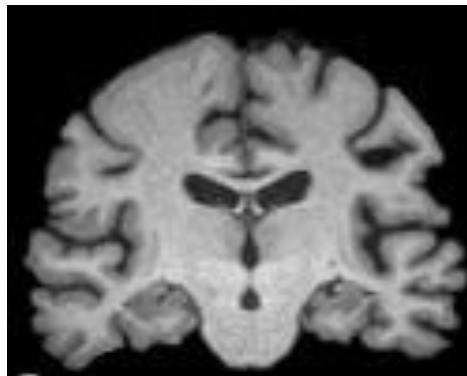
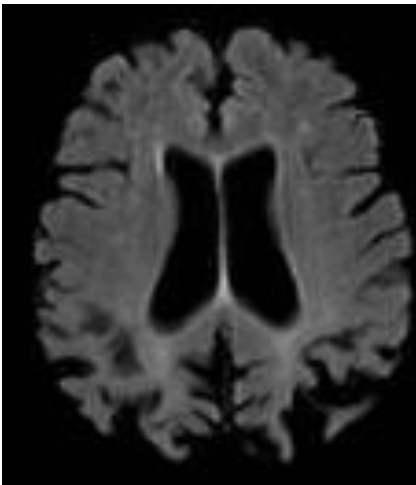
AD



LBD



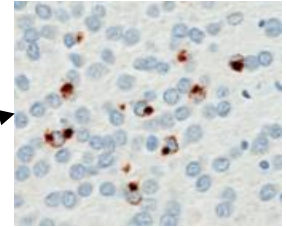
Vascular
disease



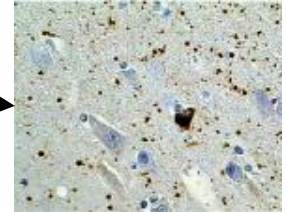
Less Common Neurocognitive Syndromes

FTLD-spectrum:

- Behavioral variant FTD
- Semantic variant PPA
- Nonfluent/agrammatic variant PPA
- Primary progressive apraxia of speech
- Corticobasal syndrome
- PSP syndrome



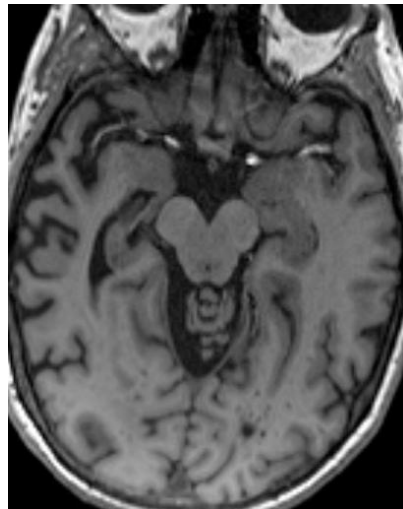
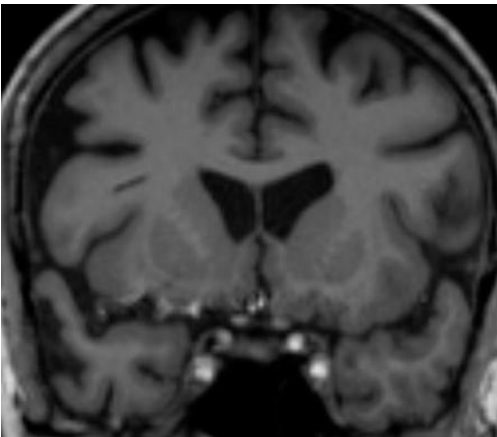
TDP-43



FTLD-tau



Vascular
disease



Atypical Features

- **Rapid progression over days to weeks**
 - Consider admission to the hospital for expedited work-up
 - Prion disease, autoimmune/inflammatory conditions high on the ddx
 - **Non-amnesic cognitive profile**
 - **Young age of onset (< 65 years old)**
- } Frequently FTLD or atypical presentation of AD
- **Family history suggesting autosomal dominant inheritance**
 - Mutations in *APP*, *PSEN1*, *PSEN2* associated with AD
 - Mutations in *C9orf72*, *MAPT*, *GRN* associated with FTLD
 - **Atypical associated medical or neurological symptoms and/or signs**

Accurate diagnosis provides the foundation for longitudinal care

	SCD/ Preclinical	MCI	Mild dementia	Mod. dementia	Sev. dementia
Symptomatic pharmacotherapy (cognition)	-	+/- (off-label)	+	+	+/-
Disease modifying pharmacotherapy	?	+++	++	-	-
Cognitive rehabilitation therapy	-	++	+	-	-
Compensatory strategies, brain health habits, vascular risk factors	+++	++	+	-	-
Safety	-	+	+++	+++	+
Advance directives/planning	+	+++	+++	++	++
Care arrangements/Caregiver support	-	+	++	+++	+++
Clinical therapeutic research (if interested)	+++	+++	++	-	-

