BRAIN HEALTH NAVIGATOR LEXICON

OBJECTIVE:

The objective of this lexicon is to provide contemporary nomenclature, terminology, and education for topics relevant to a Brain Health Navigator (BHN) model.

THERE ARE 5 SECTIONS FEATURING TERMS RELATED TO THESE TOPICS:

- **Brain Health Navigator**—roles and responsibilities of BHNs
- **Cognitive Impairment**—various types of cognitive impairment relevant to what a BHN will encounter in the field
- **Factors Contributing to Cognitive Impairment**—a range of causes and conditions that can lead to cognitive impairment
- **Assessments and Evaluations for Cognitive Impairment**—tests measuring cognitive impairment, including cognitive, functional, and behavioral assessments, blood tests, and imaging
- **Treatment for Potential Causes of Cognitive Impairment**—a variety of therapies used in the brain health setting

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TOPIC: BRAIN HEALTH NAVIGATOR

TERM	ABBREVIATION	DEFINITION/EXAMPLE
Brain Health Navigator	BHN	A health care role with responsibilities that include screening and/or case finding, assessment, and triaging of patients exhibiting symptoms of cognitive impairment; coordinating care across the health care system; and directing support for patients and their families The BHN can be an individual or a team fulfilling the various functions Those in this role are required to be health care professionals, including but not limited to: registered nurse, licensed clinical social worker, medical assistant, population health specialists, chronic care managers, etc
Health System Infrastructure		The essential physical, organizational, and technological resources and structures that support the functions of a health care providing organization (health care system [HCS])
Community Outreach		Activities intended to reach and engage communities at risk for cognitive impairment to educate about brain health, and/or connect existing patients with community organizations/partners providing services to meet social needs
Annual Visit, Preventive Care		The annual health check visit, which is a key opportunity for health care professionals (HCPs) to engage patients in conversation about brain health and/or offer cognitive screening for appropriate patients; the term “Annual Wellness Visit” is used specifically for patients on Medicare

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Triage System: **Patient Identification** **Coordination** **Support**		The main BHN responsibilities are **(1) patient identification, (2) coordination, and (3) support** **(1) Patient Identification:** a core responsibility of BHN and the first step of non-medical and medical flags to support a patient with complaints and/or signs of cognitive impairment; may also be through referral from another health care provider **Strategies using risk targeting:** • Comb electronic health record system (EHR) for red flags, such as missed appointments, unfilled Rx, or visits to ED; ensure compliance with usage agreements, which may or may not include EHR-sourced algorithms • Reach out to communities of interest: partner with community-based organizations serving older adults; target local seniors and adult children by reaching out to senior centers, health fairs, and faith-based events; educate first responders as directed by the health system strategy • Conduct community outreach to educate staff at banks (overdrafts, frequent trips to the bank), utilities (missed payments), libraries, and police and fire (wandering, falls requiring EMS) • Initiate MRI/CT, sleep studies (eg, NPSG, HSAT, overnight pulse oximetry per system), standard surveys such as ADLs/IADLs, NPI, STOP-BANG • Triage to neurology for atypical dementias (eg, language predominant), nondiagnostic studies despite cognitive symptoms, cognitive disorders with gait/motor/falls/dysarthria, movement disorders, marked frontal behaviors **Clinic-based strategies:** • Establish regular communication with PCPs to identify patients

		• Conduct initial evaluation: cognitive assessment, medication audit, other assessments, risk factor list, social needs screening, lab/imaging orders per protocol **(2) Coordination:** The 2nd step of the BHN's role with patients • **Engage health care systems (HCS):** develop consistent protocols or screeners, streamline referral process, level-set organization with same EHR and library across HCS, educate primary care and specialty settings on how to identify patients, help track down patients lost in the system, gain understanding of billing and proper documentation • **Partner with patients and families:** identify a primary care partner if possible, coordinate primary and specialty care, help organize care partner and family support • **Triage:** establish a diagnostic clarification process that navigates between primary and specialty care led by BHN and shared support staff champions; expedite neurology referrals for patients considered for treatment as determined by the HSC clinical workflow **(3) Support:** The 3rd step of the BHN role; an ongoing process throughout the journey that includes connecting with various support systems • **Local support:** regional social services such as advocacy, county services, senior living transitions; physical, financial, legal, and emotional support; guidance on local benefits • **National care:** Alzheimer's Association, Eldercare Locator, etc • **Establishing a "Brain Health Plan"** in collaboration with clinicians

