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# Practice Current: How do you manage mild cognitive impairment?

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**Abstract**

Mild cognitive impairment (MCI) is characterized by evidence of cognitive impairment with minimal disruption of instrumental activities of daily living and carries a substantial risk of progression of dementia. Whereas current guidelines support a relatively minimalistic workup to identify reversible or structural causes, the field has witnessed the rapid development of various sophisticated imaging, biomarker, and genetic investigations in the past few years. The role of these investigations in routine practice is uncertain. Similarly, while there are no approved treatments for MCI, neurologists may experience uncertainty about using cholinesterase inhibitors or other medications or supplements that have been studied in MCI with limited success, particularly when patients or families are keen to try pharmacological options. Given these uncertainties, and the paucity of high-quality data in the literature, we sought expert opinion from around the globe on how to investigate and treat patients with MCI. Similar questions were posed to the rest of our readership in an online survey, the preliminary results of which are also presented.

**Introduction**

Mild cognitive impairment (MCI) is characterized by evidence of cognitive impairment with minimal impairment of instrumental activities of daily living (IADLs).[1] In a recent systematic review, the estimated prevalence of MCI ranged from 6.7% for ages 60-64 to 25.2% for 80-84, with a cumulative dementia incidence of 14.9% in individuals with MCI older than age 65 years followed for two years.[2]

MCI can be the first clinical manifestation of Alzheimer disease (AD), or of other disease processes like vascular cognitive impairment, frontotemporal lobar degeneration, or Lewy Body disease. The workup for patients with MCI has conventionally included bloodwork and structural neuroimaging with CT/MRI to look for potentially reversible causes.[3] The 2018 practice guideline update for MCI from the American Academy of Neurology (AAN) recommend assessing for MCI with validated tools,[4] evaluating for modifiable risk factors and functional impairment, and monitoring cognitive status over time (Level B, i.e. clinicians "should assess" for these in appropriate scenarios). In recent years, there has been considerable research into more sophisticated investigations to better delineate the underlying disease process for patients with cognitive impairment, particularly to distinguish AD from other competing differential diagnoses.[5] This includes identifying underlying AD by detecting reduced levels of amyloid $A\beta_{1-42}$ and increases of total and phosphorylated tau in cerebrospinal fluid (CSF),[6] and detecting amyloid plaques with amyloid-specific tracers for positron-emission tomography (PET).[7] The pattern of hypometabolism on fluorodeoxyglucose (FDG)-PET or single-photon emission computed tomography (SPECT) scans can also help distinguish between different types of disease processes; for instance, fronto-temporal hypometabolism on FDG-PET in frontotemporal dementia compared to temporo-parietal hypometabolism in AD.[8] Our understanding of genetic risk factors has also evolved; for example, testing for apolipoprotein E (APOE) genotype (particularly the presence of ε4 alleles) helps with risk stratification of patient groups in cohort studies of MCI and AD,[9] and testing for dominant mutations like presenilin 1 and 2 identifies patients fated to develop early-onset AD.[10] That being said, the current role of these more sophisticated CSF, imaging, and genetic tests in the routine evaluation of patients with MCI remains uncertain, and the extent to which these they are available or accessible to practicing neurologists and their patients in different countries is unclear.[11]

The treatment of patients with MCI is also complicated. The 2018 AAN guidelines recommend assessing and treating behavioral and neuropsychiatric symptoms that may be associated with MCI, discontinuing cognitively impairing medications where possible, and encouraging regular exercise (Level B, i.e. clinicians "should recommend" these).[2] Whereas formal exercise training has been associated with improvement in cognitive measures, the uptake of such training in current practice is

uncertain.[12, 13] Cognitive training has been studied in some trials with promising results, but was deemed only level C in the AAN guideline (i.e. clinicians "may recommend" this).[14, 15] Although there is a general consensus that any potential therapies for causes of dementia like AD are likely to be most effective when applied early in the disease course, there is a frustrating lack of disease-modifying pharmacological options for patients with MCI.[16] In fact, the AAN guidelines support physicians *not* offering cholinesterase inhibitors (ChEIs, level B), and discussing the lack of evidence for treatments with their patients (level A).[2] Whereas there is moderate-quality evidence that ChEIs like donepezil, galantamine, and rivastigmine are effective in patients with dementia due to AD, LBD, and Parkinson's disease,[17-19] and memantine is effective in dementia due to AD,[20] these medications have not demonstrated a reduced progression to dementia in MCI. Several other medications including supplements like B vitamins,[21] ginkgo biloba,[22] and recently L-serine[23] have captured the attention of patients despite poor evidence. Stimulants like methylphenidate, dextroamphetamine, and modafinil have also generated interest in the treatment of MCI, with similarly limited evidence.[24, 25] It is unknown when or how neurologists would consider using these types of medications in patients with MCI in their practice. As awareness increases about dementia, neurologists will likely increasingly see patients at earlier stages with milder disease, and face uncertainty about their management, particularly when managing highly functioning patients keen to try any therapeutic options, but for whom there is little to offer in the way of evidence-based treatment.

