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Faculty of Old Age Psychology

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Psychology Specialists Working With Older People

AIMS

- to promote opportunities for the exchange of knowledge and expertise between members;
- to promote a greater appreciation of psychological factors in ageing;
- to advise and participate in matters of teaching and training;
- to stimulate research and disseminate research findings;
- to act in an advisory capacity on issues relating to the well-being and provision for care for older people;
- to foster an exchange of information and ideas with other professional and voluntary groups.

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Letter from the Editor

Louisa Jackman

THIS *Newsletter* is the bi-product of two separate meetings of two strands of applied psychology. The first paper is the culmination of the work presented at a day on Mild Cognitive Impairment, organised by the North East branch of PSIGE. The following papers represent the presentations from the recent joint meeting of PSIGE and the Division of Neuropsychology in London. We have recognised as a national committee the need for more joined-up working in the CPD events we facilitate. The thrust for this has emerged from the current economic climate making it both more expensive to put on an event, and more difficult for people to finance events through work. This naturally leads to a slump in attendance which we learned to our cost from the conference that did not happen in Wales. However, good things certainly emerge from adversity as it has proven extremely successful to host these two joint events, combining expertise and application. A big thanks to our contributors who stepped in at the last moment to provide papers for this issue. The planned Wessex edition will now appear in January. Our next edition is the Scottish conference edition.

Finally, thanks from me to our outgoing chair, Don, and welcome to Cath in his place.

PS: Please note my change of name following my recent marriage!

Letter from the Joint Convenors

Cath Burley & Phil Yates

AS Joint Convenors of the Joint PSIGE/DON training day on 10 June Phil and I are delighted to write a brief introduction to this Special Edition of the *PSIGE Newsletter* on Neuropsychology. The combination of articles from the presenters of the PSIGE Mild Cognitive Impairment workshop in Newcastle on 29 March and the Developments in the Neuropsychology of Neurodegenerative Disorders day on 10 June gives a timely update for clinicians both in neuropsychology and old age psychology about assessment and intervention with a range of neurological disorders.

For many trainees and newly-qualified clinicians the minefield of distinguishing between MCI and possible dementia is very difficult. These articles help to clarify the process and the thinking behind the assessment. For all clinicians, distinguishing between the enormous range of possible neurological disorders, choosing the correct assessments and carrying them out accurately to help to clarify strengths and weaknesses and contribute to the diagnostic process is one of the most challenging and rewarding aspects of the role of a psychologist. These articles, from experts in their field, bring us the best in current thinking.

The process of jointly planning, organising and delivering a training day including PSIGE and DON members has been a different challenge, but one we hope to repeat, given the interests we all share.

Cath Burley
PSIGE Vice Chair, Chair Training and Development Sub-committee,
Chair of the Training Sub-committee.

Phil Yates
DON, PQT Cordinator.

Letter from the Past Chair

Don Brechin

HELLO EVERYONE.
This is my last letter to you as PSIGE Chair, so it allows me the opportunity to review the last two years.

When I came into the role in 2009, we had just cancelled our annual conference for the first time and the British Psychological Society office then immediately withdrew our office services. Clearly, not the easiest start. However, it was an opportunity to look at how we take forward our work in the future and, as an immediate response, the national committee met with the Geographical Group Convenors to clarify our purpose and seek support for how we take forward our activities as a faculty. This was enormously helpful, and provided a clear steer that we needed to work to support each other as clinical psychology practitioners and researchers working with older people, and to do this within the Division of Clinical Psychology and the Society.

The biggest areas of work that we have engaged in over the last two years are the IAPT for Older People workstream and the Dementia workstream. Fuller updates on these workstreams are available in the AGM papers that you will have received in May, but suffice to say that both of these are enormously productive, and PSIGE members are now in contact with government departments and other bodies in England, Scotland and Wales to take these work programmes forward. Form me, it has been a great privilege for me to be Chair at a time when national developments around access to psychological interventions for older people have become national priorities, and it has been good to see how the hard work of PSIGE members has been accepted and taken on board by policy makers and will feature within these national work programmes. I am also very pleased to say that our business is now very much part of the DCP's business. Our priorities now feature within the DCP strategic objectives, our work programmes have been funded by the DCP, and we have the support of the unit directors in taking our work programme forward at all levels. We also receive a great deal of support from the Society's office, and I am thankful to Annjanette Wells and Helen Barnett for their work in supporting us.

Another key area of work for the committee is CPD, and my thanks go to the Training and Development Subcommittee for all their work in ensuring that there is a comprehensive and successful national and regional programme of events available. This will continue, but we always need members to support this so please contact the committee with thoughts and ideas for events.

Finally, I would like to thank the national committee members for their hard work throughout the last two years. It has been a privilege to work with such a diligent, dedicated and good humoured group of people. I would also like to thank the numerous members who continue to support the work of PSIGE through their activities, whether it be organising local groups, putting on CPD events, taking part in working groups to develop PSIGE documents, or simply championing the work of psychologists with older people. You are too numerous to mention individually, but thank you. It has been a wonderful experience for me to be the Chair of our faculty for the last two years, and I pleased to hand over to Cath Burley as the new Chair and wish her every success.

Best wishes.

Don

Please note: From 11 March my new email address is: DonaldBrechin@nhs.net

An introduction to Mild Cognitive Impairment

Papers from the PSIGE Northern Group Workshop on Mild Cognitive Impairment, Newcastle-upon-Tyne, March 2011.

Definitions and biological aspects

John-Paul Taylor

THERE HAVE BEEN many attempts over the years to define a subclinical state of cognitive dysfunction which exists somewhere between normal ageing and a frank dementia (Table 1). However, diagnostic criteria, nosological status and how one defines the boundaries of this state continue to pose difficulties for both clinicians and researchers alike. Mild Cognitive Impairment (MCI) represents the most recent attempt to define this subclinical state.

Petersen et al. (1999, 2004) initially defined MCI as:

- Memory complaint by patient, family or physician.
- Normal activities of daily living.
- Essentially preserved general cognitive abilities.
- Objective impairment of cognitive function (below 1.5 or 1.0 SD of age mean, i.e. seven per cent or 16 per cent of normal population).
- Clinical dementia rating score of 0.5.
- Not clinically demented.

More recently, a number of subtypes of MCI have been proposed:

Amnestic mild cognitive impairment, single domain
Isolated memory impairment of more than 1.0 SD compared with the age- and education-specific norms, and no difficulty in any other area of cognitive functioning.

Amnestic mild cognitive impairment, multiple domain
Two or more cognitive domains are impaired, one of which is memory impairment (impairment of more than 1.0 SD below the mean of the respective age- and education matched population).

Non-amnestic mild cognitive impairment, single domain
Impairment in a single domain other than memory of more than 1.0 SD.

Non-amnestic mild cognitive impairment, multiple domain
Impairments in two or more domains of more than 1.0 SD but no memory impairment.

However, debate continues in many of these areas. For example, the need for subjective memory complaint has been questioned, for example, some subjects may not be aware/have insight into their deficits. Is corroboration by informant needed in those cases and what happens when there is no-one? To what extent are co-morbid conditions that may be associated with cognitive impairment excluded?

Table 1: Examples of different definitions that have been used to define a subclinical cognitive impairment state between normal ageing and dementia.

1962	Benign senescent forgetfulness
1986	Age-associated memory impairment
1993	Mild cognitive disorder
1994	Age-associated cognitive decline
1994	Age-related cognitive decline
1994	Mild neurocognitive disorder
1999	Mild/minimal cognitive impairment (MCI)

Derived from Gauthier, Reisberg, Zaudig, Petersen, Ritchie, Broich, Belleville, Brodaty, Bennett, Chertkow, Cummings, de Leon, Feldman, Ganguli, Hampel, Scheltens, Tierney, Whitehouse, & Winblad, 2006; Collie & Maruff 2002; Ritchie, Artero & Touchon, 2001.

On the measurement side, MCI criteria require general cognitive function to be 'essentially normal/preserved' and that activities of daily living are 'largely intact'. However, this allows patients with MCI to have minor impairments and thus how does one define an acceptable level of impairment objectively? In addition, if they have significant cognitive impairments in a second, non-memory domain they may well reach criteria for dementia.

Epidemiology of MCI

Making a diagnosis of MCI requires a comprehensive clinical and neuropsychological assessment of the patient. The definition therefore does not lend itself well to prevalence estimation studies using retrospective data and there are large disparities in rates depending upon the selection sample considered, for example, hospital vs. community setting. Prevalence estimates vary between three to 25 per cent of older samples and incidence rates of eight to 77/1000 person years. Another controversial issue has been the observation that up to 50 per cent people with MCI at baseline appear to return to normal after one year (Ritchie et al., 2001; Ganguli Dodge et al., 2004).