Brain Health Plan		A comprehensive, individualized plan that includes coaching on nutrition, physical activity, mental stimulation, social engagement, sleep, stress management, mental health, and treatment of comorbidities (eg, blood pressure, diabetes/blood sugar control, hyperlipidemia, coronary artery disease/congestive heart failure, cerebrovascular disease (CVD), chronic kidney disease, thyroid disorders, vitamin deficiencies)
Brain Health		According to the World Health Organization (WHO), brain health is the state of brain functioning across cognitive, sensory, social-emotional, behavioral, and motor domains allowing a person to realize their full potential over the life course, irrespective of the presence or absence of disorders
Brain Risk Score/Brain Care Score		An individual score composed of 3 categories: physical, lifestyle, and social/emotional; measures what a person is doing to protect their brain and prolong brain health One example is the McCance Tool <https://www.massgeneral.org/neurology/mccance-center/about/brain-care-score>
Medication Audit		Evaluates a patient's current medications to determine whether they might contribute to cognitive impairment (eg, anticholinergic burden (ACB), sedative burden); provides the results and recommendations to an HCP
Electronic Health Record Library	EHR Library	An electronic library of patients' medical information that is maintained over time (eg, Epic) and can be used to determine application of cognitive screening tools, work-up protocols, and establish a pathway supported by billing codes and order sets

TOPIC: COGNITIVE IMPAIRMENT

TERM	ABBREVIATION	DEFINITION/EXAMPLE
Cognitively Unimpaired		Cognitive performance in the nonimpaired range for that individual's age and demographics, defined as not having mild cognitive impairment (MCI) or dementia. Despite non-impairment, brain pathology may still be present
Signs and Symptoms of Cognitive Impairment		Earliest symptoms may include: subjective sense of increased effort or not as sharp in performing usual activities, slow processing, lack of confidence/interest in pursuing new activities and interests, restricting driving/travel to familiar routes and destinations, trouble concentrating, reliance on mnemonics (writing more notes to self), lack of precision with word finding Common symptoms of early cognitive decline include: • Repeating questions and stories • Frequent poor judgment and decision-making • Losing track of time or current season • Difficulty having a conversation • Losing items and being unable to backtrack or locate them Changes in memory, language, and the ability to complete routine tasks may help guide the decision to proceed with a cognitive test
Mild Cognitive Impairment, Mild Neurocognitive Disorder (DSM-V term)	MCI	MCI defines the clinical state between normal aging and dementia, and is defined by 3 key features: 1. Cognitive complaint, decline, or impairment: reported by the patient, care partner, or clinician

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		2. Objective evidence of impairment in ≥1 of the following domains: attention, executive function, visuospatial function, and episodic memory 3. No dementia: performs activities of daily living (ADLs) independently. However, cognitive difficulty may have a mild but detectable impact on more complex activities, either self-reported or corroborated by the care partner
Mild Behavioral Impairment		New onset or exacerbation of stable irritability, anxiety, depression, apathy/lack of motivation, delusions, hallucinations (early in dementia with Lewy bodies [DLB]), and frontal behaviors (extreme apathy, disinhibition, lack of empathy, obsessions/rituals/compulsions) Can present with or without cognitive symptoms
Mild Motor Impairment		Impairment that includes slow walking speed, overall slow movement, balance difficulties, falls
Dementia, Major Neurocognitive Disorder (DSM-V term)		Progressive cognitive impairment that affects several domains and/or neurobehavioral symptoms and interferes with ADLs
Activities of Daily Living	ADLs	The tasks of everyday life, including but not limited to the following: Simple ADLs (SADLs): • Eating • Getting dressed • Bathing • Basic hygiene • Getting in and out of a chair or bed • Using the toilet Instrumental ADLs (IADLs): • Managing household chores • Cooking • Transportation • Managing finances

