



# Memory difficulties are not always a sign of incipient dementia: a review of the possible causes of loss of memory efficiency

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## Abstract

**Introduction or background:** Memory problems are a very common reason for presenting to primary care. There is a need for better treatments for dementia. Increased government and media interest may result in greater number seeking help for memory problems, which may not reduce the dementia gap but rather increase numbers seen who do not have dementia. This review highlights the issues around the diagnostic criteria and terminology used for people with memory complaints.

**Sources of data:** A comprehensive literature search using PubMed using keywords for articles on subjective memory decline (SMD)/impairment/complaints, subjective cognitive decline (SCD), mild cognitive impairment (MCI) and functional memory disorder (FMD).

**Areas of agreement:** There is a need for early accurate detection of dementia syndromes so that trials of new treatments can begin earlier on the disease process.

**Areas of controversy:** Diagnostic criteria and terminology used for disorders of memory including SCD, MCI and FMD.

**Growing points:** This article reviews SCD and whether this can be used to predict Alzheimer's disease. The review also discusses the terminology used for non-progressive memory problems and the long-term outcomes for this patient group.

**Areas timely for developing research:** The accurate distinction of premorbid dementia syndromes from benign non-progressive memory problems.

Studies of treatment options for people with benign non-progressive memory problems and longer-term follow-up to determine which patients develop chronic problems.

**Key words:** dementia, functional memory disorder, mild cognitive impairment, subjective cognitive decline

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## Introduction

Subjective memory symptoms are frequent complaints in general practice.<sup>1-3</sup> More than half of an elderly population who complained of memory difficulties were worried about incipient dementia.<sup>3,4</sup> Metamemory, that is varieties of reflective insight into, or awareness of, the functioning of one's own memory differs between different types of dementia and also between healthy individuals. Although the recognition of one's own memory problems may be an early manifestation of a dementing illness, the self-perception of memory ability often fails to align with objective memory problems and may give rise to inaccurate beliefs about memory and ageing.<sup>5,6</sup> Factors that influence a misperception that one's memory is failing include low mood and impairments in activities of daily living.<sup>6</sup> Self-perceived memory difficulties, therefore, frequently do not indicate an incipient dementia. A variety of other factors can influence subjective memory efficiency, including psychological, environmental and pathological features.

This review examines the most frequent causes of memory dysfunction and the clinical advantages and disadvantages of the current diagnostic criteria and terminology used for people with cognitive complaints who are not demented. The classification and terminology of people with memory complaints, but who do not fulfill clinical diagnostic criteria and are not diagnosable with dementia, has a long history and includes age-associated memory impairment, age-associated cognitive decline, questionable dementia, cognitive impairment not dementia and benign senescent forgetfulness. Some of these terms are still used, but this review will mainly discuss focus on the concepts of mild cognitive impairment (MCI), subjective cognitive decline (SCD) and functional memory disorder (FMD). A review of the usefulness

of MCI as a diagnostic category has been published elsewhere.<sup>7-9</sup> As such, we have focused especially on SCD and FMD. We have chosen to use SCD [previously called subjective memory decline (SMD) and subjective memory impairment (SMI)] because that term is used in the recent framework paper published this year<sup>10</sup> but when referencing specific articles use the terminology from that article. This nosological category intends to capture patients with the earliest manifestations of Alzheimer's disease (AD). This is in contrast to FMD, a term used to describe memory complaints thought to be caused by psychosocial factors. It is difficult to compare these terms, as much of the research on subjective memory decline (SMD) has been on people aged over 70, while that on FMD has involved younger populations. However, age cannot be used as the only diagnostic indicator to distinguish between these two fundamentally different symptom presentations.

Dementia is broadly defined as a decline in cognitive abilities from a previous higher level, sufficient to impair normal function. Dementia is a syndrome with several possible underlying causes and does not indicate a specific disease. The condition has recently been identified as a UK national priority in the recent 'Prime Minister's challenge on dementia' (All Parliamentary Group Report, 2012). National guidelines promote an early diagnosis of dementia. In 2009, the UK government decided on the strategy of a 'memory clinic in every town' to facilitate early diagnosis, increase rates of patients formally diagnosed and improve access to effective treatments. The Commissioning for Quality and Innovation (CQUIN) guidelines require all acute hospitals in the UK to screen for dementia every person aged over 75 admitted to hospital. There is debate and controversy about potential harm from screening for memory disorders.<sup>11</sup>