Prospective studies suggest that the prevalence of MCI in people over the age of 70 is between 14 to 18 per cent (Petersen et al., 2009). In addition, the rate of progression to dementia in memory clinic or hospital samples is now estimated to be approximately 10 to 15 per cent/year. In community samples, progression rates are around six to 10 per cent; still much higher than the expected general population incidence rate of dementia of one to two per cent.

Factors which influence the occurrence of MCI and its potential progression to dementia

Clearly age is a major risk factor for the development of MCI, particularly if MCI is seen as an at-risk state for incipient dementia. However, it is important to recognise that there are numerous other factors such as low educational achievement, underlying hypertension, untreated depression and medications that can predict the occurrence of MCI. Indeed the latter iatrogenic effect is particularly pertinent given the high frequency of polypharmacy in the older adult and the significant effect of a large range of drugs on cognition, particularly those agents which have an anticholinergic effect. Antiemetics, antispasmodics, bronchodilators, antiarrhythmic drugs, antihistamines, analgesics, antihypertensives, antiparkinsonian agents, corticosteroids, skeletal muscle relaxants, ulcer drugs, and

psychotropics all have potential effects on cognition which can give rise to a stable non-progressive MCI (Ancelin et al., 2006).

Some, but not all of these factors, including genetics, are also predictors for the progression of MCI to dementia (see, for example, Artero et al., 2008).

Biomarkers in MCI

Determination of biomarkers in MCI and identification of those individuals with MCI who are most likely to progress to dementia has been the focus of intense research interest in recent years. A wide variety of different approaches have been considered ranging from neuroimaging through to the characterisation of specific cerebrospinal fluid components (examples included in Table 2). However, no biomarker as yet has proved definitive, and it is likely that algorithms that combine neuropsychological assessment with appropriate neuroimaging and chemical biomarkers are likely to be the best predictor for MCI progression (Petersen et al., 2009).

Conclusions

The definition and diagnosis of MCI remains a controversial area but consensus on the criteria is building. Currently, the amnestic subtype MCI is probably the best defined entity. It is a clinically important group at risk of progression to Alzheimer's dementia. The nosological status of non-amnestic MCI remains to be defined but may direct suspicions to a non-AD dementia. In addition there is a further tendency now in the literature to separate MCI patients into progressive MCI and stable MCI with the former representing a likely prodomal or early stage in Alzheimer's disease. A future development in the refinement of diagnosing MCI is likely to

Table 2: Examples of biomarkers in MCI.

Neuroimaging	● Volumetric magnetic resonance (MR) imaging studies – have demonstrated subtle hippocampal, entorhinal, and whole brain atrophy with ventricular enlargement in MCI patients who progress to dementia. ● Functional studies – positron emission topography (PET) and single photon emission computerised tomography (SPECT) show reduced metabolism/ perfusion respectively in MCI patients in posterior cingulate and parietal regions and may provide dynamic information on progression. ● Amyloid imaging – Pittsburgh compound B (PIB) PET imaging studies have shown that between 52 to 87 per cent of MCI patients (Quigley, Colloby & O'Brien, 2010) show elevated PIB binding, depending on the criteria used to diagnose MCI. While longitudinal evidence is still lacking, this technique may provide an early biomarker of risk of progression to Alzheimer's dementia.
Genetics	● No definitive gene of large effect has been demonstrated for late onset Alzheimer's disease although the presence of the apolipoprotein E4 polymorphism increases the risk of conversion to dementia from MCI (OR 3 – 4).
Cerebrospinal fluid (CSF)	● Alzheimer's disease is associated with raised tau (especially phosphorylated tau or P-tau) and low beta-amyloid (A 1–42) in CSF. In MCI similar alterations have been observed albeit less marked. One recent longitudinal study (Mattsson, et al, 2009) has suggested that a combination of A 42/P-tau ratio and total-tau identified incipient AD in MCI with a sensitivity of 83 per cent and specificity 72 per cent.

be the inclusion of behaviour or psychological symptoms, for example, anxiety, apathy, irritability, etc. Assessment of these 'non-cognitive' symptoms may enhance the prediction of progression to dementia including non-Alzheimer dementias (Petersen et al., 2009). Finally biomarkers in MCI, particularly if combined with in-depth clinical assessment show significant promise, but more research is needed.

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Neuropsychological assessment of Mild Cognitive Impairment

Daniel Collerton

DESPITE ADVANCES in other investigatory techniques, neuropsychological testing has remained the gold standard for identifying MCI; distinguishing it from normal ageing at the upper end, and mild dementia at the lower (Diniz et al., 2008; Jacovaa et al., 2007). Although more advanced screening tests such as the Addenbrooke's Cognitive Examination – Revised (ACE-R), or the Montreal Cognitive Assessment (MOCA) can distinguish between normal ageing and mild dementia with reasonable effectiveness, they struggle to make reliable classifications between these two states and the intermediate classification of MCI. More complicated still, in clinical practice, apparent cognitive change is often associated with poor physical or mental health; factors which are particularly likely to lead to misclassification.

The content of assessment

MCI definitions require objective impairment in memory, or in any of the cognitive domains of learning, memory, attention, thinking, language, or visuospatial function for diagnosis, while the NINCDS-ADRDA clinical criteria for probable AD require abnormal performance in two or more areas of cognition including orientation, memory, language, praxis, attention, visual perception, and problem solving. Thus a comprehensive assessment should cover at least these cognitive areas, in addition to the clinical rating of disability derived from the Clinical Dementia Rating Scale. Different clinicians will have preferences for particular tasks, but recent age norms are essential in gaining reliable estimates of performance.

Imprecise assessments

However, even as the gold standard, neuropsychological assessment is not entirely free from tarnish. Difficulties arise both from the definition of MCI itself, and from the imprecision of our neuropsychological instruments.

MCI is partially defined as performance on one or more cognitive tasks at least one (or 1.5 in some definitions) below age norms. But there will always be a proportion of the normal population, either 16 per cent or seven per cent depending upon the cut-off point, who have scored in that range throughout their lives. Hence, scores alone can not distinguish between lifelong poor cognitive performance and acquired MCI. Added to this, our neuropsychological

instruments have large margins of error (though repeated measures can reduce this). As an instance, a score at the 50th centile on the Wechsler Adult Intelligence Scale, an IQ of 100, could with 95 per cent confidence reflect a true level of ability anywhere between the 16th and the 84th centile.

Thus there will always be overlaps in scores – at the upper limit between people who have normal cognition for their age and those who have a mild cognitive impairment, and, at the lower limit, those with a mild cognitive impairment and those with mild dementia. Dealing with any classificatory overlap requires a trade off between two types of errors; missing true problems, and identifying false problems. The size of overlaps that we see between MCI and normality and mild dementia on cognitive tests scores means that any test which uses a cut-off score is acutely sensitive to where that cut-off is placed.

The efficacy of any classification system can be estimated using sensitivity – the proportion of cases that a test correctly identifies – and specificity – the proportion of non cases that a test correctly identifies. With scores on cognitive assessments; either one or the other can be high, but both can not. Table 3 shows data from Busse, Angermeyer and Riedel-Heller (2006) which illustrates this type of trade off – in this instance showing how different diagnostic criteria for cognitive impairment short of dementia are able to predict progression to dementia. Those criteria which are poorest in predicting progression are best at predicting non-progression and vice versa. The best trade off, in this instance AACD-modified, has an error rate for predicting progression of one-in-three, and of predicting non-progression of one-in-eight; both of which are problematic in clinical practice.

The classic means of improving classification systems is to move from the use of a cut-off score alone, to incorporating how far away from a cut-off someone scores. The likelihood ratio takes a particular score and uses this to estimate the chances of the person falling into a particular group. This is what most clinicians do – the greater the difference between a person's current performance and estimates of his or her previous performance, the more likely it is that we will conclude that they have a true impairment. However, the position of MCI between normal ageing and mild dementia makes this more difficult – the more confident that we are that a person does not have normal cognition, the less confident we are that they do not have a mild dementia.

A further, often under recognised, factor is the strong effect of the frequency of a disorder on the accuracy of assessments (Sackett, 1991) Sensitivity and specificity are properties of a test. Intuitively, most people think that a test is accurate or not regardless of which population it was assessing. However, when disorders are either very common or very rare, the same test result has very different implications. As disorders

Table 3: Effects of diagnostic criteria on sensitivity and specificity for predicting progression to dementia.

