

Subjective cognitive impairment: When to be concerned about ‘senior moments’

Thorough evaluation can differentiate benign memory problems from dementia

Ms. F, age 66, requests genetic testing because she is concerned about mild memory difficulties, such as forgetting names and where she puts her keys or checkbook, and fears she may be developing Alzheimer’s disease (AD). Her mother and sister were diagnosed with AD in their early 60s. Ms. F has 20 years of education and reports no problems with driving, managing her finances, remembering to take her medications, or maintaining social activities, which her husband confirms.

Detailed questioning about anxiety and depressive symptoms reveals substantial worries about future cognitive decline and some concerns about her finances and her husband’s health. Ms. F says she occasionally feels down and has low energy but denies other depressive symptoms. She reports no sleep disturbances—including snoring and daytime sleepiness, which could indicate obstructive sleep apnea—which her husband confirms. Ms. F takes levothyroxine for hypothyroidism, atenolol for hypertension, aspirin and clopidogrel for coronary artery disease, and atorvastatin for hyperlipidemia. In addition, she provides a long list of over-the-counter (OTC) supplements—ginkgo, huperzine, ginseng, phosphatidylserine, B1, B12, folate, vitamin D, alpha-lipoic acid, and vinpocetine—that she takes to “protect” her brain from AD.

Subjective cognitive impairment (SCI) in older persons is a common condition with a largely unclear prognosis. Many older adults (age ≥ 65) express concern about mild cognitive problems—“senior moments”—such as word-finding difficulties and forgetfulness.¹ Individuals may wonder if walking into a room only



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Subjective cognitive impairment

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Patients with SCI do not show objective evidence of cognitive impairment and their cognitive problems cause no functional decline

Table 1

Why SCI should be taken seriously

SCI may create emotional distress because patients are aware of decline in their 'mental sharpness'
SCI patients might consume unnecessary and potentially harmful OTC supplements touted to promote memory
Patients might limit their driving and financial management to avoid making mistakes
SCI might impair medication adherence ²
SCI may be an early sign of dementia ³
Patients' worry about their self-perceived memory loss might predict dementia ⁴
SCI may predict nursing home placement ⁵
Addressing SCI gives health care providers an opportunity to address anxiety or depression that often accompany SCI
Evaluation of potential causes of SCI may uncover reversible conditions that can be treated
OTC: over-the-counter; SCI: subjective cognitive impairment

to forget why might be the first sign of dementia. Some older adults try to counteract these memory problems by engaging in brain exercises—including costly computer games—and taking OTC "brain-enhancing" vitamins, herbal remedies, and other supplements.

Although some clinicians may view SCI as benign, that is not always true (Table 1).²⁻⁵ This article discusses the clinical significance of these mild cognitive complaints by examining:

- age-related cognitive decline (ARCD)
- SCI
- how SCI can be differentiated from more serious conditions, such as mild cognitive impairment (MCI) and early stages of AD and other dementias.

We also will discuss assessing and treating cognitive complaints. Although distinctions between SCI and ARCD may be controversial, evidence suggests clinicians need to adopt a more nuanced clinical approach.

'Normal' cognitive decline

ARCD is subtle decline in cognitive abilities, such as episodic memory, attention,

and time needed to complete complex activities.^{6,7} Individuals with ARCD might not have subjective memory complaints or objective cognitive deficits, and their ability to live independently may not be compromised.⁷ The degree of decline in ARCD may be smaller than previously thought.⁸ Park⁹ summarizes 4 main mechanisms thought to underlie age-related declines in cognition:

- reduced speed of processing
- decreased working memory capabilities
- declining inhibitory control (eg, impaired complex attentional capabilities)
- sensory changes (eg, visual and auditory deficits).