The early diagnosis of dementia continues to represent a significant medical challenge and it has been argued that the diagnostic accuracy of current screening tools, neuropsychological profiles and biomarkers is still too low to justify large-scale screening. There is considerable uncertainty about the terms, which healthcare professionals should use to describe or diagnose syndromes characterized by memory problem complaints, but short of meeting criteria for dementia. Even the use of the term ‘dementia’ has been disputed. This term has been dropped in the new Diagnostic and Statistical Manual of Mental Disorders (DSM-5) and supplanted by the terms major and minor neurocognitive disorder.<sup>12</sup> Nosological debate is even greater when attempts are made to delineate very early stages of progressive memory disorders.

## MCI

The identification of individuals at a prodromal or preclinical stage of disease (most commonly AD) is of great importance if we want to test the potential of current and future treatments for disease modification and to reassure people with non-progressive memory problems. The concept of MCI (MCI-defined in [Box 1](#)) was based on earlier work and has supplanted replaced older terms such as age-associated memory decline in everyday clinical practice. MCI has been subdivided into amnesic and non-amnesic, single or multiple domains and according to suspected etiology (AD, vascular, psychiatric or secondary to other medical conditions).<sup>7,14</sup> The suspected etiological categories other than presumed AD and vascular are rarely used because the other subtypes are associated with a much lower risk of progression to AD. The label MCI, however, in current practice is still used to refer to a broad category or a syndrome, which includes not only patients with neurodegenerative pathology but also depression-related cognitive impairment, cognitive impairment due to alcohol and other medical co-morbidities. This heterogeneity means that the term MCI is used to describe clinical scenarios with considerable prognostic variability. The original purpose to identify individuals at high risk of AD or other neurodegenerative conditions leading to dementia is, therefore, not achieved. The

### Box 1. Definition of MCI

#### MCI<sup>13</sup>

1. The individual is neither normal nor demented.
2. There is evidence of cognitive deterioration, shown by either objectively measured decline over time or subjective report of decline by self or informant in conjunction with objective cognitive deficits.
3. Activities of daily life are preserved and complex instrumental functions are either intact or minimally impaired.

#### Summary of clinical and cognitive evaluation for MCI due to AD

1. Cognitive concern reflecting a change in cognition reported by patient or informant or clinician (i.e. historical or observed evidence of decline over time).
2. Objective evidence of impairment in one or more cognitive domains, typically including memory (i.e. formal or bedside testing to establish level of cognitive function in multiple domains).
3. Preservation of independence in functional abilities.
4. Not demented.
5. Examine etiology of MCI consistent with AD pathophysiological process.
6. Rule out vascular, traumatic, and medical causes of cognitive decline, where possible.
7. Provide evidence of longitudinal decline in cognition, when feasible.
8. Report history consistent with AD genetic factors, where relevant.

current practice of arbitrarily labeling individuals with memory complaints due to potentially reversible causes (such as depression and drug-related) as MCI (accounting for 10–30% of all cases to whom this label is applied<sup>14</sup>) can be misleading. Non-specialists and the general public may believe that any degree of ‘MCI’ indicates that these patients will inexorably progress to a form of clinical dementia. Even a survey of American neurologists with an interest in cognitive disorders reported that 20% of them either agreed or

strongly agreed that, 'MCI is usually better described as early AD'.<sup>15</sup> This suggests a subgroup of specialists considers that 'MCI' refers to the specific subtype of amnesic MCI, or else to prodromal AD. In other words, contrary to the original MCI concept, MCI is seen as a diagnostic category identical to early AD, rather than a clinical syndrome of cognitive impairment with a number of possible causes, which may be stable or even improve.

Jack *et al.* (2013)<sup>16</sup> proposed a theoretical model of premorbid to clinical AD, beginning with the deposition of amyloid, followed by neuronal injury, tau phosphorylation and structural change. This model suggests that MCI is an intermediate stage on progression to AD. In order to improve the utility of MCI and create homogenous patient groups, investigators have researched biomarkers to try to predict differentiate between those cases of MCI that who converts to dementia (MCI-c) versus and those that who do not (MCI-nc). Pathological levels of cerebral spinal fluid (CSF) Abeta 42 and tau have shown to be associated with an increased risk of converting to AD.<sup>17,18</sup> Imaging algorithm to detect AD-like atrophy (SPARE-AD) predicts conversion from MCI-c to AD,<sup>19</sup> and normal healthy individuals who progress to MCI.<sup>20</sup> The imaging algorithm correlated more closely with cognitive performance than CSF biomarkers, such as amyloid and tau.<sup>21</sup> This is plausible because atrophy is more closely related to neuronal and synaptic healthy than the presence of markers of neuropathological damage. Having said that, atrophy on a brain scan is increasingly common with age and is not a specific or sufficiently sensitive diagnostic indicator of dementia. Levels of asymptomatic atrophy in aged populations are high; in a study of over 700 community dwelling older adults (aged over 73 years); 25% had atrophy excessive for their age and 20% had infarcts or hemorrhages.<sup>22</sup>