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		• Using electronics (mobile phone, laptop/computer)
Clinical Staging of the Alzheimer's Disease (AD) Continuum		• Stage 1—Asymptomatic, biomarker evidence only • Stage 2—Transitional decline: mild detectable change, but minimal impact on daily function • Stage 3—Cognitive impairment with early functional impact • Stage 4—Dementia with mild functional impairment • Stage 5—Dementia with moderate functional impairment • Stage 6—Dementia with severe functional impairment
Preclinical AD		Asymptomatic with evidence of AD pathology or cognitively unimpaired with evidence of AD pathology; currently out of scope for routine clinical practice
MCI due to Alzheimer's Disease	MCI due to AD	Mild cognitive symptoms appear but do not interfere with daily activities. The MCI stage of AD is the first symptomatic stage paired with positive pathology
Early AD		Continuum of patients with MCI due to AD and patients diagnosed with mild AD dementia. If "AD" is used, includes pathology
Mild AD dementia		A stage of dementia where cognitive symptoms interfere with some IADLs. If "AD" is used, includes pathology
Moderate AD dementia		A stage of dementia where daily activities (basic ADLs and IADLs) become more difficult, behavior may change, and care partner assistance is required for basic ADLs. If "AD" is used, includes pathology
Severe AD dementia		A stage of dementia with progressive functional and cognitive impairment. Patient becomes completely dependent on caregiver. If "AD" is used, includes pathology
Signs and Symptoms of MCI		Symptoms may include missing appointments; unintentionally repeating comments or stories; trouble retrieving names, places, and common items; difficulty navigating and following instructions; change in mood and/or behavior; confusion regarding date and time; and apathy
Aβ-Positive, Cognitively Normal (CN)		A person with detectable levels of amyloid beta (Aβ) protein in their brain who is not showing signs of cognitive impairment. This can also be referred to as preclinical AD and/or asymptomatic AD according to

		National Institute on Aging and Alzheimer's Association (NIA-AA) guidelines
Dominantly Inherited AD, Autosomal Dominant AD, Familial AD	DIAD, ADAD	AD that is caused by a genetic mutation (eg, *PSEN1*, *PSEN2*) often diagnosed in families in which multiple persons are affected in more than 1 generation; usually early onset; impacts a very small portion of ADRD cases (<2%)
Sporadic AD		Accounts for >95% of AD cases; predominantly later onset; most likely results from a combination of genetic and environmental factors
Typical AD		An early significant and progressive episodic memory deficit that remains dominant in the later stages of the disease
Mixed AD		AD plus other brain pathologies that may contribute independently to cognitive symptoms and signs (eg, cerebrovascular disease, frontal temporal dementia (FTD), Lewy body dementia)
Alzheimer's Disease and Related Dementias	ADRD	An umbrella term for the most common forms of dementia that does not need pathological confirmation for definition
Atypical AD		Less common and well-characterized clinical phenotypes of the disease that occur with Alzheimer's pathology (eg, dysexecutive, primary progressive aphasia, logopenic aphasia, progressive posterior cortical atrophy, cortical basal syndrome) Based on pathological effect centered on other nodes than medial temporal lobe (memory center) in the default network

TOPIC: FACTORS CONTRIBUTING TO COGNITIVE IMPAIRMENT

TERM	ABBREVIATION	DEFINITION/EXAMPLE
Potential Common Causes of MCI/ Identifying Reversible Causes of MCI		MCI can be caused by several factors, but more than 60% of cases are due to AD. Before testing for Aβ pathology, consider ruling out the following: • Medications and medical comorbidities that can contribute to cognitive impairment • Psychiatric disorders • Sleeping issues • Hearing problems • Alcohol or drug abuse • History of head trauma • Evidence of small or large strokes • Fluid buildup in the brain • Epilepsy/seizures • Vitamin B_{12} deficiency • Thyroid diseases There can be concurrent Aβ pathology even with any of these other causes, some of which may be comorbid conditions for AD or other dementias
Amyloid Cascade		Changes in amyloid precursor protein (APP) and/or Aβ homeostasis lead to the aggregation of Aβ and deposition in plaques and protofibrils in the brain and that these events are sufficient to initiate the cascade of pathological abnormalities associated with cognitive decline, dementia, and AD