Given these uncertainties about the investigation and treatment of patients with MCI in routine clinical practice, we sought expert opinion from around the globe on the question of how to best manage patients with MCI. In particular, we sought to better elucidate existing expert perspectives on this topic to identify areas of agreement and disagreement that could help clinicians critically examine and refine their own practice patterns.

**Expert Opinion**

Questions were posed to experts from three different continents, representing differing medical systems and patient populations. The following summary of their responses addresses their preferred investigation and treatment approaches for patients with MCI, and clinical characteristics that influence their management decisions. Similar questions were posed to the rest of our readership in an online survey using a representative case (see **Appendix e-1** for case and multiple-choice questions), the results of which are presented following the expert commentaries.

Expert commentaries in alphabetical order by last name.

**Masud Husain, DPhil, FRCP, FMedSci, FAAN, FEAN (United Kingdom)**

***Clinical Evaluation of MCI:*** History-taking guides everything in suspected MCI. If we detect cognitive impairment in a screening examination in a patient reported to have cognitive concerns, then the most important issue is to verify whether the patient is impaired in their instrumental activities of daily living, as this will determine whether the patient has MCI or dementia. The best way to do this is often to interview the patient's partner, family member, or other informant who knows the patient well, separately; spending this extra time on the history is crucial. The key question is the extent to which there has been change from their pre-existing level of functioning – how much they are relying on family, friends, or other community supports. For our initial cognitive screening, we use the Addenbrooke's Cognitive Examination, which overall assesses certain domains such as semantics, visuospatial and executive function in more detail than the Mini-mental State Examination (MMSE) or Montreal Cognitive Assessment (MoCA), and also has a wider dynamic range, being scored out of 100 rather than 30.[26]

***Investigation of MCI:*** As standard neuroimaging for such patients, we use MRI, which is better than CT for imaging small-vessel disease,[27] and include volumetric MRI, which allows us to assess regional atrophy more confidently. We will soon be routinely using nomograms from the UK Biobank data to compare a given patient's hippocampal volume to age-matched controls.[28] In addition, we find neuropsychological testing quite helpful as our screening cognitive tests are still rather blunt. We have a joint agreement with our neuropsychologists about the kind of tests that would be most valuable – we want a general assessment across all major domains in most cases, but in more language-related impairment, we might do a more language-focused assessment. I don't routinely obtain FDG-PET or CSF biomarkers as I do not find them too helpful in practice, unless the clinical diagnosis is still unclear even after neuropsychological testing and if the MRI seems to be within normal limits. We do not have routine access to amyloid or tau PET imaging in our practice.

We might consider a sleep study if the history is suggestive of sleep apnea or REM-sleep Behavior Disorder (RBD). We might consider dopamine transporter imaging if we suspect Lewy Body disease. Genetic testing would be considered only if there is a strong family history, especially of an early-onset dementia. Routine testing for ApoE genotype is not permitted in the National Health Service (NHS) in the United Kingdom; given that it is such a common genotype, it is easy to cause undue worry.

***Treatment of MCI:*** As for management, we focus on non-pharmacological strategies for MCI. This includes improving sleep, providing advice about regular aerobic exercise, and addressing mood and anxiety (over a third of our patients have psychiatric comorbidities). We educate the patient and family about the condition and about the risk of developing dementia (roughly 10% per year) – and that sometimes patients can improve from MCI – but we don't routinely provide written materials. We suggest audiology when it seems appropriate. We don't recommend cognitive training routinely. If we are convinced that the patient remains independent for their instrumental activities, we would not offer pharmacological intervention, and would see them in follow-up in a year's time. At that point, should they have a decline in their cognitive scores and/or be reported by an informant to be more dependent (usually both together), this suggests we are more likely to be dealing with AD or another neurodegenerative disorder. Such follow-up helps us be more confident about the diagnosis. If the patient was particularly high functioning pre-morbidly, then their assessment may not show much, and I have a much lower threshold for considering what constitutes impairment in instrumental activities, and for offering therapy.