The use of positron emission tomography (PET) or CSF biomarkers of amyloid deposition show increased risk of progression to AD in aggregate group data.<sup>23,24</sup> However, the use of PET and/or CSF biomarkers is not suitable for large-scale screening. A good biomarker should correlate with disease severity but the presence of amyloid depositions does not correlate well with the presence of clinical dementia as amyloid deposition is

also seen in healthy elderly. Notably, large-scale unbiased pathological series of elderly brains has shown that, although the presence of typical AD pathology increased the risk of manifesting dementia, there were people with pathological features of AD (Braak Stages 5 and 6 corresponding to high levels of classical Alzheimer pathology<sup>25</sup>) who were not demented in life.<sup>26</sup> In fact, the correlation between amyloid pathology and manifest dementia becomes less strong with age and other factors, such as synaptic loss, and related cortical thinning reducing cerebral reserve might lower the dementia threshold.<sup>26</sup> A prospective observational cohort studies study observing over 600 Roman Catholic sisters of the School Sisters of Notre Dame (often referred to as the Nun Study) showed that the presence of a second pathology (vascular or other neurodegenerative protein signal such as TDP43) was necessary before brain changes result in clinical dementia.<sup>27</sup> Finally, two Phase 3 studies of amyloid immunization of over 1000 patients found no difference in cognitive or functional scores,<sup>28,29</sup> suggesting the presence of amyloid was not directly responsible for cognitive impairment. This invites caution when assessing the diagnostic significance of biomarkers, which currently lack sufficient sensitivity on an individual patient basis. A meta-analysis of potential biomarkers in AD concluded that there needs to be a standardized methodology and reporting of future studies.<sup>30</sup> Currently, a combination of markers incorporating imaging and detailed neuropsychological profiles (including semantic tasks) offers the best approach for an early diagnosis of AD.<sup>31</sup>

## SCD

As stated earlier, subjective memory complaints are a common reason for requesting a consultation with a primary care physician.<sup>1-3</sup> There is evidence that AD can start many years before if it is clinically apparent.<sup>16</sup> To better characterize these very early stages of AD researchers have begun to study SMD. SCD is considered an earlier clinical manifestation of AD than MCI. A working group is currently forming a consensus definition of SCD.<sup>10</sup> Studies of patients with SCD in populations aged over 75 have described neuroradiological features similar to those seen in

AD patients such as volume loss in hippocampal/parahippocampal areas<sup>32,33</sup> and evidence of amyloid deposition using PET-imaging.<sup>34</sup> Taken together, these studies suggest that the mediotemporal regions in individuals with subjective memory impairment (SMI) aged over 75 show some indication of a degenerative process that mimics that of AD and, therefore, these patients represent a population with very early neurodegenerative pathology who, although experiencing subjective decline, would appear cognitively unimpaired when formally assessed. Along these lines, some authors have suggested that SCD may indicate subjective awareness of a degenerative process that can still be functionally compensated.<sup>33</sup>

The prognostic significance of SMI has been investigated. Studies have, in particular, focused on the question whether the amount of worry or concern about memory complaints can predict conversion to dementia.<sup>35</sup> Jessen *et al.*<sup>36</sup> investigated the risk of conversion to clinical AD in early and late MCI subcategories and in controls. The study group was a community-living population aged over 75 having attended their GP's practice at least once in the last 12 months. The rates of conversion to dementia (per year) were 3.7% in controls, 10.8% in early mild cognitive impairment (EMCI) and 24.9% in late mild cognitive impairment (LMCI). EMCI with no SMD had lower conversion rates of 2.5% (versus 10.8% in all EMCI) and LMCI with no SMD 12.1% (versus 24.9% in all LMCI). People with SMD alone and thus not fulfilling criteria for EMCI (i.e. within 1 SD of normative scores on objective memory tests) had a 6.2% conversion rate to AD, which is similar to EMCI patients. But if SMD was associated with 'no concern' then the risk of developing AD was the same as in controls. The presence of depressive symptoms was higher in 'SMD plus concern' than in 'SMD without concern' but this did not affect the risk of developing AD. This observation suggests that people's perception of their own memory and the registration of whether this perception is associated with concern could be useful adjuncts to neuropsychological testing and can increase the accuracy of predicting the risk for the development of AD in the over 75 age group. SMD without