Diagnostic criteria	Sensitivity	Specificity
Mild Cognitive Impairment	10.1	97.6
Mild Cognitive Impairment without subjective memory complaint	11.2	95.5
Age Associated Cognitive Decline	37.1	95.2
Age Associated Cognitive Decline without subjective memory complaint	61.8	87.4

become rarer, our ability to identify when they occur (what is called the positive predictive value – PPV) falls substantially, but our ability to rule them out (the negative predictive value) rises to compensate – and vice versa. To illustrate: if a disorder occurs in only 10 per cent of people assessed, a score of 1.0 standard deviation below normal (the cut-off for MCI) has a PPV of 0.25 (i.e. only a quarter of people who are identified as having MCI actually have it), and a NPV of 0.95 (i.e. only one person in 20 who is identified as not having MCI actually does have MCI). Reverse the proportions of MCI and non MCI in the assessed population, and PPV and NPV reverse – we become much better at indentifying when it is present, than when it is absent.

However, when we look at a population in which there is a high degree of uncertainty before assessment, for example, in which half of referrals have MCI and half do not, the power of assessments becomes much greater. In this case a score on the 1.0 standard deviation line still gives a PPV or NPV of 0.5, but even a slightly better or worse score shifts the odds considerably. Thus, a score of only 0.5 standard deviations down reduces the chance of having MCI to one in five, whereas a score of 1.5 down raises it to 75 per cent.

Putting all of this together, neuropsychological assessment is most effective when there is genuine uncertainly before the assessment is done.

Implications for clinical practice

The above shows that even in the best hands, neuropsychological assessment alone can never be entirely accurate. Therefore, a key aim is to develop a system which can support patients through unavoidable uncertainty and revisit decisions to check for misclassifications. Experience within a specific population so that base rates of normal cognition, MCI, and mild dementia can be estimated allows the relevance of specific test scores to be judged. The ability, which only comes with experience, of estimating the likelihood of MCI before doing the assessment, and then interpreting the scores in the light of that knowledge is the most essential skill to acquire.

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Neuropsychology of Mild Cognitive Impairment (MCI): Characterisation, progression and outcome in a Scottish sample

Claire Donaghey

THE TWO FACTORS that most commonly separate MCI from clinically diagnosable early dementia are: (1) presence of impairment in one domain only, usually memory; and (2) absence or insufficient evidence of functional decline. Having said that, clinically speaking, determining the point at which a patient moves from ageing normally to meeting criteria for MCI and from meeting criteria for MCI to meeting criteria for very early Alzheimer's disease can be difficult and is a highly clinically subjective process.

Our study comprised of 46 patients with single and multiple domain amnestic MCI. We followed up these patients for a maximum of five years using a comprehensive battery of neuropsychological tests covering each of the primary domains of cognitive function. Broadly speaking, the study aims were two-fold. Firstly, to examine differential diagnostic utility of neuropsychological measures in the assessment of early Alzheimer's disease (AD) and MCI. Secondly, to determine the prognostic utility of neuropsychological task performance as a predictor of which MCI participants would and would not develop dementia over the course of the study. The battery of tests was also administered to a group of 24 healthy age and IQ matched controls, 20 patients with very early stage AD and 20 elderly patients with depression. This allowed us to examine baseline performances cross-sectionally. We re-administered the battery of tests to 16 of the 24 healthy controls after a mean two-year interval to examine retest reliability.

Characterisation of amnestic MCI (aMCI)

We were interested in determining whether amnestic MCI (aMCI) patients could be differentiated from healthy elderly age and IQ matched controls on the basis of their neuropsychological task performances (Lonie et al., 2008). In addition, the team wanted to find out which neuropsychological measures were most useful in differentiating aMCI from the healthy elderly and whether there was evidence for a more generalised impairment of cognition. That is, beyond a sole episodic memory impairment.

We compared the mean performances of an age and IQ matched control group to that of the aMCI cohort and found statistically significant lower mean performances of the aMCI group on the vast majority of neuropsychological measures. In particular, the aMCI group performed poorly on the ACE, measures of verbal free recall and object and face naming ability. We then examined the pattern of performance across cognitive domains and individual cognitive tasks by looking at the proportion of aMCI patients who were performing below the 10th percentile of control subjects on each of the neuropsychological measures. Not surprisingly, and indeed by definition, the greatest proportions of aMCI patient performances below 10th percentile were seen in association with episodic memory measures and a large number of aMCI patients were performing below the 10th percentile of controls on two or more of these measures

(i.e. HVTL-R total recall, HVLT-R delayed recall, ACE delay). More surprisingly, 76 per cent of aMCI patients were also performing below the 10th percentile of controls on one more of the semantic memory measures included in our battery (i.e. BNT, GFT, Category fluency). There was also a notable disparity in the numbers of aMCI patients showing impairment of an executive nature as a function of the specific measure employed. Thirty-three per cent of aMCI participants performed below the 10th percentile of controls on the Trail Making Test B as compared to just five per cent and seven per cent on the dual task and lexical fluency (F, A & S) respectively. This suggests that there is a good deal of variability in the sensitivity of executive measures to aMCI, presumably in accordance with the aspect of frontal systems functioning tapped. These findings highlight the extent to which the identification of aMCI and categorisation of its subtypes is reliant on selection of cognitive assessment measures (Lonie et al, 2008).

Cognitive screening in aMCI

One of the first things we looked at was the comparative abilities of two of the most commonly used cognitive screening measures, the ACE and the MMSE, to differentiate between aMCI, early AD, depression and healthy elderly. We were keen to determine how useful cognitive screening is in detecting and differentially diagnosing aMCI and very early AD among the healthy and depressed elderly. More specifically, whether the sensitivities and specificities of these two screening measures were comparable. The comparison was based on the MMSE and the ACE performances of 46 patients with aMCI, 20 with early AD, 20 with depressive symptoms and 24 healthy elderly. The groups were matched for age and gender, however, the NART estimated FSIQ for the early AD group was statistically significantly lower than that of the healthy control and MCI groups.

After adjusting for the lower mean NART FSIQ score of the early AD group, both measures differentiated the early AD patients from all other groups. However, only the ACE differentiated the aMCI patients from the healthy controls. The lower mean performance of the depression group, relative to the healthy controls on the ACE, no longer reached statistical significance following adjustment for multiple comparisons. The mean performance of the depression group was not significantly different from either the control or the aMCI group on either screening measure.

Sensitivity and specificity

When we examined the sensitivity and specificity values that correspond to the most commonly applied cut-off scores used for the MMSE and the ACE. The most favourable pairings are consistently seen in association with the higher cut-off value, i.e. 88/100 for the ACE, 27/30 for the MMSE. In differentiating early AD and aMCI from healthy elderly controls, the highest cut-off values give the most favourable combination of sensitivity and specificity. However, even using the higher cut-off value (i.e. 88/100) for the ACE, sensitivity to aMCI remains unacceptably low at 48 per cent. This suggests that more than half of all patients who fulfil criteria for aMCI may perform inside even the most stringent set of normal limits on comprehensive cognitive screening.

Cognitive screening using the MMSE and the ACE appears justified as a means of detecting early AD and both measures perform similarly in this regard when their respective highest cut-off points are employed. However, both measures lack sensitivity to aMCI.

The use of fluency in differential diagnosis between AD and aMCI

Aside from cognitive screening measures like the ACE and the MMSE that give the clinician a feel for a patient's global or overall level of cognitive functioning, sometimes additional bedside cognitive tasks are administered as part of a psychiatric consul-

tation. Among the most common, are measures of verbal fluency: lexical fluency (usually FAS, CFL or P as in the ACE) and category fluency (usually animals) but sometimes an average of a number of different categories such as animals, fruits and vegetables. The usual pattern of performance on fluency tasks among the healthy elderly and adult populations is performance on category fluency tasks better than lexical fluency. It has been noted that the opposite pattern tends to occur in AD. Because of this qualitative difference in fluency performances of healthy elderly and AD patients, together with evidence to suggest that semantic fluency is impaired in aMCI where lexical fluency is intact, we were interested in determining whether fluency discrepancy scores were a useful and accurate means of differentiating early AD from aMCI. More specifically, we wanted to find out whether the pattern of performance on the two types of fluency tasks differed in early AD and aMCI relative to healthy and depressed elderly controls (Lonie et al., 2009).