Donepezil is my first choice of medication as the evidence base is longest and strongest for this drug.[18] Rivastigmine is probably next line, then galantamine, then memantine – this is driven by our relative familiarity and experience with the medications. If we think the patient has Lewy Body Disease, we favour rivastigmine.[29] In purely vascular dementia, I don't think the evidence base is particularly strong for offering ChEIs, but we would do so for patients with evidence of mixed disease (e.g. small vessel disease and hippocampal or parietal atrophy on MRI) who are more common anyway. However, the key point here is that these medications are not curative and there isn't good evidence that they modify the illness trajectory; they also come with costs and side effects. If the patient has vascular risk factors, or we think there is underlying vascular MCI, then vascular risk factor optimization is important. This includes aggressive blood pressure management (we would consider ambulatory BP monitoring), and considering an anti-platelet or statin. This is all based on shakier evidence. We don't use any vitamin or other supplements or stimulants routinely. We may use modafinil for patients with prominent sleep disturbance. There is emerging evidence for methylphenidate in the setting of apathy (for dementia, not MCI) from the Apathy in Dementia Methylphenidate Trial (ADMET); ADMET II is ongoing.[30]

Evaluation of benefit from these pharmacological therapies is very difficult because if your diagnosis is correct, the patient will inevitably worsen over time. Often the patient will say they have not noticed any change but the carer may have noticed a benefit, which is often clearer and more prominent in Dementia with Lewy Bodies than in AD. A more objective method is to

consider testing patients on and off the medication, a week apart; in some patients, we have observed with objective measures that there is a significant noticeable difference even with missing one tablet. If the patient is tolerating the medication, we generally continue it without a final endpoint. We may add memantine on top of a ChEI if a patient is declining rapidly. If we have started the patient on a medication, we will see them back within 4-months; if not, usually within 6-months and then on an annual basis.

**Kathleen L. Poston, MD, MS (United States of America)**

***Clinical Evaluation of MCI:*** In a patient with suspected MCI, assuming that the bloodwork and neuroimaging rule out reversible causes, I try to clinically differentiate between the two most common neurodegenerative causes – AD and LBD. This includes asking about fluctuations or behavioral symptoms and examining for gait imbalance or parkinsonism on exam, which would be more in keeping with LBD. It is important to ensure that the patient's cognitive decline is not substantially affecting the IADLs, to ensure that this is still MCI and not dementia. Interviewing the family is especially helpful for this, because family members have instinctively started doing or supervising one or more core activities for the person – such as bills or driving – because they felt the patient was not doing it correctly.

***Investigation of MCI:*** As for investigations, I find formal neuropsychological testing – including episodic memory, verbal memory, visual memory, executive tests, and visuospatial function (beyond just a bedside MoCA) – helpful in determining the pattern of the patient's cognitive impairment, which then has implications for practical recommendations to improve their functioning. For example, if the patient's problem is primarily executive dysfunction, then having the family give the patient only one activity at a time could help, whereas if the problem is primarily memory, then we can promote strategies like writing things down or keeping reminders. Regional atrophy patterns on CT/MRI are helpful; coronal slices help identify hippocampal atrophy in AD. MRI is preferred for identifying vascular pathology and other structural lesions. An additional investigation I would consider is lumbar puncture to measure amyloid and tau, as opposed to amyloid PET scans which only detect one of these proteins. The absence of amyloid on a scan may be helpful; however, many patients with LBD can also have amyloid deposition. That being said, we use amyloid PET or SPECT for research purposes. Similar, genetic testing is generally performed only in our research subjects, unless the patient is young and has a strong family history (accompanied by genetic counselling). I do not find ApoE testing helpful in practice. I consider a sleep study if there is a suspicion of sleep

apnea, or of LBD but without a convincing story of REM (rapid eye movement) sleep behavior disorder. If I get a reliable history from a bed partner describing no active movements in sleep and no snoring and the patient wakes up feeling refreshed, I wouldn't do a sleep study. A hearing assessment is essential if the patient has behavioral changes like seeming disengaged in conversation without a clear speech/language deficit. Similarly, if the patient is complaining of having visual changes, they may have a mild cataract; I have had a couple of patients with suspected LBD who had a dramatic improvement in hallucinations after cataract surgery.

***Treatment of MCI:*** Once we confirm this is MCI, my focus is on educating the patient and family about MCI and ideally about the suspected underlying disease process, and on identifying what we can do to improve their quality of life. In addition to aforementioned practical strategies, I counsel patients to do exercises that keep them physically and socially engaged, like tai chi, and if appropriate for their availability/capability, I refer them to an exercise or physical therapy program. I provide written material on exercise options including classes at Stanford for those less knowledgeable about exercise. I encourage them to stop smoking and to follow a healthy diet like a Californian diet and offer the option of referral to a dietician, especially if they have several cardiovascular risk factors. If their vascular risk factors appear to have been neglected, then I might refer them to colleagues in stroke prevention. I encourage patients to be cognitively active with enjoyable activities but am not convinced about the benefits of time-consuming and expensive cognitive training. Supporting caregivers through this difficult journey is essential; caregiver support groups can be very helpful in this regard.