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Apolipoprotein E (ApoE) ε4	ApoE ε4	The epsilon 4 variant of apolipoprotein (the dominant cholesterol and lipid carrier in the brain), ApoE ε4, is a genetic risk factor for AD
Amyloid Precursor Protein	APP	Mutations in these genes disrupt pathways that are directly involved in amyloid processing
Presenilin-1 Protein	*PSEN1*	Mutations in these genes disrupt pathways that are directly involved in amyloid processing; PSEN1 causes the most severe forms of dominantly inherited AD, with an earlier age of onset and rapid progression
Presenilin-2 Protein	*PSEN2*	Mutations in these genes disrupt pathways that are directly involved in amyloid processing
Tau Aggregation		Tau is a brain-specific protein generated by neurons; disruption of tau metabolism is thought to lead to accumulation of abnormal pTau, which causes tau to aggregate and form neurofibrillary tangles (NFTs) Linked to, but not the same as, tau hyperphosphorylation
Tau Hyperphosphorylation		Excessive attachment of phosphates to a molecule or ion; when tau hyperphosphorylates, it becomes toxic to neurons and can lead to neuronal death and dementia; tau hyperphosphorylation can lead to tau aggregation pTau disrupts neuronal communication and signaling, and is most directly associated with severity and progression of cognitive symptoms
Aβ Deposition, Amyloid Burden		Accumulation of Aβ plaques in the brain, which can be quantified using positron emission tomography (PET) imaging with specific radiotracers. Amyloid deposits can also be found lining blood vessels Aβ deposition has been shown to cause cerebrovascular degeneration, while vascular lesions are directly involved in AD pathogenesis

Neuritic Aβ Plaques		Deposits of Aβ on the outside of neurons in the cerebral cortex; the appearance of these plaques in imaging (amyloid PET) is associated with potential cognitive decline (Term not to be used interchangeably with Aβ deposition)
Impaired Aβ Clearance		When Aβ is not cleared effectively from the brain; an important factor in AD development
Cerebral Amyloid Angiopathy	CAA	When amyloid builds up on arteries in the brain; a contributor to AD. Cerebral amyloid angiopathy can also exist independently of AD. Often leads to both spontaneous (not induced by anti-amyloid drugs) micro and macrohemorrhages
Neurodegeneration, Neuronal Injury		As neurons and connections are lost, the brain's ability to process and store information becomes impaired, resulting in cognitive and/or functional decline, as well as potential behavioral impairment
Blood-Brain Barrier Dysfunction	BBB dysfunction	The blood-brain barrier protects the central nervous system by limiting what substances can enter the brain from blood vessels. Dysfunction of the BBB can be an early indicator of AD
Homozygous ε4 Carriers		People who have inherited ApoE ε4 alleles from both parents
Cognitive Reserve		The brain's ability to make flexible and efficient use of cognitive networks (networks of neuron-to-neuron connections), allowing a person to continue to carry out cognitive tasks despite brain changes
Cognitive Decline		The reduction in mental processes involved in acquiring, processing, storing, and retrieving information: perception, learning, recall, judgment, decision-making, problem-solving, goal-directed behavior, visual-spatial abilities, language, executive function
Traumatic Brain Injury	TBI	An injury to the brain caused by an external force, such as a fall, blow, bump, or penetrating object. TBIs can be a risk factor to later development of AD
Synaptic Failure, Synaptic Loss		One of the key contributors to AD, synaptic failure is a disruption of signaling at the junction between neurons affecting the brain's ability to process information. Synaptic loss occurs before neuronal loss

Neuronal Dysfunction, Neuronal Loss		Loss of function of nerve cells. Neuronal loss begins before AD symptoms present and increases as AD progresses
Dystrophic Neurites		Swollen and/or misshapen neurites (a process or part that extends from the neuron; eg, axons or dendrites)
Neurofibrillary Tangles	NFTs	Aggregates of hyperphosphorylated tau; a biomarker for AD
Proinflammatory Cytokine Expression		Proinflammatory cytokines activate the immune response and promotes inflammation. They are overexpressed in AD and contribute to cognitive decline
Neurotoxicity		When neurotoxic agents (eg, heavy metals, industrial chemicals, and other pollutants) accumulate, there is an increased risk for AD. These agents are linked with Aβ peptide formation and tau hyperphosphorylation, leading to neuronal death
Cerebrovascular Disease	CVD	Conditions that occur when blood flow to the brain is affected. CVD exacerbates cognitive impairment and increases the likelihood of clinical dementia symptoms; known to induce Aβ deposition and affect the age of onset of sporadic AD. Includes periventricular and subcortical white matter changes, lacunar and large vessel infarcts, perivascular spaces, microhemorrhages

TOPIC: ASSESSMENTS AND EVALUATIONS FOR COGNITIVE IMPAIRMENT

TERM	ABBREVIATION	DEFINITION/EXAMPLE
Clinical Dementia Rating–Sum of Boxes	CDR-SB	An assessment of cognitive and functional ability across 6 domains; memory, orientation, judgment/problem-solving, community affairs, home and hobbies, and personal care