Thank you



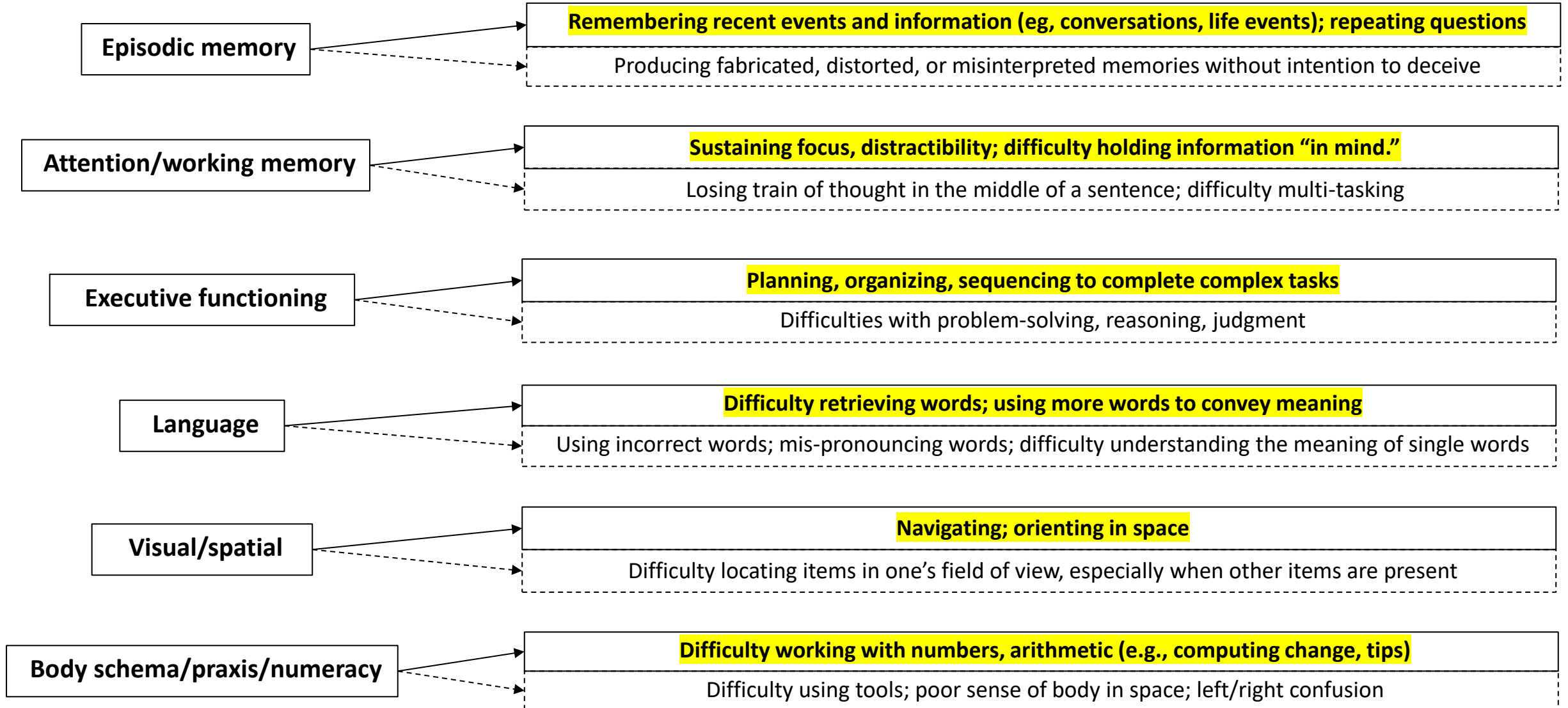
- BWH Center for Alzheimer Research and Treatment:
617-732-8085; BWHmemory@partners.org
- MGH FTD Unit: 617-726-5571; MGHFTDunit@partners.org



Notes/References: Delirium risk medications

- **High risk:**
 - Benzodiazepines
 - Opiates
 - Dihydropyridines (e.g., amlodipine, nifedipine)
 - H1 antagonists
- **Moderate risk:**
 - H2 antagonists
 - Anticholinergics (including but not limited to tricyclic antidepressants)
 - Anti-Parkinsonian agents
 - Corticosteroids
 - Antimuscarinics
- **Some risk:**
 - Selected antimicrobials, anticonvulsants, antineoplastic agents, cardiac drugs, salicylates, NSAIDs, stimulants