The fluency discrepancy score was derived from the number of animals generated in one minute minus the number of P words generated in one minute (part of the ACE). This score was then compared with a measure of episodic memory (HVLT-R delayed recall). In the case of the aMCI comparisons with controls and the depression group, the fluency discrepancy scores were more accurate in classifying these groups of patients than delayed verbal recall scores from an episodic memory measure. Classification accuracy among early AD and healthy controls remained high for the fluency discrepancy scores but was in this case less good than delayed recall.

In summary, the pattern of performance on the two types of verbal fluency task appears to differ in early AD and aMCI patients relative to the healthy elderly with a greater number of P words than animals retrieved within a one-minute time period. Observation of the discrepancy in performance on the two types of fluency task provides an equally good if not more accurate means of discriminating aMCI patients from the healthy and depressed elderly than a measure of delayed verbal recall. This suggests that assessment of domains in conjunction with or other than that of episodic memory alone may be helpful in identifying aMCI (Lonie et al., 2009).

Longitudinal outcome in aMCI

There were a number of questions we wanted answered in terms of longitudinal outcome for those who have been diagnosed with aMCI. We wanted to determine what proportion of aMCI patients converted to dementia. Secondly, of those that did not convert to dementia, how many remained cognitively impaired and how many displayed progressive cognitive decline as opposed to a static course (Lonie et al., 2010).

Using our study criteria, 31 per cent of aMCI participants converted to dementia over the course of the study. This equates to a rate of 14 per cent per year. Of the remaining 69 per cent of aMCI participants who did not meet study criteria for dementia, half returned normal psychometric performances and the other half did not, i.e. still met criteria for aMCI. For those who met criteria for aMCI at the study endpoint but not dementia, half experienced further cognitive decline across the course of the study and half remained stable. When using a clinical diagnosis, as evidenced in their medical notes, rather than our study criteria, a slightly higher proportion, i.e. 41 per cent of aMCI participants had received a clinical diagnosis of dementia. However, the conversion rate per annum was slightly lower as a result of the longer follow-up period. In most cases the clinical diagnosis was AD followed by a diagnosis of dementia of mixed aetiology.

In summary, within our study sample of aMCI patients 41 per cent met criteria for dementia after four years. Of those who did not convert to dementia, 58 per cent remained cognitively impaired. Of this 58

per cent, half (i.e. 50 per cent) displayed evidence of progressive cognitive decline across the course of the study (Lonie et al, 2010).

Neuropsychological predictors of aMCI

Finally, we wanted to determine whether performance on any specific neuropsychological measures or combination of measures would be helpful in predicting which aMCI participants would convert to dementia. More specifically, we were interested in determining whether the initial baseline neuropsychological performance of aMCI non-converters differed from that of aMCI converters. If this was the case, then we wanted to identify which measures were more sensitive. Finally, the team also wanted to determine how useful neuropsychological measures were in a prognostic sense.

Following corrections for multiple comparisons, performance on two neuropsychological measures at baseline retained significance in differentiating between MCI converters and MCI non-converters. The two measures were the ACE and the HVLT-R-discriminative index score (Lonie et al., 2010). Each of these measures were entered into a backward stepwise logistic regression equation alongside the clinical variables of age, NART IQ and months of follow-up in a bid to determine the extent to which they added over and above such information to the prediction of outcome in aMCI. The results showed that the performances on the ACE along with the HVLT-R-discriminative index were able to discriminate those who converted from those who did not with 74 per cent accuracy (Lonie et al., 2010).

Conclusions

The findings from this longitudinal study have demonstrated that aMCI can be differentiated from healthy elderly controls based on their performance on measures assessing episodic memory and semantic memory. In addition, the ACE and the MMSE have been shown to be useful screening tools to differentiate early AD from controls. However, this was not the case for aMCI, as both measures lacked sensitivity. Findings also suggest the use of the higher cut-off scores for both screening tools. Fluency discrepancy scores were also shown to be useful to use to differentiate between early AD, aMCI, depression and healthy elderly controls. In terms of longitudinal outcome, 41 per cent of aMCI patients converted to dementia over a four-year period. Finally, aMCI patients who later received a diagnosis of dementia (i.e. converters) performed poorly on the ACE and the HVLT-R Discriminative Index at baseline assessment. The combination of the performance on these two measures at baseline was able to predict those who converted to dementia with 74 per cent accuracy.

Further research is required to determine whether the findings from this study can be replicated using different study cohorts. This would increase the generalisability of the study findings. As the ACE is no longer commonly used in clinical practice and has been superseded by the ACE-R, this could be an area for further research.

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Motor Neurone Disease: Not only a disease of the motor neurones!

Sharon Abrahams

MOTOR NEURONE DISEASE (MND) is a rapid and fatal neurodegenerative condition and although traditionally viewed as a disease solely of the motor system, recent years have seen a substantial increase in the awareness of cognitive change in the disease. The most common form – Amyotrophic Lateral Sclerosis (ALS) is diagnosed on the basis of both upper and lower motor neurone degeneration. Peak age of onset is 58 to 63 and patients experience progressive muscle wasting and spasticity which at first may be focal beginning in the arms/legs/mouth (bulbar) region. The disease typically spreads to other regions and death occurs within 30 months of onset of symptoms in the majority of patients (Kiernan et al., 2011). Patients experience a range of physical symptoms including severe limb weakness, dysarthria and dysphagia, and cognitive deficits and behaviour dysfunction are now viewed as an integral part of this multisystem disorder (Andersen et al., 2010; Tsermentseli & Goldstein, in press).

Recent evidence points to considerable overlap between MND and Frontotemporal Dementia (FTD), the second most common dementia in those under 65, and a clinico-neuropathological continuum has been proposed (Seelaar et al., 2011). Fifteen per cent of MND patients suffer from a full-blown dementia syndrome (MND-Dementia) which is of a frontal type (Neary, Snowden & Mann, 2000). FTD consists of three distinct clinical syndromes behavioural variant (bvFTD), progressive non-fluent aphasia (PNFA) and semantic dementia (SD) (Neary, 1998) and ALS-Dementia most strongly resembles bvFTD. MND may precede the dementia, develop concurrently or FTD may be the presenting complaint. MND is present in up to 10 per cent of FTD cases (Neary, Snowden & Mann, 2000).

A clinical continuum from MND to FTD has been further supported by the finding of executive dysfunction in a further third of non-demented MND patients (Rakowicz & Hodges, 1998; Lomen-Hoerth et al., 2003; Ringholz et al., 2005; Elamin et al., 2011). These deficits have been found on a range of tasks including those tapping rule deduction, cognitive flexibility, attention, switching and monitoring (e.g. Wisconsin Card Sorting Test and Trail Making Tests) and letter and category fluency (Abrahams et al., 1997, 2000; Abrahams, Leigh & Goldstein, 2005; Gordon et al., 2010; Massman et al., 1996; Ringholz et al., 2005). The most striking and consistently reported deficit has been on letter fluency tests (Elamin et al., 2011; Abrahams et al., 1996, 1997, 2000, 2004; Abrahams, Leigh & Goldstein, 2005; Massman et al., 1996; Gordon et al., 2010). Using a version of this task which controlled for physical disability with incorporation of a writing/ motor control condition and the production of a Verbal Fluency Index (V*fi*) (Abrahams et al., 2000.) this deficit was shown to be independent of physical disability, and of language dysfunction (naming deficits) and has been related to executive dysfunction (Abrahams et al., 2000). This deficit is present soon after diagnosis (Abrahams, Leigh & Goldstein, 2005), is more pronounced in MND patients with pseudobulbar palsy (Abrahams et al., 1997), and in some familial forms of the disease (Wicks et al., 2009) and is absent in those with only lower motor neurone involvement (Wicks et al., 2006). The deficits have also been shown to correlate with eye-movement

abnormalities (ocular fixation) – a neurological marker (Donaghy et al., 2009). Moreover evidence of the underlying cerebral substrate for this deficit has come from a range of brain imaging studies (Filippi et al., 2010). Functional imaging activation studies using verbal fluency have revealed predominant dysfunction in dorsolateral prefrontal cortex and anterior cingulate gyrus in those with verbal fluency deficits using fMRI and PET (Abrahams et al., 1996, 2004; Kew et al., 1993); while ligand PET scans have shown a correlation between V*fi* and reduced neuronal receptor binding of Flumazenil in the inferior frontal gyrus (Wicks et al., 2008). Frontotemporal white matter abnormalities have also been shown in those with verbal fluency deficits using structural neuroimaging (Abrahams et al., 2005) and recent studies have in particular highlighted callosal involvement (Filippini et al., 2010).