As for medications, I would only consider ChEIs if patients have reached a stage of no longer being able to do one or more activities (i.e. dementia, not MCI). If patients have gastrointestinal side effects on once-daily donepezil, I usually switch to a daily rivastigmine patch (applied in the morning to avoid vivid dreams), though this can cause itching and stick to the skin. I would consider adding memantine if there was some initial improvement with ChEI but things worsened later. Importantly, I would reiterate that none of these medications reverse or stop disease progression. I don't recommend ChEIs in patients with suspected frontotemporal dementia or likely vascular dementia as there is no data for efficacy in the former and quite limited data for the latter.[31, 32] Although supplements are popular, we just don't have the data to support their use in MCI or dementia, so I do not recommend them to my patients. I would check for hypertension before using stimulants in patients with major attentional issues, but I can't think of any of my patients who are on modafinil.

To evaluate treatment response, I am guided by family, caregiver, and patient feedback. I ask them to think of a couple of things important to them, and ask them to compare the current state of those things to a few months ago. I repeat the MoCA once a year to get a sense of how things are changing over time, but it is primarily to help counsel the patient/family and not to guide treatment. I usually follow-up every 6-months unless I notice anything new or different. If I start a medication, I might see them in 3-months to see where things are and if no changes, then I follow-up every 6-months.

**Kirti Ranchod, MBBCh (South Africa)**

***Clinical Evaluation of MCI:*** MCI is a very interesting and challenging subject from diagnostic and management perspective. It is important to distinguish whether the cognitive impairment is normally expected for that age verses MCI or dementia. This can be accomplished by good history taking from patient and as well as collateral history from the family member and by performing the MMSE or MoCA in clinic followed by formal neuropsychological testing.

***Investigation of MCI:*** As a part of the initial work up, I use brain MRI/CT as the initial neuroimaging modality to exclude secondary or potentially reversible causes. FDG-PET imaging, SPECT imaging, and apolipoprotein E genotype are available, but these are rarely requested. I consider a sleep study and psychiatric evaluation in patients in whom the history is suggestive of any sleep disorder or mood disorder respectively. Amyloid or tau PET imaging or CSF biomarkers ($A\beta_{1-42}$, total tau, phosphorylated tau) are currently not available.

***Treatment of MCI:*** In terms of management, I consider non-pharmacological approaches for MCI which includes cognitive training, audiology for hearing loss, counselling about sleep hygiene, regular exercise and referral to a dietician. For MCI, I do not start any pharmacological therapy because there is no good evidence that these pharmacological therapies are beneficial, and in my opinion, the costs are not justifiable for people with limited financial resources. MCI patients are followed up to determine if there is any cognitive deterioration or any change in their clinical picture which requires further investigations or treatment. I consider pharmacological therapy if the patient's cognitive tests continue to show deterioration and the patient loses independence for one or more IADLs. If the patient meets these criteria for offering pharmacological treatment, I consider donepezil or rivastigmine or galantamine based on insurance coverage. I evaluate the benefit of these pharmacological therapies by interviewing the patient and family and by repeating cognitive testing on follow-up. If the patient is tolerating

the medication, I usually continue it and see them back in 3-months followed by 6-months and then on an annual basis if everything is stable.

***Lower-/middle-income country (LMIC) challenges:*** LMICs are of course quite varied in terms of challenges to access and affordability. In South Africa, I work in an urban environment where state and private healthcare options are available. Within this context, there is reasonable access to clinicians, investigations and treatment. Choices of investigations and treatment are determined by understanding patient preferences and resources available. These include, but are not limited to, the availability of investigations or treatment, costs of investigations or treatment and health insurance coverage of these. For example, for uninsured patients in whom I am considering a pharmacological agent, the cost of the medication drives the selection of pharmacological agent.

**Preliminary survey results**

We collected a total of 477 complete responses between November 22, 2019 and May 10, 2020. Respondents were primarily adult neurologists (n=443; 89%); 24% reported sub-specializing in cognitive neurology or dementia care. The majority of respondents had a primarily hospital-based practice (n=285, 58%) and reported having treated more than 10 patients with cognitive impairment or dementia in the 12 months preceding the survey (n=353, 65%; 39% reported seeing 25 or more such patients). 43% had been in practice for 10 or more years, 39% for less than 10 years, and 18% were physicians in training; 9% identified as physician assistants or nurse practitioners. Most respondents reported practicing outside the United States of America (n=366, 76%), with 75 other countries represented; Brazil (n=50), Spain (n=35), and India (n=34) had the highest representation.

For the presented case of MCI, in addition to standard bloodwork and neuroimaging (CT/MRI), most respondents (n=337, 60%) would refer the patient for further neuropsychological testing **(Figure 1)**. The next three most popular choices were lumbar puncture for amyloid beta (40/42), total tau, and/or phosphorylated tau (n=208, 37%) PET with an amyloid ligand (n=151, 27%), and FDG-PET (n=123, 22%). When asked about which investigations were actually available to them in their current practice, most respondents had access to neuropsychological testing (n=404, 72%) and sleep studies (n=319, 57%) and close to half had access to CSF testing of amyloid and tau species (n=270, 48%). FDG-PET was available to many (n=239, 43%) but less than a quarter had access to amyloid PET (n=126, 23%).