HPI: Cognitive Symptoms

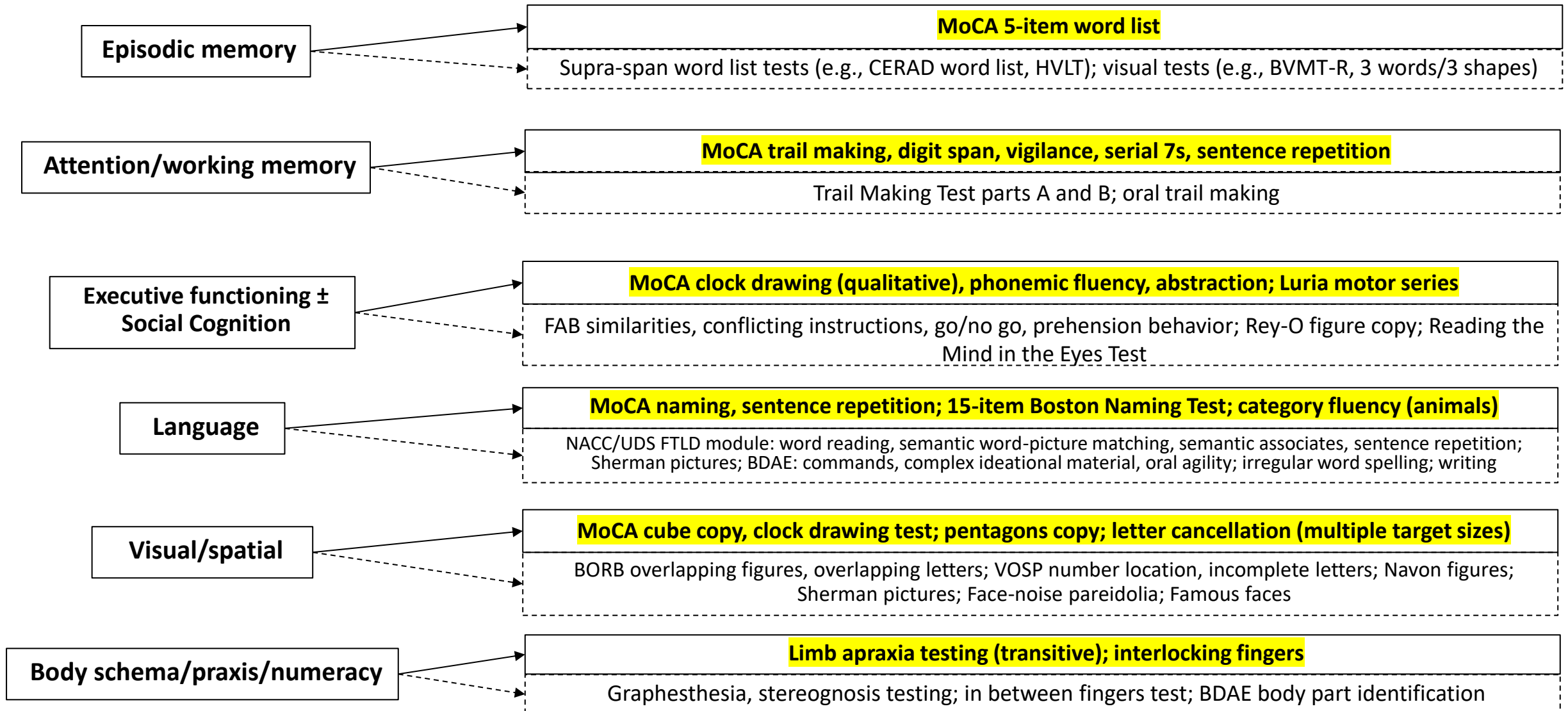


Cognitive Examination: Goals

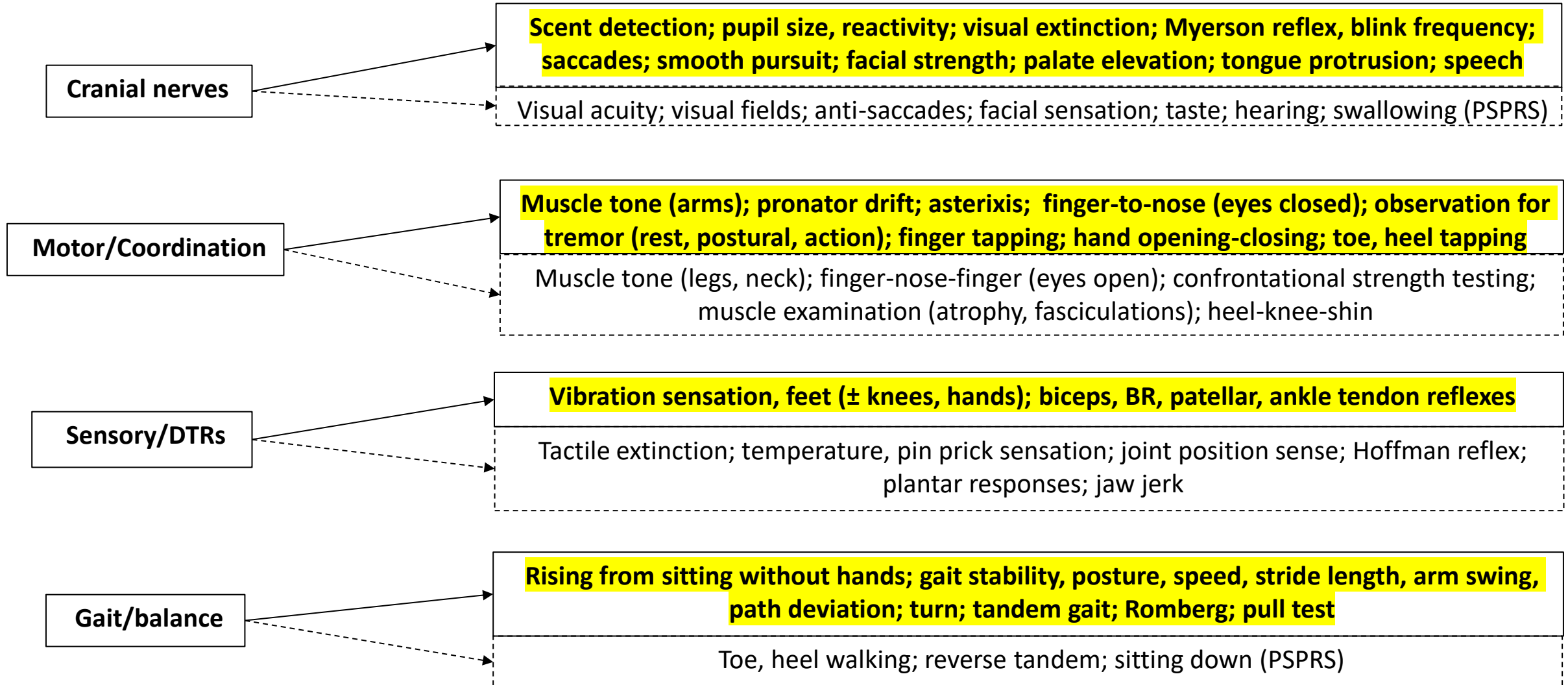
- Goal of determining whether there are **changes above and beyond those attributable to normal aging**, considering baseline intellect/education.
 1. Survey major cognitive domains, testing hypotheses generated from the history (MMSE alone is not sufficient; MoCA, SLUMS, ACE-III may be sufficient in 'typical' cases, particularly if supplemented).
 2. Standard evaluation is geared towards discriminating between common conditions and identifying atypical features, corroborating evidence from history.
 3. Flexible evaluations employed in specific atypical situations (e.g., primary dysfunction in a domain other than memory, attention/executive functioning).
 4. Global scores on selected measures can provide a gauge of severity but should always be considered with functional status.
- Billing possible using Medicare CPT code 96116, neurobehavioral status exam

If time doesn't permit a detailed cognitive examination/interpretation, refer to a specialist (e.g., neuropsychologist or cognitive neurologist) for initial assessment.