Language changes have also been reported in some patients with in particular naming impairment (Rakowicz & Hodges, 1998; Massman et al., 1996; Abrahams et al., 2004). Spelling errors are being reported particularly in Japanese cases in which errors in the more phonological Kana writing system have been described (Ichikawa et al., 2010). Moreover some patients may display a full aphasia syndrome with problems with comprehension and expression and a particular deficit has been shown using the Test of Reception of Grammar which exceeds that found in typical FTD (Bak et al., 2001; Bak & Hodges, 1999, 2001). A deficit in the processing of verbs/actions as opposed to nouns/objects has also been described an related to atrophy of the motor cortex (Grossman et al., 2008).

There are now also increasing reports of changes in behaviour (Lomen-Hoerth et al., 2003) with disinhibition (Murphy & Lomen-Hoerth, 2007) and apathy (Grossman, Bradley & Miller, 2007) being the most commonly reported symptoms. A recent study of 225 MND patients showed changes in the Frontal Systems Behaviour Scale in at least one domain (apathy, disinhibition, executive dysfunction) in up to 39 per cent of cases (Witgert et al., 2010). Insight appears intact in those with mild behaviour change in contrast to those with a full blown FTD syndrome (Woolley & Katz, 2010). Gibbons, Neary and Snowden (2008), described increased self-centred/selfishness and loss of interest/apathy as common symptoms. Of note, some questionnaires standardised on the head injured population may exaggerate dysfunction in patients with physical disability. Additional factors of reactions to progressive disability and a terminal diagnosis should also be considered.

Recent studies have also shown deficits in social cognition similar to those found in bvFTD which may underlie these behavioural changes. Two studies have demonstrated evidence of a deficit in Theory of Mind in MND. Gibbons et al. (2007) showed an impairment in interpreting cartoons and stories which involved understanding a character's mental state and included false beliefs and deception similar to that found in FTD. More recently Girardi et al. (2011) revealed impairments on a range of tasks of social and emotional cognition. The most sensitive of these was a simple Theory of Mind task in which participants had to infer preference judgements about a face by using a common social cue, eye-gaze. An analysis of the errors made revealed that MND patients' responses were more often influenced by their own preference rather than that of the face. Hence, MND patients had difficulty inhibiting egocentric responding to stimuli leading to difficulties in using a simple social cue. MND patients also showed evidence of increased behavioural dysfunction and measures of Apathy were significantly related to poor performance on this task.

Deficits in affective decision making have also been described with poor performance on a modified version of the IOWA gambling task (Girardi et al., 2011). Moreover dysfunction of more primary emotional processing has been revealed with problems in facial recognition of emotions (Girardi et al., 2011; Zimmerman, Simmons & Barrett, 2007),

(although not consistently (Papps et al., 2005)) and in complex and simple emotional understanding of expression (Girardi et al., 2011). Further studies which have investigated the social significance of emotional processes have shown that MND patients rated faces as more approachable than controls (Schmolck & Schulz, 2007), showed a more positive valence towards emotive social situations and demonstrated a more even arousal state (Lule et al., 2005). Papps and colleagues demonstrated that this dysfunction was not a consequence of general attentional/memory dysfunction with the demonstration of a selective failure to show enhanced recognition of emotional words despite superior memory for neutral words (Papps et al., 2005). Finally the relationship between emotional processing deficits and emotional lability (loss of control of emotional expression) which occurs in 19 to 49 per cent of MND patients, has not yet been investigated although Palmieri et al. (2009) demonstrated that levels of lability did not relate to other cognitive deficits indicating distinct neuronal pathways.

The findings of cognitive and behaviour change in a subgroup of MND patients has implications for the everyday care. The findings predict potential difficulties in tasks relying on effective executive processes, such as those involving planning, organising and decision making, some language dysfunction which may hinder communication, and behaviour change and social cognition deficit which may affect interaction with others. MND patients often experience other changes to their lives as a consequence of their physical disability such as stopping work and an increased reliance on others for physical tasks. This will impact on daily activities and the need for fast effective cognition. However, problems may emerge in new situations such as learning to cope with increased disability or when learning to use new aids such as lightwriters or feeding tubes. Clinicians should be directing appropriate strategies to educate those involved in the direct care of MND patients. These may include strategies to cope with executive dysfunction, language changes and behaviour. Information for other health and care professionals can be found in a new booklet entitled Cognition and MND published by the Motor Neurone Disease Association.

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Understanding changes in cognition due to multiple sclerosis

Steven Meldrum

MULTIPLE SCLEROSIS (MS) is a neurodegenerative inflammatory – demyelinating condition of the central nervous system with a variable clinical course involving several disease subtypes. Multiple sclerosis means 'many scars' and disease progression follows four main subtypes, however, the most common subtype is relapsing-remitting multiple sclerosis, characterised by acute attacks of neurological dysfunction, followed by partial or complete recovery (SNAP, 2000). Current prevalence rates in the UK are calculated at 150 to 200 cases per 100,000 of the population with the highest prevalence in Scotland. The pathology and ensuing disability is linked with the process of axonal demyelination, remyelination, and axonal and synaptic degeneration (Orhun et al., 2005). The accumulating lesion profile in the brain is diffuse, affecting central nervous system functioning, motor systems, and multiple brain regions involved in diverse functions.

Characteristic changes in areas of the multiple sclerosis brain are lesions in the subcortical white matter fibres, particularly in the periventricular areas, corpus callosum, and infratentorial areas (Filippi & Rocca, 2007). Long-term axonal and myelin loss can contribute, along with other tissue loss, such as grey matter, to atrophy within the MS brain (de Stefano et al., 2007). The clinical manifestation of the disease varies considerably between sufferers and is related to lesion site, brain atrophy and subsequent effects on function.

Cognitive impairment is a well recognised and now accepted major symptom of multiple sclerosis with prevalence rates estimated to be anywhere between 45 to 65 per cent, (Rao, 1995). However, it is only in the last few decades that this has become widely accepted and investigated (Bobholz & Rao, 2003). Due to predominately subcortical white matter aetiology in the MS brain, the cognitive domains or processes most affected are attention, speed of processing, and memory (DeSousa, Albert & Kalman, 2002). More cortical subserved functions such as language ability are generally preserved with findings suggesting that widespread damage to white matter leads to a functional disconnection between different cortical areas and deep grey matter structures. Axonal fibre damage can also lead to a slowing in neuronal communication affecting the speed of cognitive processes (DeLuca et al., 2004). Due to the somewhat unpredictable and quasi-random distribution of lesions in the MS brain, presentation and progression of cognitive deficits vary enormously between sufferers (Gainotti, 2006). The anatomical distribution of inter-individual MS pathology, with respect to functionally eloquent neural areas and networks, determines the clinical phenotype (Guttmann, Meier & Holland, 2006).

Understanding brain imaging in multiple sclerosis

During the last 10 years MRI has been increasingly utilised in the study of MS and 'the overall landscape has dramatically improved compared to that of the mid-1990s' (Filippi et al., 2007). The aetiology and relationship between MS brain changes and cognitive impairment have been of great interest in the last decade with many researchers seeking to establish a link between MRI detectable abnormalities and the association with physical and cognitive disability (Rovaris, Comi & Filippi, 2006).

In multiple sclerosis conventional T2 weighted lesions appear hyperintense or very bright against the non-diseased tissue and are the most readily visible MS lesions reflecting different pathologies of various stages including inflammation, oedema, and demyelination. This imaging method can be used to measure the total visible lesion volume. In a conventional T1 weighted scan, MS lesions appear hypointense and within the white matter areas these are known as 'black holes'. These types of imaging findings are thought to reflect more destructive pathology and axonal loss (Rovaris et al., 2006). Brain atrophy is used as an index to monitor pathologic evolution of MS activity and several studies have shown that brain volume is significantly reduced in patients and that cognitive impairment in MS may be related to decreasing brain volume rather than just increasing lesion load (De Stefano, Battaglini & Smith, 2007).

More contemporary non-conventional imaging analysis techniques such as Magnetisation Transfer Imaging and Diffusion Tensor Imaging allow assessment of tissue damage in what is known as Normal Appearing White and Grey Matter (NAWM, NAGM). Microscopic areas of damage are not readily detectable on T1 or T2 weighted images and these techniques can quantify more fully the extent of MS 'occult' pathology in the brain (Rovaris et al., 2006).