When asked about their preferred non-pharmacological options, the majority of respondents would refer such a patient for a cognitive training program (n=310, 56%), with the next most popular options being verbal advice on exercise and diet (n=265, 48%), providing written materials with such advice (n=215, 39%), and referral for a supervised exercise program (n=176, 32%). There was clear disagreement about whether pharmacological treatment should be offered to such a patient with MCI **(Figure 2)**; the most frequent answer was "No" (n=247, 45%), but many respondents said they would offer treatment regardless of ancillary investigations (n=147, 27%), or that they would do so if investigations demonstrated specific amyloid pathology or were consistent with a specific disease pattern (n=137, 25%). Among those who would offer treatment, the most popular choice by far was donepezil (n=226, 80%) followed by rivastigmine (33%), memantine (28%), and galantamine (25%); stimulants and supplements were chosen by a small minority of respondents (generally <10%).

When those who indicated that they would not offer treatment at this time were asked when they might consider doing so **(Figure 3)**, the most common responses were that they would do so if the patient's cognitive tests continued to show deterioration in follow-up, even if they remained independent for IADLs (n=111, 45%), or if the patient lost independence for one or more IADLs (i.e. developed early dementia, n=106, 43%). Only 6% (n=14) indicated that they simply would not consider treatment at all. When asked to select a treatment, assuming that the patient now met their criteria for offering treatment, the most popular choice among these respondents was again donepezil (n=189, 81%), followed by rivastigmine (n=112, 48%) and memantine tied with galantamine (n=62 or 27% each).

Among those respondents who selected a treatment either initially or based on additional developments in follow-up, most indicated that they would rely on caregiver/family reports about the patient's functioning in daily life (n=393, 77%) to evaluate whether the treatment was making a difference. The next most common choices were relying on the patient's report about their functioning (n=341, 67%) or using cognitive testing at follow-up (n=330, 64%). When asked how long they would continue the treatment, the most common choice was to do so only as long as benefit was evident to the patient/caregiver or on clinical measures (n=221, 43%); others indicated that they would continue as long as the patient was able to tolerate the medication, even if benefit was not evident (n=151, 30%) or that they would continue until the patient developed severe dementia, becoming dependent for all ADLs (n=102, 20%). Most respondents said they would follow patients with MCI every 6 months (n=310, 57%), the next most frequent choices being every 3 months (n=173, 32%) or annually (n=47, 9%).

These preliminary results indicate uncertainty in the neurology community about how to investigate patients with MCI, particularly when it comes to CSF testing or PET techniques. Although there seems to be consensus about the value of non-pharmacological strategies for managing MCI, the relative enthusiasm of our respondents for referral to cognitive training programs stood in contrast to the differing opinions of our experts on the value of such programs. These results also indicate considerable clinical equipoise about the use of medications, specifically ChEIs, in patients with MCI, particularly in the face of deteriorating cognitive tests or evidence of an underlying pathology. At odds with the experts, most respondents (52%) would consider offering treatment with ChEIs to patients with MCI either regardless of investigations or if investigations demonstrated specific amyloid pathology or other clear disease patterns, even if IADLs were still spared. We look forward to seeing the final results of this survey on a larger worldwide scale, and to exploring these results by differences in reported expertise, experience, and practice settings.

**Discussion**

Neurologists seeing patients with MCI can choose from several different options for investigations and management strategies with differing levels of evidence. All three interviewed experts agreed that a careful clinical evaluation of MCI, particularly history-taking involving family members or caregivers, is of paramount importance not only to gain potential insights about the underlying pathological process but also to distinguish MCI from early dementia. The experts also agreed that formal neuropsychological testing can have value to better characterize the pattern of cognitive impairment. There was general agreement that sophisticated FDG-PET, SPECT, or amyloid imaging remain unnecessary in typical cases, even though the experts were able to access some or all of these technologies at least in research settings. The experts differed in their consideration of CSF evaluation for amyloid and tau proteins in MCI. They favoured pursuing a sleep study on a case-by-case basis. As for the treatment of MCI, they would all encourage exercise and healthy diets but differed in their enthusiasm for formal exercise programs or referral to a dietician. They would encourage audiology evaluation if needed. One expert supported cognitive training while the others would not typically recommend it. Two experts cited the need to assess for vascular risk factors and whether they were being adequately treated. There was clear consensus around avoiding pharmacological treatments given poor evidence, with the experts only considering treatment with ChEIs if the patient shows impairment of IADLs consistent with early dementia rather than MCI. If starting a

medication, they would recommend seeing the patient again in 3-months but otherwise would follow the patient every 6-months or annually to evaluate progression and address other neuropsychiatric or behavioral symptoms.