Standard and Flexible Cognitive Examination



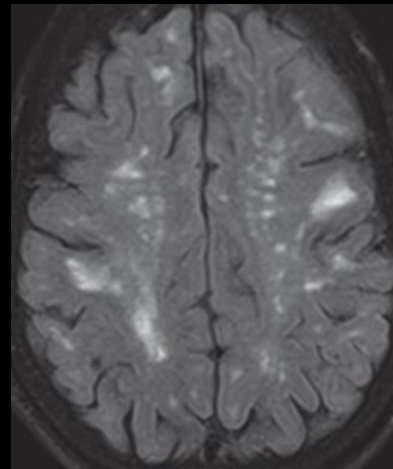
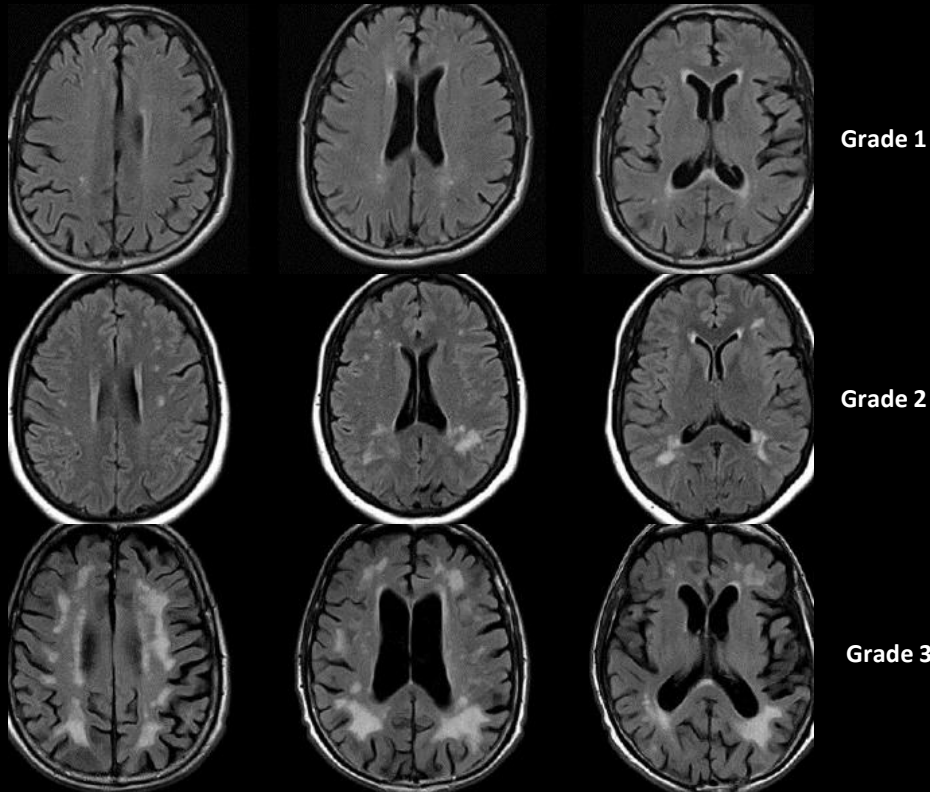
General Neurological Examination



Standard Imaging: Vascular Disease

T2/FLAIR hyperintensities

- Location:
 - Small vessel ischemic disease: periventricular (ependymal loss, ischemic demyelination), subcortical (lipohyalinosis), juxtacortical (arteriole atherosclerosis)
 - Cerebral amyloid angiopathy: centrum semiovale
- Severity (Fazekas Scale):

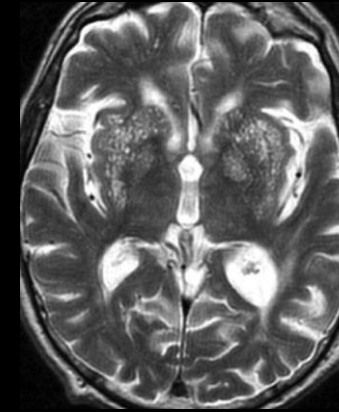


Cerebral amyloid angiopathy

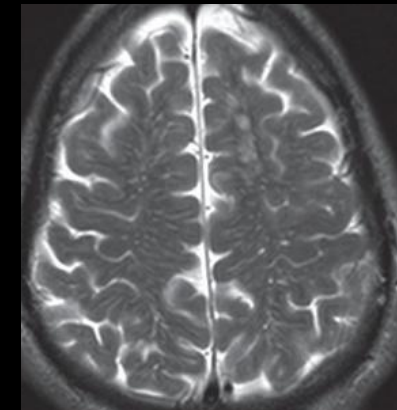
Charidimou *Lancet Neurol* 2022

Dilated perivascular spaces

- Hypertensive vasculopathy: basal ganglia
- Cerebral amyloid angiopathy: centrum semiovale



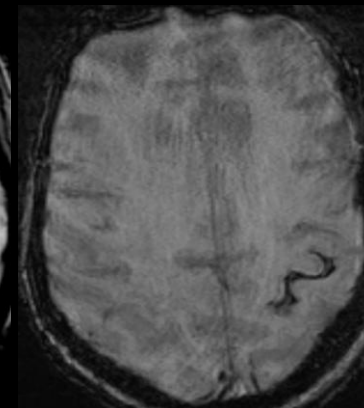
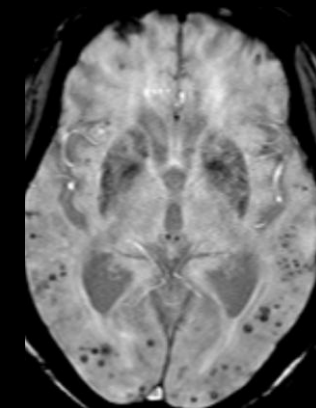
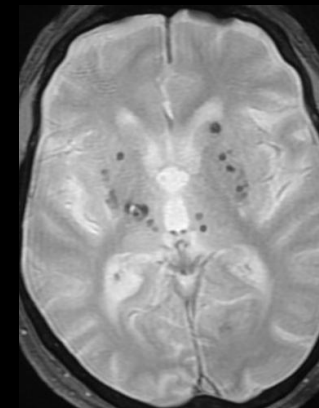
Fergus *Stroke* 2010