subjective concerns had a similar risk of conversion to AD as controls in this study. Therefore, 'SMD becomes predictive only if the self-evaluation of the experienced impairment causes concern'.<sup>36</sup>

However, subjective memory complaints and risk of developing AD are clearly age-dependent. A large study of a population aged 45–64 found 12% had SMD.<sup>37</sup> Vascular risk factors such as smoking (OR: 1.18; 95% CI: 1.03–1.35) were not independently associated with complaints of SMD in a multivariate logistic regression, whereas psychological distress was (OR: 7.00; 95% CI: 5.41–9.07). Thus, in a younger population affective disorders are much more likely to be relevant to SMD than vascular risk factors, and the research findings in populations aged over 75 years cannot simply be extrapolated to younger age groups. This is important because subjective memory complaints are common throughout life. Ponds *et al.*<sup>38</sup> investigated SMD in a population study aged 24–86 and found that even in younger participants, 29% reported memory complaints (compared with 52% in the older age group).

Researchers investigating the implication of awareness of memory performance or metamemory in normal ageing, MCI and AD have reported variable results. The variance is in part due to age, cognitive scores and levels of anxiety.<sup>39,40</sup> A biopsychosocial model best explains this variance and how it affects the new MCI criteria,<sup>41</sup> which include patients' awareness of memory deficits as a diagnostic feature. In addition, the type of memory complaint requires further study; Amariglio *et al.*<sup>42</sup> found that 50% of the sample reported a 'change in memory' as the most common symptom. A rarer complaint—getting lost in familiar surroundings (reported by only 1.6%)—correlated with impairment on tasks also impaired in AD, i.e. delayed recall, semantic fluency and confrontation naming. The complaint most associated with normal ageing, i.e. forgetting something 1 s to the next, showed no relationship with scores on neuropsychological testing.

## FMD

Memory complaints are recognized as a possible functional symptom (i.e. a distressing somatic

symptom associated with abnormal thoughts, feelings and behaviors). Other functional symptoms include non-epileptic attacks (non-epileptic attack disorder; NEAD—also known as ‘psychogenic non-epileptic seizures’ or ‘dissociative seizures’), functional tremor and non-neurological conditions such as irritable bowel syndrome or non-cardiac chest pain.<sup>43,44</sup> There is no universally accepted term for ‘functional dementia’. The term ‘FMD’ (definition criteria in **Box 2**) is used to label people who have (potentially) reversible memory complaints, which are secondary to emotional or psychological factors<sup>45,46</sup> The term ‘functional’ in this label resonates with the use of this word in the new DSM-5 category ‘functional neurological symptoms disorder’, which has replaced the DSM-IV entity of ‘conversion disorder’.

The term FMD could be used as a more meaningful and precise diagnostic label than SMD for persistent, subjective deficits of memory and attention

because, particularly in young patients, the risk of progression to a dementia syndrome is very low. Patients with FMD tend to present with fairly stereotyped complaints about their memory. These include daily memory failures, forgetting chores while walking somewhere to start or complete them, prospective memory complaints (forgetting appointment and anniversaries), encoding deficits (conversations) and ‘memory blocks’ (people’s names, PIN numbers).<sup>47</sup> These types of memory failure are very similar to those reported in surveys by members of the general population and are not a cause of distress to many who experience them. However, in most cases of FMD the frequency and emotional/psychological consequences of these memory failures and an increasing attentional focus on the symptoms result in distress and the concern that the symptoms must be explained by a neurological disease. The awareness of memory failure can produce heightened anxiety and vigilance for other memory failures, as well as embarrassment, social withdrawal and isolation.<sup>46</sup> Thus, it can be argued that it is not the symptoms themselves, which are abnormal in FMD but rather the patients’ perceptions and behaviors in relation to the subjective memory/cognitive failures.