The magnetisation transfer imaging provides a ratio with which to determine the integrity of grey and white matter tissue in normal appearing brain tissue (NABT) and has highlighted the global central nervous system involvement in MS pathology (Filippi & Agosta, 2007).

Diffusion tensor imaging is a technique that allows assessment of the integrity of white matter tracts in the brain. This non-conventional image analysis is of great interest in multiple sclerosis as white matter plaques and subsequent disconnectivity between brain regions has been reported as being significant in multiple sclerosis related cognitive deficits (Miller et al., 1998; Goldberg-Zimring & Warfield, 2006). DTI is based on the impeded movement of water within axonal bundles due to myelin sheaths which leads to water diffusion parallel to the fibres: anisotropic diffusion. Neuropathological process, as in multiple sclerosis, that lead to microstructural changes in white matter and reduced axonal integrity are thought to interfere with normal anisotropy. MRI pulse sequences allow the assessment of white matter tracts and can show directionality and abnormal connectivity in white matter (Ge, 2006).

The imaging literature has elucidated our understanding of the underlying pathology and its relationship with cognitive impairment and reveals quite clearly that the model of cognitive dysfunction is not due to one primary variable such as white matter lesions but a combination of many pathogenic variables affecting the global brain. Cortical and subcortical atrophy along with disconnection of interneuronal networks within the cortex and white matter play a role in the cognitive decline.

Assessment of cognition in multiple sclerosis

The assessment of the cognitive dysfunction in MS has been evaluated with research highlighting optimal tests that should be used with this group to assess any potential cognitive impairment (Sartori & Edan, 2006). Recommendations have included test batteries with the following characteristics:

1. Tests independent of motor coordination and visuospatial ability.
2. Focus on attention, working memory, and speed of processing.
3. Brief administration to minimise confound of fatigue.

Sartori and Edan (2006) recommend a brief 30-minute test battery that includes the Paced Auditory Serial Addition Test (PASAT), new learning with the California Verbal Learning Test, and digit span backwards. Importantly, the authors recognise the confounding factor of depression impacting on cognitive test results. The PASAT has remained as one of the most

common neuropsychological test measures used in MS clinical evaluation and research studies and is a core measure of the Multiple Sclerosis Functional Composite. Deficits on the PASAT are one of the most robust findings in the neuropsychology of MS (Hoffmann, Tittgemeyer & von Cramon, 2007).

The Brief Repeatable Battery of Neuropsychological Tests (BRBNT) (Rao, 1990) is a well-established and frequently referenced assessment battery of cognitive change in MS (Gainotti, 2006). This compound of tests includes:

PASAT	*Measure of Attention*
Symbol Digit Modalities Test	*Processing Speed*
Selective Reminding Test	*Verbal Memory*
10/36 Spatial Recall Test	*Visuospatial Learning*
Word List Generation	*Verbal Fluency Task*

These tests were decided upon by administering a comprehensive neuropsychological test battery of 31 test indexes to 100 patients with MS and 100 matched healthy controls and selecting the tests on which the MS group were most impaired. Rao (1990) found that the final test selection demonstrated a sensitivity value of 71 per cent and a specificity value of 94 per cent in discriminating between cognitively intact and cognitively impaired patients with MS.

Understanding attentional problems in multiple sclerosis

Even though the cognitive profile in MS is often heterogeneous, a consistent deficit found in multiple sclerosis is deficits of attention and the ability to attend to more than one thing at the same time, divided attention (Bobholz & Rao, 2003). In addition, an intact attentional system is vital for the efficient processing of other various cognitive systems, for example, memory encoding. There is some suggestion that deficits in higher cognitive operations are actually secondary to primary attentional problems in MS. However the extant MS literature examining attentional dysfunction leaves unanswered questions as to the extent and nature of deficits (McCarthy et al., 2005).

When considering the concept of attention there are various theoretical cognitive models of attention and one of the most influential and frequently referenced theories is Baddeley's (1986) working memory model. He proposed a structural model involving multiple interactive components including a central executive controller that regulates and distributes the limited available attentional resources that a system possesses, and visuospatial and phonological slave systems (Baddeley, 2003). The slave systems are responsible for storage of modality specific inputs and one of the principle roles of a central executive system would be to efficiently allocate and manage attentional resources when two or more tasks are being executed simultaneously or when attention is divided between the visual and auditory slave systems (Baddeley, 1997).

Dual tasking or divided attention paradigms have been used extensively to study this fundamental property of an executive control system in various patient populations (Baddeley et al., 1997). Dual tasking designs involve performing two different tasks on their own and then concurrently, and comparing performance levels on one or more of the tasks. For example, established formats involve participants performing a verbal digit span task with a visual tracking task. In clinical populations, a dual task performance decrement is frequently observed and reported in the context of damage to an executive co-ordinator responsible for dividing and allocating attention; for example, in Alzheimer's disease (Logie et al., 2004).

Further, Baddeley, Bucks and Wilcock, (2001) found that Alzheimer's disease patients have a specific difficulty with dual task performance, even when controlling for

the general overall cognitive demand, suggesting that available attentional resources is not the problem, but how they are allocated is. Also, in a frontal lobe lesion patient group Baddeley et al. (1997) found that patients with dysexecutive syndrome showed impaired capacity for dual tasking.

In multiple sclerosis, evidence indicates that the multifocal lesion profile may affect brain areas which form the working memory substrate, leading to working memory impairment in the early stages of the disease (Pelosi et al., 1997). D'Esposito et al. (1996) investigated central executive functioning using dual tasking methodology comparing an MS group with a control group. In their study, the dual tasking paradigm involved performing a primary task concurrently with one of three secondary tasks.

The primary task involved a line orientation judgment with the concurrent secondary tasks being finger tapping, alphabet recitation, and humming a melody. Results showed that the MS group performed less well during the more demanding dual task conditions (humming a melody and alphabet recitation) than the control group. The authors concluded that the dual task decrement found in the MS group, reflected an impaired central executive of working memory resulting in difficulty allocating sufficient attentional resources to support concurrent task execution.

Paul et al. (1998) investigated several aspects of automatic and controlled attentional processing in MS using tests of focused and divided attention. They used the Posner spatial attention task (Posner, Cohen & Rafal, 1982), which involves visual stimuli presented with valid and invalid spatial cues, as a test of automatic processing and found that the MS group performed as well as the control group. The Paced Auditory Serial Addition Task (PASAT) was used as a test of divided attention in this study and the MS group performed significantly worse than the controls.

McCarthy et al. (2005) considered the profile of attentional dysfunction in MS using divided and sustained attention methodology across unimodal and bimodal visual and auditory trials. The authors developed two new measures of sustained and divided attention as part of their study and recognised that their approach to investigating divided attention was in contrast with conventional dual tasking methodologies where primary task decrement is measured during concurrent secondary task execution. In the divided attention task, targets were digits that were consecutive pairs either ascending or descending with a temporal delay between digit presentation in a trial.

Participants were required to divide their attention between retention of the first digit and presentation of the next digit. Thirty MS participants were compared with 30 controls across all six conditions (sustained vs. divided task and auditory, visual, bimodal presentations). The results suggested that the MS group had slower reaction times and were less accurate than controls on both sustained and divided measures of attention. Of note, the MS group were disproportionately slower on the bimodal trials of the divided attention task, relative to unimodal trials. The authors concluded that their results were not related to motor slowing or information processing speed deficits, but linked with the task demands and the modality targeted. When the MS group were performing the divided attention task in visuo-auditory bimodal trials, their performance suffered most and this could be explained within Baddeley's (2003) theoretical framework with a deficient central executive component, impaired in allocating attentional resources efficiently between visual and auditory modalities.

More recently, (Hamilton et al., 2009) examined cognitive – motor dual tasking in 18 relapsing and remititng patients and 18 age and gender matched controls. Participants took part in five conditions: walking alone, a fixed digit span task alone, a individually titrated digit task alone, walking with

a fixed digit task, and walking with a titrated digit task; the latter two conditions representing the cognitive – motor dual tasking conditions. Compared to controls, the authors found that MS participants had greater decrements in dual task performance. These decrements were evident in walking speed, digit task, and swing time in fixed demand dual tasks and decrements in walking speed in titrated demand dual tasks. They concluded that the dual task decrement in the cognitive-motor paradigm could be due to a divided attention deficit or overloading of working memory as a result of walking requiring greater attention.

The implications of this are that people with MS may have everyday difficulty walking and talking and that this problem could be linked to an increased risk of falls. The authors highlight the need for a clinical tool to assess cognitive-motor dual tasking ability in MS.