Charidimou *Lancet Neurol* 2022

Susceptibility

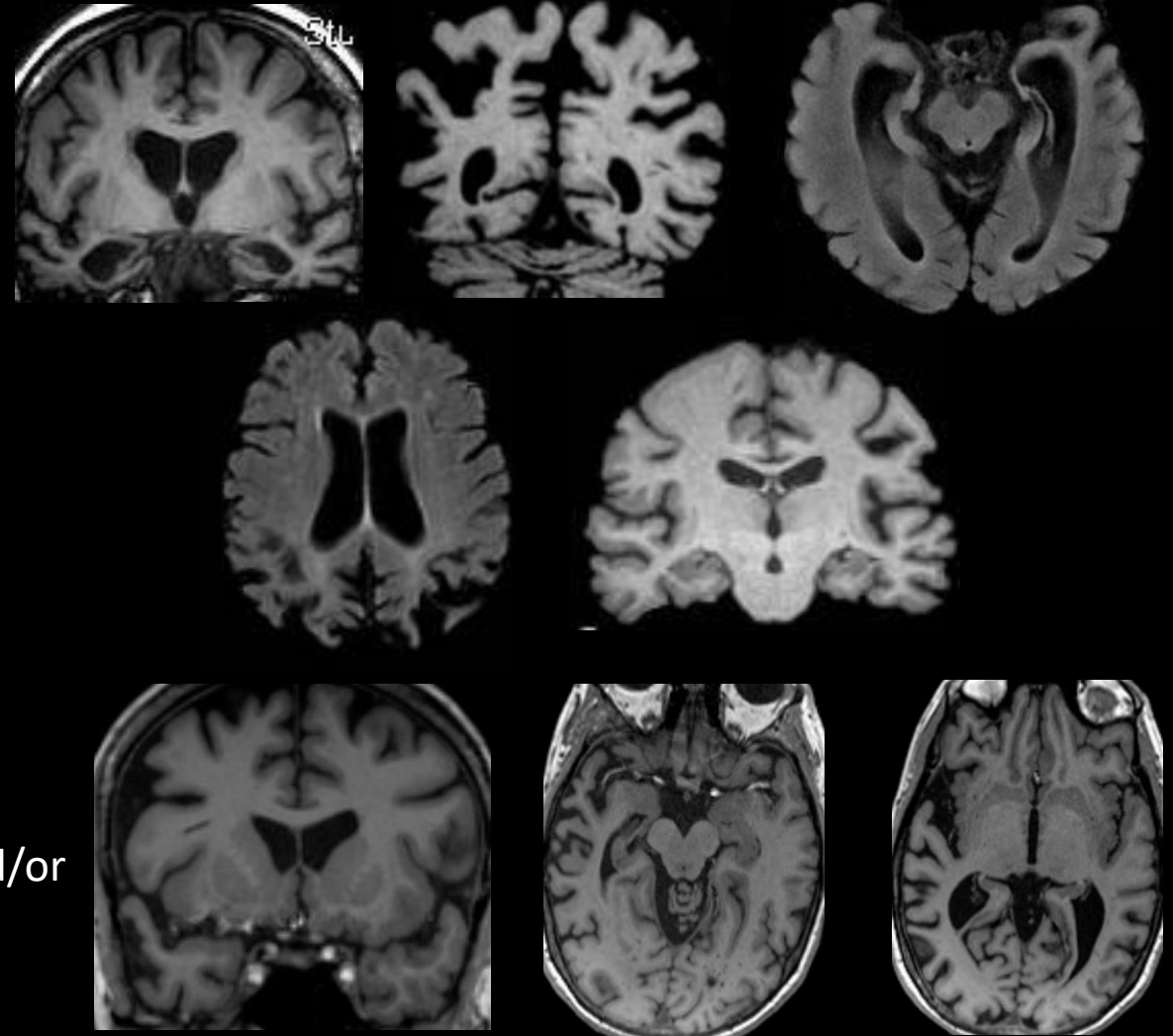
- HTN: subcortical > cortical microhemorrhages
- CAA: cortical microhemorrhages, superficial siderosis