#### **Box 2. Definition of FMD**

##### **Diagnostic criteria:**

##### **FMD proposed by Schmidtke<sup>45</sup>**

1. Complaint of acquired (6 months) dysfunction of memory that, as perceived by patients, significantly affects their level of functioning in professional and/or private life.
2. Presence of external and/or subjective factors, addressed as psychosocial burden factors that cause significant psychological stress.
3. Verbal memory and attentional capacity >1.5SD (age-corrected), as assessed by standardized tests.
4. Absence of a recognizable organic cause of cognitive impairment. A physical examination, not including imaging, was routinely performed.
5. Absence of major psychiatric disease, e.g. psychosis, major depression, dissociative disorder, obsessive–compulsive disease, etc. (previous or present). Patients with dysthymia or adjustment disorder with depressed mood were included if the Beck Depression Inventory score was 15.

##### **Exclusion criteria for FMD**

- Age >70.

#### **Etiology of FMD**

There are many factors that might determine the onset of FMD. FMD is more common in people with above-average educational, professional and socio-economic attainment.<sup>48,49</sup> People with a perfectionist attitude toward memory may be at particular risk of developing a maladaptive response to lapses or failures of memory, to mood-, stress- or age-related changes in memory. Some people may be more sensitive to ‘normal’ age-related decline in learning and memory. Schmidtke *et al.*<sup>45</sup> suggests that factors such as interpersonal conflict, overwork, a distressing life changing event provoking psychological distress and handicap could trigger increased attention to cognitive processes, launching a vicious cycle of symptoms and worry that precipitate FMD. Put simply, a mismatch between cognitive demands (for instance related to professional or social multitasking) and capacity, or between emotional processing



needs and resources may result in cognitive symptoms. Further work is required to try and define or describe predisposing, precipitating and perpetuating symptoms of the disorder using a biopsychosocial framework (which has been used to 'explain' a more episodic functional neurological condition, NEAD).<sup>50</sup> In addition pre-existing personality traits, preferred coping strategies or features which increase resilience to life stresses, such as subjective psychological and physical well-being and good social integration, may influence the ability to compensate for change and on-going stress.<sup>38,51,52</sup> Many of the potential etiological factors listed above are non-specific, and to date it remains uncertain, why some patients develop functional memory problems and others, for example, functional weakness or (non-epileptic) seizures. There is evidence, however, that being in a state of continuous distress alters a person's ability to sustain attention and focus, resulting in poor encoding and retrieval of memories, which may result in temporary blocking of well-established contents of memory such as numbers, names or routine activities.<sup>53</sup> The resulting deficits and lapses may trigger rumination, self-accusation and fear of organic disease. Personal acquaintance with an elderly relative or friend with dementia or the increased awareness of dementia via media and government strategies may reinforce this fear.

### **Depression, FMD and memory complaints caused by neurodegenerative disorders**

People with FMD (in common with those with other functional symptoms) score more highly on self-perceived stress, physical and depression scales.<sup>46</sup> Severe depression and psychosis are exclusion criteria for FMD. While symptoms of anxiety and depression are not uncommon in FMD (and although the condition is associated with an increased risk of dysthymia), FMD is not simply caused by depression.<sup>45</sup> A distinction may be made between FMD and primary major depression with secondary memory impairment. The differentiation between FMD, depression with memory complaints (sometimes labeled 'depressive pseudo dementia') and memory impairment caused by

preclinical AD (often co-morbid with depression) is not straightforward, and further work is required to improve the accurate early separation of these diagnostic entities.