These expert commentaries may serve to guide neurologists and other clinicians in their management of this frequently encountered and potentially challenging patient scenario. Our preliminary survey results suggest several uncertainties regarding this topic, especially regarding the use of additional sophisticated testing and pharmacological strategies. In this regard, it may be heartening to note that our experts emphasize the continued dominance of clinical evaluation of MCI over ancillary investigations, and the importance of non-pharmacological management strategies.

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**FIGURE LEGENDS**

**Figure 1.** Preferred investigations among respondents in a patient with mild cognitive impairment (MCI), aside from regular screening bloodwork and brain CT/MRI.

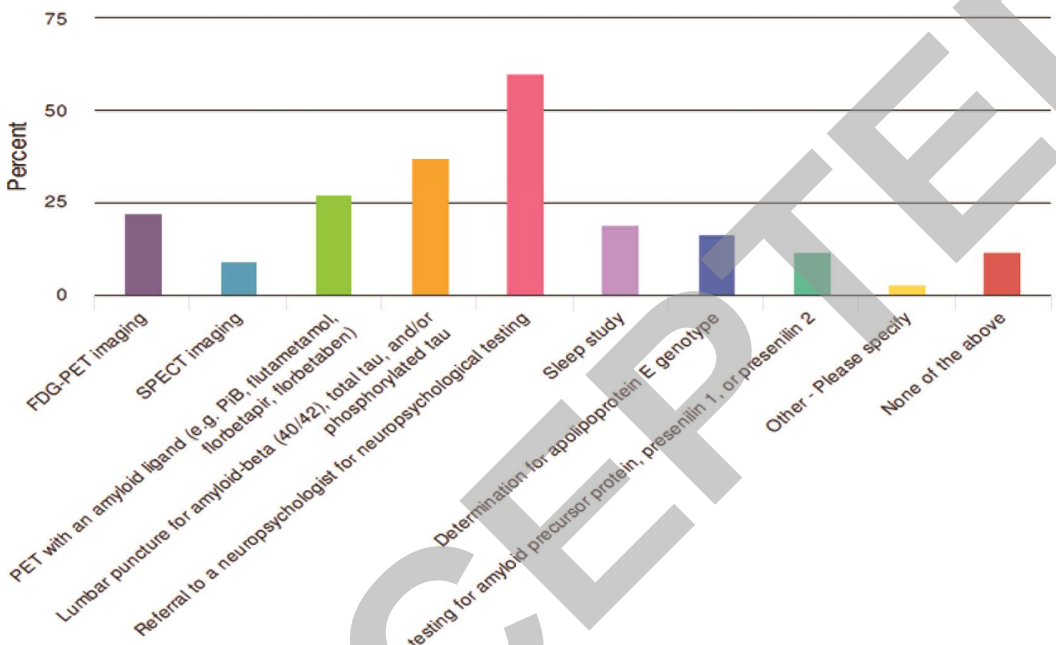


**Figure 2.** Preferences of respondents around the world regarding whether to offer a pharmacological treatment to a patient whom they have diagnosed with MCI.