Standard Imaging: Atrophy

High resolution T1 imaging (e.g., SPGR, MPRAGE); 3d reconstruction

- Typical (amnesic) AD: early atrophy in medial temporal, posterolateral temporal, parietal cortices.
- Atypical (non-amnesic) AD: early atrophy in posterolateral temporal, parietal $\pm$ dorsal frontal, occipital cortices
- Atypical, non-AD: early asymmetrical atrophy in frontal and/or temporal lobes



Brief Cognitive Instruments: Comparison

	MoCA	MMSE	ACE-III	SLUMS
Memory	++	+	++	++
Attention/Executive	++	+	+	+
Language	+	+	++	+
Visual/Spatial	++	+	+++	++
Time efficiency	++	+++	+	++
Typical range: MCI*	~19-25	~25-29	~82-89	~21-26
Typical range: Mild dementia*	~14-18	~20-24	~62-81	~12-20

*Most applicable in typical (amnesic) Alzheimer disease

Labs: Non-molecular biomarker

Labs	Utilities/Comments
Standard: Comprehensive metabolic panel, TSH, B12, CBC	Abnormalities are rarely the principal cause of cognitive dysfunction, but may contribute; note eGFR
Also pertinent: Hgb A1c, lipid profile, homocysteine, 25-OH vitamin D, PT, PTT	Consider ordering if recent results not available
Situation-specific (blood): thiamine; treponemal Abs; HIV 1/2 Ab/Ag; rheumatologic labs; autoimmune encephalopathy panel; neurofilament light chain (NfL), GFAP	Informed by social history, general medical illness, time course
Situation-specific (CSF): protein, glucose, cell counts; IgG index, oligoclonal bands; autoimmune encephalopathy panel; cytology, flow cytometry,, IL-10; VDRL, viral PCRs/cultures, bacterial, mycobacterial, fungal stains/cultures, BioFire array; 14-3-3	Informed by history, time course, associated symptoms/signs; usually conducted inpatient

Labs: Molecular Biomarker and Genetic

Labs	Utiliti
CSF: Abeta42, total tau, p-tau181*; alpha-synuclein sAA; IgH gene arrangement, <i>MYD88</i> analysis	Standard of care ; CSF amyloid accuracy to amyloid PET
Plasma p-tau217* , Abeta 42/40*	Recommended for use by sub comprehensive evaluation
Skin biopsy: alpha-synuclein	Useful in Parkinson disease, de system atrophy
Apolipoprotein E (<i>APOE</i>) genotype	Used for risk stratification for immunotherapies; not sensitiv
Genetic: Amyloid Precursor Protein (<i>APP</i>), Presenilin 1 (<i>PSEN1</i>), Presenilin 2 (<i>PSEN2</i>) mutations*	Useful when family history sug of inheritance
Genetic: Progranulin (<i>GRN</i>), Microtubule-associated protein tau (<i>MAPT</i>) mutations; <i>C9orf72</i> repeat expansion*	Most common autosomal dom FTLD

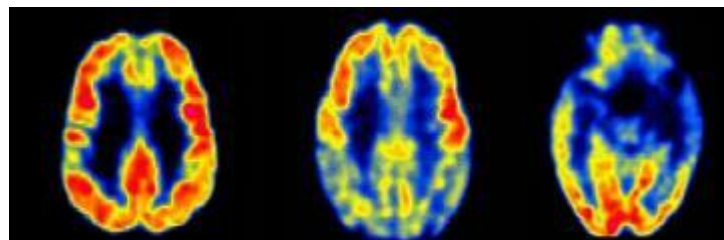
* FDA approved

Appropriate use criteria, CSF AD biomarkers: Shaw *Alzheimers Dement* 2018

Extended Imaging: Neurodegeneration

Modalities	Utilities	Limitations
^{18}F-fluorodeoxyglucose (FDG) PET	- Discriminate syndromic FTD, AD, DLB with some predictive value for underlying neuropathology - Discriminate degenerative from non-degenerative cognitive impairment (negative predictive value)	- Lack of molecular specificity - Potential for false-positive results (e.g., asymptomatic <i>APOE4</i> carriers) - Variable insurance coverage for non-Medicare patients and indications other than discriminating FTD from AD
^{18}F-fluorodopa PET dopamine transporter (DaT) imaging	- Discriminate degenerative from non-degenerative (e.g., vascular, medication-induced) parkinsonism	- Lack of molecular specificity - Does not discriminate between neurodegenerative conditions causing parkinsonism