### **Prognosis and treatment strategies for FMD**

A longitudinal study by Schmidtke *et al.* (2008) explored the prognosis of FMD.<sup>45</sup> A total of 132 consecutive patients attending a memory clinic who did not have dementia were screened for this study. Of these, 59 patients were excluded, as they did not meet their criteria for FMD (48 with depression, 1 with bipolar disorder, 3 adult ADD, 1 personality disorder, 1 with significant organic brain disease and 5 scored >1.5 SD below the expected mean on neuropsychological cognitive testing). The remaining 73 people meeting all of their research criteria of FMD (age range 34–78; mean 55.2) were assessed. The following problems and co-morbid diagnoses were identified: 'adjustment disorder' (8/45), 'overwork in job or family' (12/56), 'interpersonal conflict in job or family' (6/60), 'somatic illness' (5/58), 'dysthymia' (8/58) and 'Alzheimer phobia' (2/59). On the same day as assessment took place, all fulfilling FMD criteria, and received individual counselling; reassured that their test scores were normal and the model of stress-induced cognitive impairment along with psychosocial burden was discussed. Twelve patients received formal psychiatric or psychotherapeutic help. Forty-six of the 73 participants were followed up for a mean time of 20.1 ± 6.3 months. One person was diagnosed with early dementia at follow-up and not included in further analyses. In six patients, symptoms had resolved at follow-up, but symptoms persisted in 39, although symptom severity reduced somewhat on an FMD inventory, FMD severity was significantly correlated with vegetative complaints at baseline but not with neuropsychological test scores. Self-reported effectiveness of the counseling was mixed; 21 found it helpful and 18 said it was not. This study suggests that FMD is a chronic but stable rather than a progressive neurodegenerative disorder. In keeping with this, we have seen several patients who have been discharged from

our memory clinic with reassurance that they did not have a dementia only for them to be re-referred by their primary care physician 1–2 years later with very similar complaints and stable scores on cognitive testing. Thus reassurance, even when it has been provided by an ‘expert in a secondary care memory clinic’ or one-time counseling about the likely non-progressive nature of the memory complaints, is not likely to be sufficient to resolve symptoms in most cases of FMD.

A group-based therapy for FMD incorporating psychoeducation, cognitive restructuring, stress management, relaxation and mindfulness techniques lasting 3 months showed improvement on measures of metamemory (the Metamemory in Adulthood Questionnaire) at 6 months but no change on measures of general distress and somatization.<sup>54</sup> Brain training programs have not been extensively tested in people with FMD; however, in one study a brain training program did not improve self-reported memory function.<sup>55</sup> A brochure designed to increase knowledge of ageing and memory did improve knowledge of memory function but did not affect levels of anxiety.<sup>1,56</sup>

## Conclusion

Subjective memory complaints are a common and frequent cause of attendances to GPs and of referrals to memory clinics. An integrated approach, beginning with clear diagnostic criteria for patients with relatively minor cognitive complaints, is important for future studies focusing on prognosis and treatment. At present, there is considerable confusion about the most appropriate diagnostic labels. Even when labels have been defined (such as the label MCI), they are widely misunderstood and even healthcare professionals who regularly see patients with MCI do not realize that some patients with MCI remain stable and some may even improve.<sup>15</sup> This is particularly important in younger people with memory complaints. A large resource/skill mix is required in memory clinics to help manage the variety of patients who present with memory complaints.

The early recognition of FMD is, like the early recognition of AD, necessary in order to offer

patients prompt appropriate treatment and to protect patients from iatrogenic aggravation of their symptoms. This requires updated diagnostic criteria and acceptance of an appropriate label. The definition of FMD proposed by Schmidtke *et al.*<sup>45</sup> for a research study (Box 2) is a useful start, although it requires some amendments if it is to be used in routine clinical practice. More specifically, a diagnosis of FMD may be appropriate in patients with scores  $>1.5SD$  below those from normative controls if the patient has neurological comorbidity or if there is evidence of poor or variable effort. We also believe that there will be people over the age of 70 with FMD, although careful evaluation is required to ensure that patients with early symptoms of dementia are not misdiagnosed as having FMD.

More work is required to help clinicians differentiate between benign non-progressive subjective memory dysfunction and the early stages of progressive memory disorders such as AD. The accurate early detection of AD and other dementing disorders is a health care priority so that treatment can be given and tested at an earlier stage. Biomarkers may be useful in detecting people at higher and lower risk of developing dementia but do not yet have sufficient diagnostic accuracy on an individual basis. It is important not to extrapolate results of studies on subjective memory decline in the over 65 year olds to younger patient groups. In addition to neuropsychological and neuroimaging results, a close examination of how patients present their memory complaints may be helpful. Research findings from seizure clinics are encouraging in this respect: a series of studies has demonstrated that patients with epilepsy and patients with functional seizures (NEAD) have very different communication styles and that a range of interactional, linguistic and topical features can be detected in patients’ talk, which can help clinicians to distinguish between epilepsy and NEAD.<sup>57,58</sup> A similar approach may offer important clues as to the affective or biological nature of a patient’s memory complaints.

## Conflict of interest

There are no conflicts of interest. Dr Blackburn, Sarah Wakefield, Dr Harkness, Professor Venneri and



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