ARCD traditionally is thought to result from predictable changes in the brain associated with aging, such as reduced brain volume in the hippocampus and frontal lobes, loss of myelin, loss of synapses, and cytoskeletal changes.⁷ However, not all older adults experience ARCD. Some remain highly functional in their later years and continue to actively engage in life well into very old age.^{6,9}

Subjective cognitive impairment

One-quarter to one-half of community-dwelling older adults report subjective cognitive complaints, such as forgetfulness and word-finding difficulties.¹⁰ Patients with SCI do not show objective evidence of cognitive impairment on neuropsychological tests and their cognitive problems cause no functional decline.¹⁰

Preliminary evidence indicates that SCI may be a harbinger of further cognitive decline. Reisberg et al³ found that compared with patients without SCI, patients with SCI were 4.5 times more likely to develop MCI—cognitive difficulties that can be detected by cognitive tests, but do not cause functional decline—or dementia within 7 years.³ Studies also have suggested that SCI may be a pre-MCI stage of subsequent dementia.¹¹⁻¹³ AD generally has a long (10 to 12 years) and progressive prodromal phase before dementia onset and is characterized by successive emergence of cognitive deficits, memory complaints, depressive symptoms, and functional impairment.¹⁴



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In light of this research, we believe patients with SCI and other risk factors for AD, such as a family history of AD, may be at higher risk of further cognitive and functional decline compared with individuals with ARCD and no AD risk factors. Therefore, patients with SCI and other risk factors for AD (Table 2)¹⁵⁻¹⁹ may benefit from annual follow-up to determine if cognitive problems have progressed to MCI or AD.

SCI may be a response to subclinical alterations in neurobiology—a phenomenon known as reverse causality.²⁰ Biomarkers, such as cerebrospinal fluid levels of β -amyloid and phosphorylated tau, and amyloid imaging using positron emission tomography may help identify AD in SCI patients.²¹ In these patients, SCI is a misnomer because the cognitive impairment is real—not “subjective”—but current tests are not sensitive enough to detect the cognitive decline the patient has recognized. This group of patients should be differentiated from individuals who may perceive typical cognitive aging (ARCD) as pathologic and complain about it. In the future, biomarkers may help differentiate these 2 groups.

Mild cognitive impairment

MCI is similar to SCI because MCI patients may present with complaints of memory decline and other cognitive difficulties²² but neither condition is associated with significant impairment of daily activities.²³ The key difference is that patients with MCI demonstrate impaired performance on objective cognitive tests whereas SCI patients do not.²⁴ In our experience, office-based tests do not reliably differentiate the 2 conditions because many patients with SCI may show mild impairment in tests such as the Mini-Mental State Exam (MMSE)²⁵ but comprehensive neuropsychological testing reveals no objective cognitive deficits. Neuropsychological testing is essential to reliably differentiate SCI from MCI.

The distinction between SCI and MCI is clinically relevant because evidence suggests that MCI patients have a near-

Table 2

Factors that increase SCI patients' risk for dementia

Family history of Alzheimer's disease
Mild behavioral impairment
Slow gait
Depression
Rapid weight loss
Multiple subtle neurologic abnormalities
Vascular disease (eg, peripheral vascular disease, coronary artery disease, cerebrovascular disease)
SCI: subjective cognitive impairment
Source: References 15-19

term risk of developing dementia, particularly AD.^{22,23} In a longitudinal study of 76 individuals with MCI, 12% of patients progressed to AD each year compared with 1% to 2% of healthy older adults.²⁶ Patients with MCI are at increased risk of delirium (especially during hospitalization), falls, medication errors, and difficulty managing their finances.²⁴ Older adults with MCI also have increased mortality compared with older adults with normal cognitive functioning.²² Both SCI and MCI should be differentiated from mild dementia. Common dementias in older adults include:

- AD dementia
- Vascular dementia (may occur with or without AD)
- Lewy body dementia
- Frontotemporal dementia
- Parkinson's disease dementia.

By definition, all dementia types are associated with impaired ability to perform daily activities and cognitive decline.²⁷

Assessing cognitive complaints

Evaluation of older adults' cognitive complaints should begin with a thorough history to elicit symptoms of anxiety, depression, physical complaints, and any associated functional decline; a physical exam; and a comprehensive mental status examination. This initial evaluation should be followed by routine and specific investigations as indicated (Table 3, page 40).^{22,24,28,29}

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Office-based tests such as the MMSE do not reliably differentiate SCI from MCI; neuropsychological testing is essential



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Researchers found no relationship between individuals' perception of their cognitive functioning and performance on testing