FDG-PET

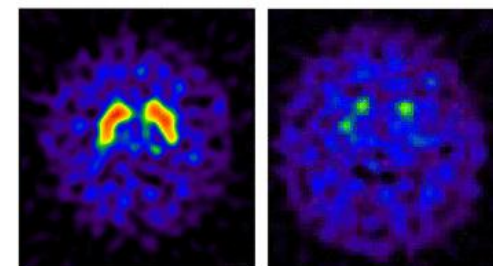


Normal

AD
syndromes

FTD
syndromes

Dopamine transporter imaging

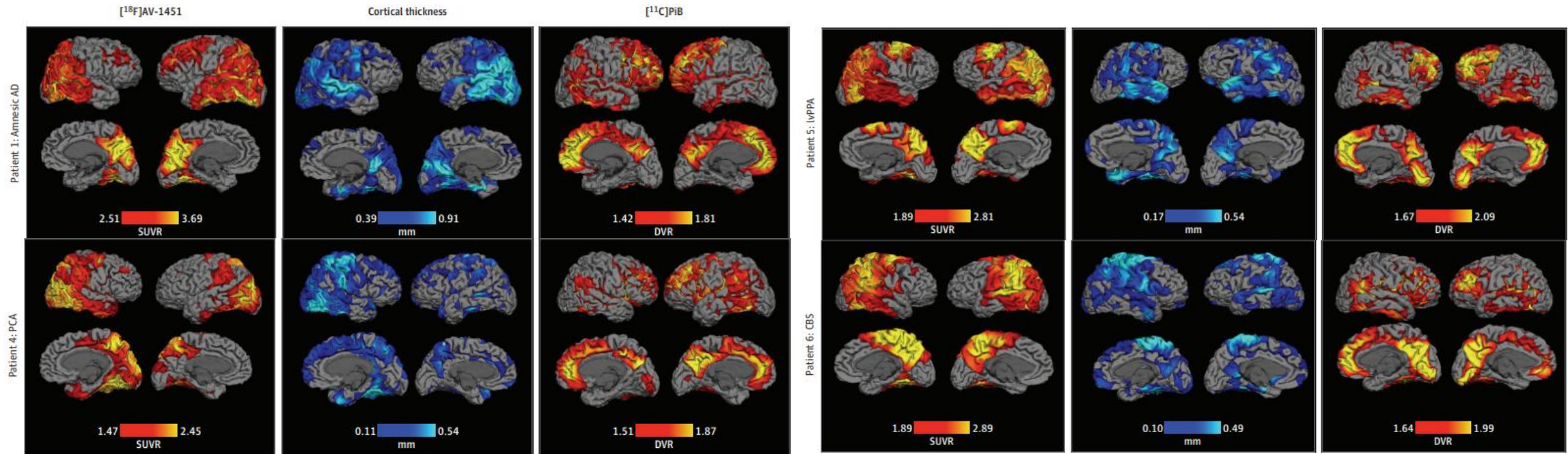


Normal

Degenerative

Extended Imaging: Molecular Pathology

Modalities	Utilities	Limitations
^{18}F-Amyloid PET (florbetapir, florbetaben, flutemetamol)	- Detect fibrillar beta-amyloid with high sensitivity and specificity against neuropathological gold standard). - FDA-approved and covered by Medicare	- Elevated fibrillar beta-amyloid does not equate to a diagnosis of AD (necessary but not sufficient)
^{18}F-Tau PET (Flortaucipir)	- Provides information about not only molecular pathology but also topography, pertinent to disease stage and phenotype. - Substantially elevated uptake in neocortical regions is highly predictive of elevated beta-amyloid.	- FDA-approved, increasingly covered by insurance



Additional Studies, Referrals

Studies, Referrals	Utilities/Comments
Neuropsychology	Establish, clarify cognitive profile; longitudinal time course; time to wait for evaluation is frequently long
Audiology	Hearing correction liberates attention, working memory resources
Optometry/Ophthalmology, Neuro-ophthalmology	Evaluate basic vision in cases of visual hallucinations, higher visual deficits
Electroencephalogram (EEG): routine, long-term ambulatory	Evaluate paroxysmal symptoms; AD increases risk of focal seizures; useful in Lewy body disorders d/t fluctuations and inattention
Polysomnogram ± Sleep Medicine referral	Evaluate for obstructive sleep apnea; RBD
Electromyogram, nerve conduction studies (EMG/NCV) ± Neuromuscular referral	Evaluate for motor neuron disease
Autonomic testing (Autonomic neurology referral)	Evaluate for dysautonomia
Direct admission to inpatient neurology service	Expedite workup for rapidly progressive dementia