Conclusion

Cognitive impairments in multiple sclerosis are now a well recognised hallmark of the disease and research investigating the breakdown of attentional processes is of current interest. However, what is less clear is the extent of the causal variables that contribute to this impairment. For example, it is accepted that white matter lesions can reduce speed of processing in the MS brain. These lesions damage the myelin sheathes leaving denuded axons that cannot efficiently execute saltatory conduction meaning neuronal signals have a decreased velocity and increased signal transit time. If the brain processes involved in cognition are assumed to operate within optimal synchronous firing patterns whose orchestration and timely arrival are the bedrock of efficient performance, then it is not surprising that this has a secondary effect on attentional systems. These systems are assumed to be subserved by a complex network of distant brain regions traveling between frontal, subcortical, and parietal cortices. Further, lesions in the cognitive-motor pathways can affect speed and automaticity of simple and complex movements, which are involved in many cognitive tasks. Also, the powerful effects of fatigue on cognition cannot be discounted and many patients testify to experiencing cognitive decline during times of fatigue. Perhaps then the attentional problem found in MS is actually multifactorial with different causal variables with different weightings depending on context rather than simply isolated damage to an attentional controller. It may be then that under experimental conditions, deficits in attention will only be highlighted when cognitive tasks involve a motor component, having to work at speed, or most likely a combination of both. There are no definitive methods of isolating and testing divided attention processes in multiple sclerosis and consensus on optimal procedures is required for future research of divided attention to help elucidate this complex breakdown.

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Atypical presentations in a typical memory clinic: The possibilities for rehabilitation

Rebecca Poz

The role of the neuropsychologist within a memory clinic is often diagnostic, and the National Dementia Strategy (DH, 2009) stresses the importance of early diagnosis. The availability of acetylcholinesterase (AChE) inhibitors has added a new dimension to dementia services over the last decade. From early 2012 the UK patents for galantamine, donepezil and rivastigmine expire and with the re-appraisal of NICE guidance in March 2011 to incorporate the prescribing of memantine for the later stages of Alzheimer's, as well as recommending donepezil, galantamine, and rivastigmine as options for the treatment of Alzheimer's disease in the mild stages; the voice of those advocating for non-pharmacological approaches within memory clinics may need to be louder than normal. Pharmacological treatments have clearly improved the experience for many people with dementia, but memory clinics, and psychologists in particular, continue to play a crucial role in the early treatment of people for whom acetylcholinesterase inhibitors are either not indicated or not beneficial.

This paper presents the case study of a patient referred to our memory clinic with a complex and atypical presentation and the formulation-based interventions that were provided.

SINCE Alois Alzheimer's 1906 lecture of what was to become his eponymous case significant progress has been made in the way we treat people with dementia, and hopefully the patients of today experience a very different pattern of care from what we know of Auguste D's hospital inpatient stay that spanned the five years from her admission in 1901 to her death in 1906 (Maurer, Volk & Gerbaldo, 1997).

The treatment options available now include pharmacological interventions, including cholinergic medication and, albeit controversially, antipsychotic medication and non-pharmacological interventions, including Cognitive Stimulation (Spector et al., 2005), spaced retrieval, dual cognitive processing (Mimura 2007), and vanishing cues (Clare et al., 2003). Probably unsurprisingly recent trials have shown that a combined approach is more successful than either pharmacological or non-pharmacological interventions alone (Manzine & Pavarini, 2009).

To recommend that 'early intervention in Alzheimer's disease (AD) should focus on psychological and social needs as well as the provision of medication' (Clare et al., 2003) is certainly not a new concept, and literature reviews and meta-analyses are now available which focus on cognitive rehabilitation in Alzheimer's disease (e.g. Sitzer, Twamley & Jeste, 2006; Manzine & Pavarini, 2009).

Although the label of Alzheimer's disease was originally used in reference to pre-senile dementia its current use is to describe senile dementia of the Alzheimer type. Auguste D herself first presented with strong feelings of jealousy towards her husband, and subsequently rapidly increasing memory impairments, disorientation, hiding objects, and paranoid ideas that someone wanted to kill her. She endured the illness for four-and-a-half years until her death.

The typical presentation of Alzheimer's disease, as we now use the term, is of memory loss as the first and primary complaint. Neuropsychological evaluation shows deficits in memory and at least one other cognitive domain – a language disorder, visuospatial impairment, atten-

tional deficit or apraxia – and there is an absence of early motor features. The cognitive rehabilitation strategies that have been developed and made it to the pages of peer reviewed journals tend to focus on rehabilitating the amnestic features. But not all patients referred to a memory clinic present in a typical way. Patients may present with language disturbance, change in personality, asymmetrical apraxia, or progressive visuospatial impairments as their primary complaint (Alladi et al., 2007). These diverse clinical presentations have been shown to be underpinned by focal cortical pathology of amyloid deposists (Ng Villemagne, Masters & Rowe, 2007).

The case presented here is of a 58-year-old man who, described in the referral as a 'complex case', was referred to our memory clinic for a neuropsychological assessment to assist in clarifying his diagnosis and to guide rehabilitation.

The clinical case of RN

RN was a 58-year-old man, born into a rural part of the east of England. He achieved normal developmental milestones, was right-handed, literate and attended school to the age of 16 and subsequently an agricultural college. On leaving college he took up the family business of farming. He was married with four children of whom three were still living at home.

Medical history

RN had a complex medical history which included heavy alcohol use. At the time of the assessment, it had reduced to approximately 12 units weekly. In the two-year period prior to the current assessment he had undergone extensive investigations; triggered by the onset of seizures; totalling nine at the time of the assessment. The results are summarised below:

- Diagnosis of moderately severe obstructive sleep apnoea.
- The seizures did not constitute epilepsy, but secondary to an underlying neurodegenerative process.
- Head CT, EEG and blood screens were all reported as normal.
- The MRI scan identified mild to moderate global atrophy greater than would be expected for his age. T2-weighted MRI identified several areas of high signal, though these were interpreted as 'really very trivial'. Our own radiologist's opinion was of no evidence of either atrophy or vascular changes.

RN had a history of depression and had been treated with antidepressants 10 years previously. Within the family; one daughter was experiencing a depressive episode for which she was receiving pharmacological and psychological interventions. RN's mother had died six years previously with Alzheimer's disease.

Current medication

Citalopram 20mgs, Aspirin 75mgs, Galantamine XL 24mgs o.d., Epilim

Presenting complaints

RN himself did not have any complaints. He showed some awareness that his memory had difficulties and that he might have made some errors at work but he was unable to give any examples of his limitations or any errors he had made due to memory problems. He denied any change in appetite, and reported that he still enjoyed activities.

Neuropsychological assessment

This included: National Adult Reading Test (NART), seven sub-tests of Wechsler Adult Intelligence Scale (WAIS-IIIUK), Wechsler Memory Scale (WMS-III UK) Logical Memory I and II and Recall List Learning I and II, Verbal Fluency, Semantic Fluency, Praxis, Trail Making Test, Rey Complex Figure Test, BADS, Graded Naming Test, Beck Depression Inventory (BDI-II).

The results are presented in Appendix 1. In summary the tests identified very severe episodic memory impairment. There was some evidence of downward trending in

global intellect, however, his cognitive domains were predominantly still functioning within normal limits.

Although the neuropsychology test scores appeared to show relatively pure, severe episodic memory impairment, the man you met presented with a much more complex set of difficulties which simply weren't captured by the tests.

Observations

RN was a tall, heavily-built man with acceptable personal hygiene. He presented as hypomanic; his speech was very fast and he launched into a monologue about his school and his father, he presented with inappropriate laughter and giggling. Subsequent to this monologue there was a general paucity of expressive language and the content, when he did speak, was tangential and egocentric. RN oscillated between impulsivity and aspontaneity. His bruxism was apparent throughout our sessions and he was observed to yawn frequently.

These observations were corroborated by his wife's report of his presentation at home. She reported that RN became agitated and paced, that he was unable to concentrate, or string thoughts together. She also reported a personality change over the previous two years with his pre-morbid personality traits, such as his 'schoolboy sense of humour', becoming exaggerated. She reported no inappropriate behaviours.

Mrs N reported that her husband's appetite had increased, that he ate fast and 'would eat all day if left to his own devices.' His mood swings were reportedly steadier since a change in his antidepressant medication, but he continued to shout and would lock himself in a room when frustrated. During Mrs N's discussion of these difficulties RN appeared to have no insight that these descriptions were accurate, nor was he alarmed by her reports.