Table 3

Investigation of older adults with SCI

Investigation	Rationale
Routine	
Neuropsychological testing	Delineation of cognitive syndromes (SCI vs MCI vs AD*)
Hematology (full blood count)	Screen for anemia
Biochemistry (electrolytes, renal function, liver function, thyroid function, B12, and folate)	Screen for treatable causes of cognitive complaints
For specific indication suggested by history, physical exam, or neuropsychological testing	
Neuroimaging	Generalized and regional imaging (eg, hippocampal atrophy, space occupying lesions)
Electroencephalography	Epilepsy/seizures (especially absence and complex partial)
Cardiac (eg, echocardiography)	May reveal cardiac arrhythmia or sources of emboli
Inflammatory markers (eg, ESR)	Screen for inflammatory processes
Treponemal serology	Tertiary syphilis
*Alzheimer's disease and other dementias	
AD: Alzheimer's disease; ESR: erythrocyte sedimentation rate; MCI: mild cognitive impairment; SCI: subjective cognitive impairment	
Source: References 22,24,28,29	

Table 4

Differential diagnosis of SCI

Cause of cognitive impairment	Potential mechanism
ARCD	Allostatic load, 'wear and tear' from a lifetime of physiological or psychological stresses and adaptations
Anemia	Neuronal hypoxia
Alzheimer's disease	Amyloid and/or tau-mediated neurotoxicity, neuroinflammation
Cerebrovascular disease	Neuronal ischemia and hypoxia, neuroinflammation
Vitamin deficiencies (eg, B1, B12, folate, D)	Impaired neuronal and neurotransmitter function
Inadequate protein intake	Impaired neuronal function
Anticholinergic drug use	Decreased cholinergic neurotransmission
Alcohol use	Direct neurotoxicity and indirect causes such as malnutrition or head injury
Depression, anxiety	Hippocampal dysfunction with or without atrophy
Obstructive sleep apnea	Neuronal hypoxia, neuroinflammation
Head injury	Neuronal and synaptic loss
ARCD: age-related cognitive decline; SCI: subjective cognitive impairment	
Source: References 28,29,31,32	

In a 6-year study of 100 older adults with and without objective evidence of memory decline, both groups showed similar rates of cognitive complaints.³⁰ Also, researchers found no relationship between individuals' perception of their cognitive functioning and performance on neuropsychological testing. Mood, education level, and apolipoprotein E epsilon 4 genotype status also did not correlate

with participants' subjective cognitive complaints. These findings highlight the need for objective test data to determine whether older adults' memory complaints reflect pathologic changes in cognition. After a thorough diagnostic workup, some patients complaining of memory decline will have no detectable evidence of cognitive dysfunction or an identifiable cause. However, others may have identifiable



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Enhancing brain plasticity and neurogenesis requires engaging older adults in demanding sensory, cognitive, and motor activities

Table 5

Strategies to improve memory and maintain cognitive vitality

Strategy	Description
Mindfulness	Focus on 1 task at a time rather than trying to multitask. Research shows that cognition is more efficient in this manner
Cognitive strategies	Use mnemonics (such as ROY G BIV to remember the colors of the rainbow). Make associations for information, such as when meeting someone new, relate their name to someone else you know well. Use cues such as memory notebooks to prompt information recall. Engage in learning new and challenging cognitive activities, such as a new language, a music instrument, or dance. Consider computer-based brain exercises
Rehearsal	Practice information you want to remember, such as repeating the information several times or writing it down
Be patient	Getting frustrated when you have memory difficulties makes it more challenging to remember information
Exercise (mental and physical)	Engage in mental activities, such as reading and crossword puzzles. Do something that you are interested in, rather than making it a chore. Research has demonstrated that physical exercise also aids memory
Diet	What is good for the heart is good for the brain. Fruits, vegetables, food rich in omega-3 fatty acids (eg, fatty fish such as salmon), whole grains, spices (eg, turmeric), and small amounts of tree nuts (eg, walnuts) are recommended as part of a balanced diet

Source: References 1,29

causes of memory impairment (*Table 4, page 40*)^{28,29,31,32}—which could be treated—some will have MCI, and others may be in an early stage of dementia.

CASE CONTINUED

No measurable deficits

Ms. F's medical history is remarkable for coronary artery disease, hypothyroidism, hypertension, hyperlipidemia, cataracts, arthritis, back surgery (secondary to spondylosis), and foot surgery. Ms. F denies a history of alcohol or illicit substance abuse. She smoked tobacco for 30 years (2 packs per day), but quit 5 years ago after her heart attack. Physical exam is unremarkable except for mild obesity (body mass index = 31 kg/m²).