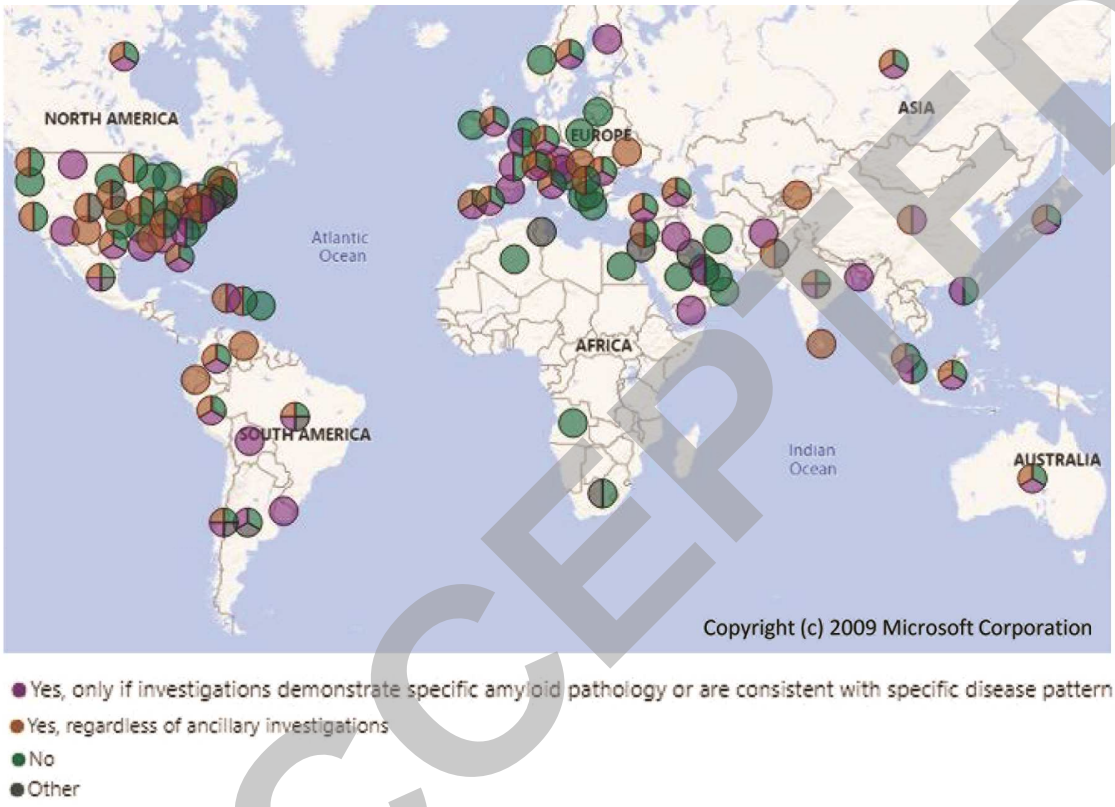
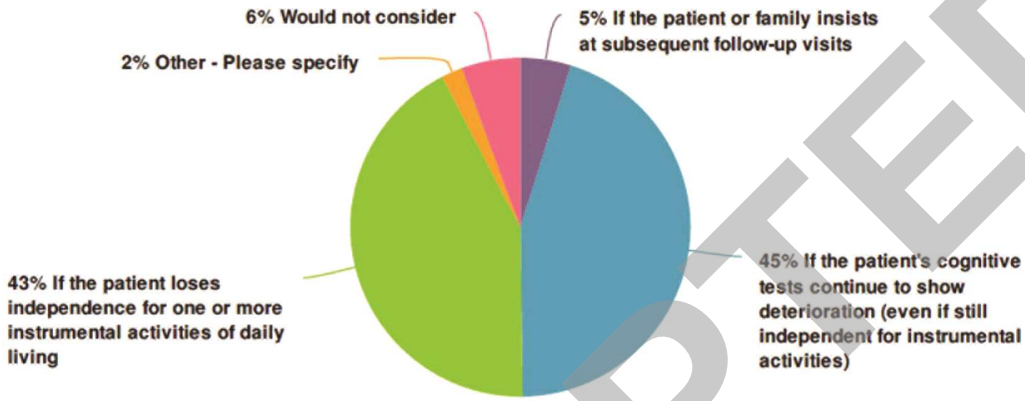


**Figure 3.** When respondents would consider offering a treatment to a patient with MCI over the course of their follow-up (if not offered initially).



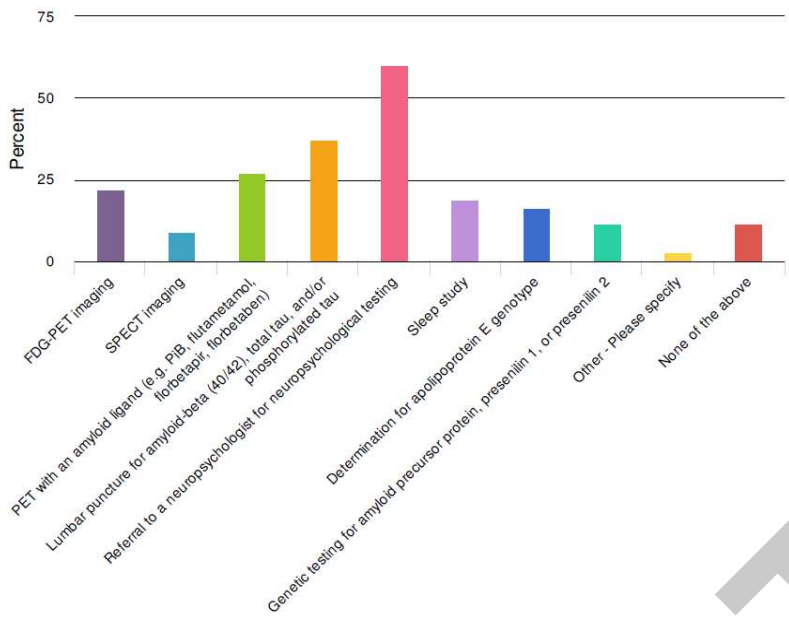



**Figure 1.** Preferred investigations among respondents in patients with mild cognitive impairment (MCI), aside from regular screening bloodwork and brain CT/MRI.


**Figure 2.** Preferences of respondents around the world regarding whether to offer a pharmacological treatment to a patient whom they have diagnosed with MCI. Copyright (c) 2009 Microsoft Corporation.