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		Often used in research and for regulatory submission; score ranges from 0 to 18 for each of the 6 domains: 0=normal; 0.5–4=very mild dementia; 4.5–9=mild dementia; 9.5–15.5=moderate dementia; 16–18=severe dementia The CDR-SB is the most commonly used score of the CDR Dementia Staging Instrument Not to be confused with the CDR-Glob or Global CDR score Learn more about the CDR <https://knightadrc.wustl.edu/professionals-clinicians/cdr-dementia-staging-instrument/>
Mini-Mental State Examination	MMSE	Measures cognition; scores range from 0 to 30: 0–24=possible cognitive impairment, 25–30=normal MMSE is sensitive to dementia, but lacks the sensitivity to detect MCI Score ranges vary depending on clinician and how the test is administered Access the MMSE <https://cgatoolkit.ca/Uploads/ContentDocuments/MMSE.pdf>
Montreal Cognitive Assessment	MoCA	Cognitive screening instrument; scores range from 0 to 30: ≥26=normal, 18–25=mild cognitive impairment, 10–17=moderate cognitive impairment, <10=severe cognitive impairment Weighted for executive dysfunction and is often more sensitive to early cognitive changes Score ranges vary depending on clinician and how the test is administered Access the MoCA <https://mocacognition.com/>

Mini-Cognitive Assessment Instrument	Mini-Cog	Simple first-stage screen for cognitive impairment; scores range from 0 to 5: 0–2=higher likelihood of cognitive impairment, 3–5=lower likelihood of cognitive impairment Access the Mini-Cog <https://mini-cog.com/download-the-mini-cog-instrument/>
Alzheimer's Disease 8 Dementia Screening Interview	AD8	Caregiver completed questionnaire; can be administered by phone or in-person; scores range from 0 to 8: 0 or 1=normal cognition, 2–8=cognitively impaired Learn more about the AD8 <https://knightadrc.wustl.edu/professionals-clinicians/ad8-instrument/>
Informant Questionnaire on Cognitive Decline in the Elderly	IQCODE	Caregiver completed questionnaire; scores range from 1-5: 1=much improved, 2=improved, 3=not much change, 4=a bit worse, 5=much worse Access the IQCODE <https://nceph.anu.edu.au/research/tools-resources/informant-questionnaire-cognitive-decline-elderly>
Saint Louis University Mental Status	SLUMS	Caregiver completed questionnaire; scores range from 0 to 30: 1–20=dementia, 21–26=mild neurocognitive disorder, 27–30=normal Access SLUMS <https://www.slu.edu/medicine/internal-medicine/geriatric-medicine/aging-successfully/assessment-tools/mental-status-exam.php>
Digital Cognitive Assessment	DCA	Measures cognition; administered digitally using technological solutions
Functional Activities Questionnaire	FAQ	Measures function; consists of 10 questions with scores for each item ranging from 0 to 3: 0=normal, 3=dependent Access FAQ <https://www.alz.org/getmedia/73ea01f0-690b-4ff3-b909-4f0fa9a022ef/functional-activities-questionnaire.pdf>
Lawton Instrumental	Lawton IADL scale	Measures function; scores range from 0 to 8: 0=low function, 8=high function

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Activities of Daily Living		Access Lawton IADL <https://geriatrictoolkit.missouri.edu/funct/Lawton_IADL.pdf>
Amsterdam Instrumental Activities of Daily Living	Amsterdam IADL	Measures function; scores range from 20 to 80: ≥60=no problems; 50–59=mild problems; 40–49=moderate problems; <40=severe problems in daily functioning Learn about the Amsterdam IADL <https://www.alzheimercentrum.nl/professionals/amsterdam-iadl/>
Functional Assessment Screening Tool	FAST	Measures function; scores range from 1 to 7: 1=no functional or cognitive impairment, 7=total dependence Access FAST <https://ohiofamiliesengage.osu.edu/wp-content/uploads/2021/08/FAST-Tool.pdf>
Neuropsychiatric Inventory Questionnaire	NPI-Q	Measures behavior; rates severity (for patient) and caregiver distress level Item scores range from 1 to 3 for severity: 1=mild, 2=moderate, 3=severe Item scores range from 0 to 5 for caregiver distress: 0=not distressing at all, 1=minimal, 2=mild, 3=moderate, 4=severe, 5=extreme or very severe Access NPI-Q <https://download.lww.com/wolterskluwer_vitalstream_com/permalink/cont/a/cont_21_3_2015_02_26_kaufer_2015-10_sdc2.pdf>
Behavioral Pathology in Alzheimer's Disease Rating Scale	BEHAVE-AD	Measures behavior; scores range from 0 to 4: 0=not present, 1=present, 2=present with an emotional component, 3=present with both emotional and physical components Access BEHAVE-AD <https://www.dementiaresearch.org.au/wp-content/uploads/2016/01/BEHAVE-AD-1.pdf>
Geriatric Depression Scale	GDS	Depression screening instrument; scores range: 0-5=normal, >5 suggests depression. Original GDS is a 30-item “yes/no” self-report questionnaire. Shorter 15-item, 10-item, and 5-item versions have been developed and validated