Ms. F's mental status exam reveals anxious mood and affect. Her recall is 2 out of 3 items. Her MMSE score is 29/30 (1 point lost on recall) and her Geriatric Depression Scale³³ score is 2/15, indicating minimal depressive symptoms. On neuropsychological testing, Ms. F demonstrates high average intellectual abilities; compared with others her age, she performs within expectations on all measures. That is, she performs within the above-average to low-average range on measures of attention, working memory, speed of processing, expressive language, learning, mem-

ory, visual spatial abilities, executive functioning, and knowledge of basic health and safety information.

Enhancing neuroplasticity

We recommend neuroplasticity-based interventions to treat SCI and promote healthy brain aging.^{20,29} For a checklist clinicians can use to promote healthy brain aging and thus improve patients' cognitive health see this article at CurrentPsychiatry.com. *Table 5*^{1,29} lists cognitive strategies to improve memory and maintain cognitive vitality.

Enhancing brain plasticity and neurogenesis requires engaging older adults in demanding sensory, cognitive, and motor activities on an intensive basis.³⁴ Therapeutic stimulation of neuroplasticity and neurogenesis might contribute to functional "repair" of the diseased adult brain before damage to whole neuronal networks has ensued.²⁹ An important treatment component is reassuring patients with SCI that they do not have AD or MCI. Treating comorbid anxiety and depression and reversible causes of cognitive complaints is key to successful outcomes.

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Visit this article at CurrentPsychiatry.com for a checklist to promote healthy brain aging

CASE CONTINUED

Reassurance and risk reduction

Ms. F's psychiatrist reassures her that she does not have AD. She receives genetic counseling and decides to forgo genetic testing. Her psychiatrist educates Ms. F about the risks of OTC supplements—especially increased risk of bleeding because she takes aspirin and clopidogrel—and lack of data supporting their use. Ms. F is counseled that a healthy lifestyle, including regular exercise, Mediterranean diet with increased intake of omega-3 fatty acids, learning new things, and being socially active, is the safest way to promote brain health. Over 3 months, Ms. F discontinues all supplements except the vitamins and omega-3, starts exercising, resumes piano lessons that she stopped 10 years ago, and becomes a vegetarian. She continues to have mild SCI but she says she is not bothered by it and feels satisfied that she is doing all she can to promote her brain health.

References

- 1. Small GW. What we need to know about age related memory loss. BMJ. 2002;324:1502-1505.
2. Hayes TL, Larimer N, Adami A, et al. Medication adherence in healthy elders. J Aging Health. 2009;21(4):567-580.

Related Resources

- Desai AK. Healthy brain aging: evidence based methods to preserve brain function and prevent dementia. Philadelphia, PA: W.B. Saunders; 2010.
Doidge N. The brain that changes itself. New York, NY: Penguin Books; 2007.
Vance DE, Roberson AJ, McGuinness TM, et al. How neuroplasticity and cognitive reserve protect cognitive functioning. J Psychosoc Nurs Ment Health Serv. 2010;48:1-8.

Brain Training Resources

- Weil A, Small G. The healthy brain kit. Boulder, CO: Sounds True, Inc.; 2007. Audio CDs, brain-training cards and workbooks.
Posit Science. www.positscience.com.
Sharp Brains. www.sharpbrains.com.

Drug Brand Names

Table with 2 columns: Drug Name, Brand Name. Includes Atenolol, Tenormin, Levothyroxine, Levoxyol, Atorvastatin, Lipitor, Synthroid, Clopidogrel, Plavix.

Disclosures

The authors report no financial relationship with any company whose products are mentioned in this article or with manufacturers of competing products.