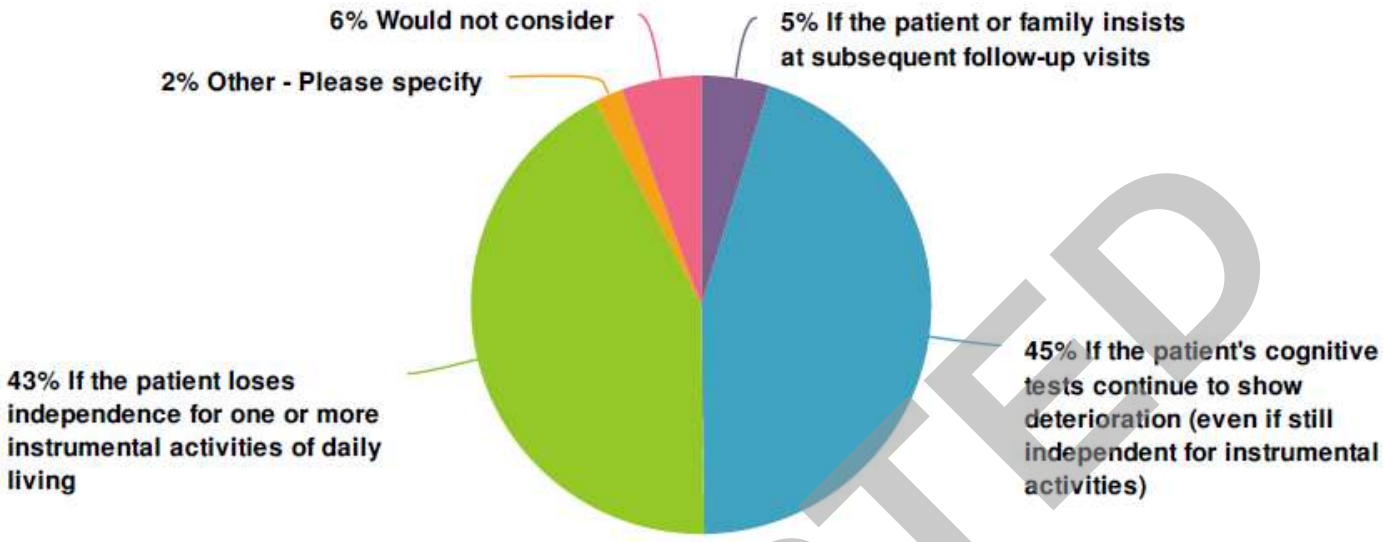



**Figure 3.** When respondents would consider offering a treatment to a patient with MCI over the course of their follow-up (if not offered initially).

**Expert bios:**

**Masud Husain, DPhil, FRCP, FMedSci, FAAN, FEAN** is Professor of Neurology & Cognitive Neuroscience at the University of Oxford and Wellcome Trust Principal Research Fellow. He leads the Neurological Conditions theme of the NIHR Oxford Biomedical Research Centre at Oxford and is Professorial Fellow at New College, Oxford. Masud is Co-Chair of the European Academy of Neurology Scientific Panel on Higher Cortical Functions and is Editor elect of *Brain.* His research focuses on mechanisms underlying motivation and memory in patients with neurodegenerative disorders, including Parkinson's disease, Alzheimer's disease, and cerebrovascular small vessel disease.

**Kathleen L. Poston, MD, MS (United States of America)** is Associate Professor of Neurology & Neurological Sciences and (by courtesy) Neurosurgery at Stanford University. She received her Bachelor of Science in Bioengineering at the University of Pennsylvania, her Master's Degree in Biomedical Engineering and her MD at Vanderbilt University. She completed her

Neurology residency training at UCSF, completed a fellowship in clinical Movement Disorders at Columbia University and post-doctoral research training in Functional Neuroimaging at the Feinstein Institute. Dr. Poston's research and clinical emphasis is to understand the motor and non-motor impairments, such as dementia, that develop in patients with alpha-synuclein pathology, such as Parkinson's disease, Lewy body dementia, and Multiple System Atrophy. Her lab uses functional and structural imaging biomarkers, along with biological biomarkers, to understand the underlying pathophysiology associated these symptoms. She holds joint appointments in Movement Disorders and Memory Disorders and is a founding member of the Stanford Alzheimer's Disease Research Center, she leads the Clinical Core for the Pacific Udall Center, which is an NIH-funded Parkinson's disease Research Center of Excellence.

**Kirti Ranchod, MBBCh (South Africa)** is a neurologist, based in Johannesburg, South Africa. She graduated with a degree in medicine from the University of Witwatersrand in 1998 (MBBCh) and obtained her specialist neurology qualification from the Colleges Of Medicine SA in 2007. She completed the Atlantic Fellowship for Equity in Brain Health in 2019. Her professional focus is on memory and on brain health. Her interest in brain health emerged while exploring ways to prevent dementia and protect memory. In doing this, she began to appreciate that a narrow focus on memory was not sufficient to protect memory, as healthy memory requires a healthy brain. Interests include the role of traditional practices in promoting health, neuroaesthetics, and understanding the different perceptions of memory.

# Practice Current: How do you manage mild cognitive impairment?

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