		Access the GDS (long form) <https://integrationacademy.ahrq.gov/sites/default/files/2020-07/Update%20Geriatric%20Depression%20Scale-30.pdf>
Blood Tests		Blood tests for getting a better understanding of potential pathologies include: • Full blood count • Thyroid-stimulating hormone (TSH) • Blood glucose • Serum B_{12} • Liver function • Renal function Potential comorbid pathologies include: • Hemoglobin A1C • Folate • TSH cascade • CBC with differential
Blood Biomarker Tests	BBM tests	Blood tests used to identify amyloid pathology; BBM tests are not intended as stand-alone diagnostic tests and should be integrated with patient history, brain imaging, routine laboratory tests, and other tests as appropriate Blood biomarkers also include neurofilament light chain (NfL), which is not specific for amyloid pathology Blood-based biomarkers also relate to stage of pathology: Aβ 42/40, pTau 181, pTau 231, pTau 217, MTBR
Amyloid Positron Emission Tomography	Amyloid PET	A noninvasive test that allows direct visualization of amyloid plaques in the brain using specific radiotracers; demonstrated high sensitivity and specificity in patients with confirmed AD

Lumbar Puncture, Cerebrospinal Fluid Testing	CSF testing	A procedure to withdraw cerebrospinal fluids with a long, hollow needle in order to diagnose certain conditions; may detect AD pathology earlier than neuroimaging biomarkers
Neuropsychological Evaluation		A comprehensive multi-domain cognitive evaluation involving standardized measures of memory, language, visuospatial attention, executive functioning, mood, and behavior, with appropriate normative adjustments for age, education, and other demographics This evaluation can help provide a more accurate diagnostic picture, clarifying interpretation of screening scores for individuals who may fall out of the normal range based on education level, demographics, psychiatric conditions, motor/sensory conditions, or multilingualism The test can also help inform multi-domain cognitive phenotyping for improved diagnostic accuracy

TOPIC: TREATMENT FOR POTENTIAL CAUSES OF COGNITIVE IMPAIRMENT

TERM	ABBREVIATION	DEFINITION/EXAMPLE
Disease-modifying Therapy	DMT	Treatments that aim to slow or reverse the disease progression
Anti-Amyloid Therapy	AAT	A type of treatment for AD that targets the accumulation of amyloid beta protein in the brain, either as plaques or as earlier forms such as a protofibrils
Monoclonal Antibody	mAB	A laboratory-derived antibody against a single target used therapeutically in many conditions, including AD. In AD, mABs have been developed to target amyloid proteins (ie, anti-amyloid treatments)
Acetylcholinesterase Inhibitors	AChEIs	Used to slow progression of dementia and improve cognitive function, eg, Aricept (donepezil), Exelon (rivastigmine), Razadyne (galantamine) Cholinesterase inhibitors block the enzyme responsible for the breakdown of acetylcholine; by indirectly increasing levels of available acetylcholine in the brain, cognition is thought to be improved These agents only show symptomatic improvement and don't target the pathology of disease. They also have efficacy for a few months, and aren't effective long-term Contraindicated for FTD
Atypical Antipsychotics		Prescribed for agitation in AD dementia (eg, brexpiprazole)
N-methyl-D-aspartate Receptor Antagonists	NMDA	Indicated for treatment of moderate to severe dementia of the Alzheimer's type (eg, memantine)
Antihypertensives		Used to decrease risk of dementia in those with hypertension; also used to reduce risk of comorbid cerebrovascular contributions to those who already have neurodegenerative disease

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