- 3. Reisberg B, Shulman MB, Torossian C, et al. Outcome over seven years of healthy adults with and without subjective cognitive impairment. Alzheimers Dement. 2010;6(1):11-24.
4. Jessen F, Wiese B, Bachmann C, et al. Prediction of dementia by subjective memory impairments: effects of severity and temporal association with cognitive impairment. Arch Gen Psychiatry. 2010;67:414-422.

continued

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Therapeutic stimulation of neuroplasticity and neurogenesis might contribute to functional 'repair' of the diseased brain

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Reassure patients with SCI that they do not have AD or MCI and treat comorbid anxiety and depression and reversible causes of cognitive complaints

5. Waldorff FB, Siersma V, Waldemar G. Association between subjective memory complaints and nursing home placement: a four-year follow-up. *Int J Geriatr Psychiatry*. 2009;23(6):602-609.
6. Salthouse TA. Selective review of cognitive aging. *J Int Neuropsychol Soc*. 2010;16:754-760.
7. Anderton B. Ageing of the brain. *Mech Ageing Dev*. 2002;23:811-817.
8. Salthouse TA. Influence of age on practice effects in longitudinal neurocognitive change. *Neuropsychology*. 2010;24(5):563-572.
9. Park D, Schwarz N. *Cognitive aging: a primer*. Philadelphia, PA: Taylor and Francis Group; 2000.
10. Reisberg B, Shulman MB. Commentary on "a roadmap for the prevention of dementia II: Leon Thal Symposium 2008." Subjective cognitive impairment as an antecedent of Alzheimer's dementia: policy import. *Alzheimers Dement*. 2009;5:154-156.
11. Reisberg B, Gauthier S. Current evidence for subjective cognitive impairment (SCI) as the pre-mild cognitive impairment (MCI) stage of subsequently manifest Alzheimer's disease. *Int Psychogeriatr*. 2008;20(1):1-16.
12. Mosconi L, Pupi A, De Leon MJ. Brain glucose hypometabolism and oxidative stress in preclinical Alzheimer's disease. *Ann N Y Acad Sci*. 2008;1147:180-195.
13. Ramakers IH, Visser PJ, Aalten P, et al. Symptoms of preclinical dementia in general practice up to five years before dementia diagnosis. *Dement Geriatr Cogn Disord*. 2007;24(4):300-306.
14. Amieva H, Le Goff M, Millet X, et al. Prodromal Alzheimer's disease: successive emergence of the clinical symptoms. *Ann Neurol*. 2008;64(5):492-498.
15. Taragano FE, Allegri RF, Krupitzki H, et al. Mild behavioral impairment and risk of dementia: a prospective cohort study of 358 patients. *J Clin Psychiatry*. 2009;70(4):584-592.
16. Jayadev S, Steinbart EJ, Chi YY, et al. Conjugal Alzheimer disease: risk in children when both parents have Alzheimer disease. *Arch Neurol*. 2008;65(3):373-378.
17. Hajjar I, Yang F, Sorond F, et al. A novel aging phenotype of slow gait, impaired executive function, and depressive symptoms: relationship to blood pressure and other cardiovascular risks. *J Gerontol A Biol Sci Med Sci*. 2009;64(9):994-1001.
18. Yamamoto N, Yamanaka G, Ishikawa M, et al. Cardio-ankle vascular index as a predictor of cognitive impairment in community-dwelling elderly people: four-year follow-up. *Dement Geriatr Cogn Disord*. 2009;28(2):153-158.
19. Inzitari M, Pozzi C, Ferrucci L, et al. Subtle neurological abnormalities as risk factors for cognitive and functional decline, cerebrovascular events, and mortality in older community-dwelling adults. *Arch Intern Med*. 2008;168(12):1270-1276.
20. Shineman DW, Salthouse TA, Launer LJ, et al. Therapeutics of cognitive aging. *Ann N Y Acad Sci*. 2010;1191(suppl 1):E1-E10.
21. Dubois B, Feldman HH, Jacova C, et al. Revising the definition of Alzheimer's disease: a new lexicon. *Lancet Neurol*. 2010;9:1118-1127.
22. Chertkow H, Massoud F, Nasreddine Z, et al. Diagnosis and treatment of dementia: 3. Mild cognitive impairment and cognitive impairment without dementia. *CMAJ*. 2008;178(10):1273-1285.
23. Rosenberg PB, Lyketsos C. Mild cognitive impairment: searching for the prodrome of Alzheimer's disease. *World Psychiatry*. 2008;7(2):72-78.
24. Rosenberg PB, Johnston D, Lyketsos CG. A clinical approach to mild cognitive impairment. *Am J Psychiatry*. 2006;163(11):1884-1890.
25. Folstein MF, Folstein SE, McHugh PR. "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res*. 1975;12(3):189-198.
26. Petersen RC, Smith GE, Waring SC, et al. Mild cognitive impairment: clinical characterization and outcome. *Arch Neurol*. 1999;56(3):303-308.
27. *Diagnostic and statistical manual of mental disorders*. 4th ed, text rev. Washington, DC: American Psychiatric Association; 2000:135-180.
28. Malhotra R, Desai AK. Healthy brain aging: what has sleep got to do with it? *Clin Geriatr Med*. 2010;26:45-56.
29. Desai AK, Grossberg GT, Chibnall JT. Healthy brain aging: a road map. *Clin Geriatr Med*. 2010;26:1-16.
30. Weaver Cargin J, Collie A, Masters C, et al. The nature of cognitive complaints in healthy older adults with and without objective memory decline. *J Clin Exp Neuropsychol*. 2008;30:245-257.
31. Wilson RS, Arnold SE, Schneider JA, et al. Chronic distress, age-related neuropathology, and late-life dementia. *Psychosom Med*. 2007;69:47-53.
32. Deal JA, Carlson MC, Xue Q, et al. Anemia and 9-year domain-specific cognitive decline in community-dwelling older women: the Women's Health and Aging Study II. *J Am Geriatr Soc*. 2009;57(9):1604-1611.
33. Yesavage JA, Brink TL, Rose TL, et al. Development and validation of a geriatric depression scale: a preliminary report. *J Psychiatr Res*. 1983;17:37-49.
34. Mahncke HW, Bronstone A, Merzenich MM. Brain plasticity and functional losses in the aged: scientific bases for a novel intervention. *Prog Brain Res*. 2006;157:81-109.

Bottom Line

Patients with subjective cognitive impairment (SCI) often worry that their perceived cognitive decline could be an early sign of Alzheimer's disease. Such patients should be thoroughly evaluated to identify reversible causes of cognitive impairment and undergo neuropsychological testing to differentiate SCI from mild cognitive impairment and dementia.

Checklist to promote healthy brain aging: A clinicians' guide

1	Counseled regarding smoking cessation	<input type="checkbox"/>
	Comments:	
2	Advised to follow guidelines proposed jointly by the American Heart Association and the American College of Sports Medicine regarding daily physical activity	<input type="checkbox"/>
	Comments:	
3	Counseled regarding healthy nutrition (eg, Mediterranean diet, Dietary Approaches to Stop Hypertension diet)	<input type="checkbox"/>
	Comments:	
4	Counseled regarding the importance of intellectually challenging and creative leisure time activities	<input type="checkbox"/>
	Comments:	
5	Counseled regarding strategies to promote emotional resilience and reduce psychological distress and depression (eg, relaxation, mindfulness meditation practices)	<input type="checkbox"/>
	Comments:	
6	Advised to maintain an active, socially integrated lifestyle	<input type="checkbox"/>
	Comments:	
7	Discussed strategies to achieve and maintain optimal daily sleep	<input type="checkbox"/>
	Comments:	
8	Provided education about strategies to reduce risk of serious head injury (eg, wearing seat belts, wearing helmets while bicycling, etc.)	<input type="checkbox"/>
	Comments:	
9	Provided education about strategies to reduce exposure to hazardous substances (eg, wearing protective clothing when using pesticides, fumigants, fertilizers, and defoliants)	<input type="checkbox"/>
	Comments:	
10	Provided education and counseling provided regarding negative health effects of alcohol consumption more than recommended as safe by the National Institute of Alcohol Abuse and Alcoholism	<input type="checkbox"/>
	Comments:	
11	Provided education about importance of achieving and maintaining healthy weight to promote overall health	<input type="checkbox"/>
	Comments:	
12	Discussed and implemented strategies to achieve optimal blood pressure control	<input type="checkbox"/>
	Comments:	
13	Discussed and implemented strategies to achieve optimal control of dyslipidemia	<input type="checkbox"/>
	Comments:	
14	Discussed and implemented strategies to achieve optimal control of blood sugar and/or diabetes	<input type="checkbox"/>
	Comments:	
15	Discussed risks and benefits of medications, supplements, herbal remedies, and vitamins to promote brain health	<input type="checkbox"/>
	Comments:	
16	Discussed and implemented secondary prevention of stroke strategies (eg, daily aspirin)	<input type="checkbox"/>
	Comments:	