Mrs N reported difficulties of a dysexecutive nature in RN including difficulties with initiation, confusion, being 'unable to get a chain of thoughts going', having difficulty in changing from one thing to another, and difficulties with 'upgrades and technology'. She reported that RN had a limited repertoire of routines and a lack of inclination to do other tasks. Finally Mrs N was able to provide examples of rather large episodic memory failures in RN; for example, forgetting that he had sold his tractor to another farmer. In the session RN continued to be rather confused about the reality of this.

Formulation

RN owned the farm where the family lived and he continued to derive a great deal of pleasure from farming. He felt pressure to continue farming to support his wife and four children, the youngest being at school. RN's love for farming and identity as a farmer, coupled with his lack of insight into his cognitive difficulties, made him determined to continue doing the jobs he had done throughout his working life; including tractor-driving and crop-spraying.

The risk of an accident occurring whilst crop-spraying was increased due to RN's memory and executive dysfunctions and this created tension and anxiety within the family. The tension and anxiety was often expressed, predominantly by Mrs N, as frustration and anger. A way of processing this anger was to blame RN and his memory difficulties which left him feeling 'nagged at'. This in turn made him more defensive and determined to continue with all his usual jobs. The family system was additionally burdened by historic marital friction which included low level episodes of physical aggression, ongoing mental health problems in at least two of the members living at home, and the usual financial strains of a farming livelihood.

It was hypothesised that RN's anosagnosia would confer less motivation for him to engage in rehabilitation or identify his own goals (Tham et al., 2001). And even if identified, his lack of insight and episodic memory impairment would make it less likely that he would to utilise specific strategies or remember spoken reminders. The

formulation indicated that a systemic approach may be more successful than an intrapersonal approach. Specifically, engaging the whole family to help identify goals which were in RN's 'best interests', reframe their concept of the problem and identify ways in which they could all support change, was hypothesised to externalise the problems rather than maintaining the person-focussed culture of blame.

'The point of rehabilitation… is to enable people with disabilities to function as adequately as possible within their own most appropriate environments' (Wilson, 2005). RN's assessment identified cognitive rigidity which highlighted a greater importance for the rehabilitation to be carried out within his own environment to reduce the need for generalisation.

Mr and Mrs N's overarching aim was to enable RN to retain as much involvement as possible in the running of their farm. In order to achieve this subsidiary goals were identified:

1. Ensure that risks to RN and the public were kept to a minimum.
2. To help RN deal with his frustration.
3. To provide cognitive strategies.
4. Address the family's fears for the future.

Intervention

The intervention was carried out across eight one-hour sessions in the family home.

1. Ensure that risks to RN and the public were kept to a minimum

RN was doing all the crop-spraying on the farm which Mrs N felt was putting RN, the farm, and the public at risk. She wanted this to be contracted out but believed this would be prevented by RN's lack of insight and determination to run the farm himself.

When this issue was initially discussed RN, as expected, was unable to identify the potential hazards, and it was apparent that RN and his wife would be drawn into their usual ineffective pattern of conflict resolution. The issue was re-framed as being part of a 'normal' winding-down process towards retirement. As RN had friends in the farming community who had also contracted out some of their farm tasks as they approached retirement, it fitted with his frames of reference, became less of a marital battle and was thus accepted by RN.

RN was continuing to drive his tractor on the public roads. Given his dementia, and recent seizures, the DVLA and insurers needed to be informed. Initially the family was encouraged to take ownership of this and inform the appropriate parties themselves, but it became apparent that they felt unable to do so. It was hypothesised that Mrs N did not want to be responsible for having her husband's licence revoked. Mrs N gave her permission in front of RN for the DVLA to be contacted. She then felt able to contact their insurers herself.

2. To help RN deal with his frustration

It was formulated that in order to reduce the RN's frustration the frustration would need to be reduced by the whole system. This was approached by encouraging the family to reframe RN's difficulties and personality change as a product of dementia (i.e. as an illness) rather than to RN 'being stubborn'. A copy and explanation of the neuropsychological results gave the family a 'label' which they could use to talk about the problems and an understanding of the probable impacts of RN's cognitive deficits on his day-to-day life.

Discussions were facilitated regarding ways that it was considered acceptable for RN to express and release his frustration within their family culture. These discussions helped the family to see that they were holding unrealistic expectations by wanting RN to not experience frustration.

3. To provide cognitive strategies

Techniques incorporated memory rehabilitation literature (e.g. Wilson, 1987), and included making RN's environment more accessible and user-friendly. The family was supported to redesign the farm office space; providing clearly labelled folders and

drawers, a white board for messages, and clear instructions next to the computer. As RN's verbal memory was more impaired than his visual memory the family was encouraged to provide information in a written or picture form, for example, using his mobile telephone to programme reminder messages.

Two boxes were introduced to help RN choose which farm jobs could be kept and which could be contracted out. He was asked repeatedly over the course of two weeks to 'post' photos of the jobs into either the 'keep' box or the 'contract out' box. This helped identify consistency in his opinion and linked with acting in his best interests.

4. Address the family's fears for the future

The family not only had concerns about RN's cognitive deficits but also his seizures, which remained poorly controlled and undiagnosed. The intervention provided 'space' for Mrs N to air her fears, which she would not do with friends. Mrs N was provided with information sheets from the Alzheimer's Society and signposted to a carer support group for Younger People with Dementia. It was beneficial for Mrs N to observe the small improvements that RN did achieve throughout the rehabilitation, to give her a sense of hope within the context of a progressive illness.

Outcome

Despite RN's initial protest, he allowed Mrs N to contract out the crop-spraying and accepted it as a step towards retirement. RN does forget that this has been done which can trigger arguments, but by modelling the discussions that we had during the sessions, Mrs N is able to defuse the arguments more quickly.

The couple responded well to the DVLA being contacted on their behalf; Mrs N expressed a sense of relief that the responsibility had been taken away from her. The DVLA did revoke RN's licence, which although he needed reminding at times, was a clear 'rule' that was being imposed on him by a third party (not his wife) and which the family attributed to his seizures. RN found this rule easier to adhere to.

The cognitive strategies were successful to a degree, RN was able to turn on his computer alone by referring to his sequencing instructions, and he was better able to find items in the office. However, the categorising system may have been more beneficial to Mrs N who was able to find things that RN had lost. The use of prompts by mobile telephone was less successful as RN would switch off his telephone if he considered that he was being 'nagged'!

Over the weeks the feeling of hopelessness in the family reduced and Mrs N commented that she felt more empowered to make changes. RN continued to slam doors to release his frustration but this was 'allowed' within the family system.

Conclusions

There is a wealth of research which highlights that Alzheimer's disease is significantly less homogenous than previously believed, in terms of the nature of onset, clinical pattern and underlying pathology. Our role as psychologists within a memory clinic should be to foster an assumption that 'one size does not fit all'. We are uniquely positioned to integrate neuropsychological data with psychological theories in order to provide accessible and personally-meaningful rehabilitation and the importance of our formulation skills becomes even more acute when confronted with patients with atypical presentations.

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Appendix 1

Test	Sub-test/Index	Score	Descriptor
NART	Predicted FSIQ	106	Average
	Predicted VIQ	105	Average
	Predicted PIQ	107	Average
WAIS-III[UK]	FSIQ	93	Average
	VIQ	93	Average
	PIQ	92	Average
	Verbal Comprehension	95	Average
	Perceptual Organisation	97	Average
	Working Memory	89	Low Average
	Processing Speed	90	Average
Rey Complex Figure	Copy	33	Average
	Immediate Recall	11	Borderline
	Delayed Recall	10	Borderline
WMS-III[UK]	Logical Memory		
	Immediate	6	Low Average
	Delayed	1	Extremely Low
WMS-III[UK]	List Learning		
	Immediate	8	Average
	Delayed	6	Low Average
Verbal Fluency	Phonemic Fluency (FAS)	46	
	Semantic Fluency	22	High Average
Trails	Part A	43s	Low Average
	Part B	60s	High Average
BADS	Age corrected score	83	Low Average
Graded Naming	Confrontational naming	20	Bright normal
Western Aphasia Battery	Apraxia	57/60	Average
BDI-II		4	Normal

When is progressive not progressive? A consideration of vascular dementia *versus* stroke

Donna Maria Coleston-Shields

The aim of this paper is to highlight distinctions and overlap between stroke and vascular dementia, with particular reference to interaction between services for the two conditions. With a considerable quantity of research emerging to link stroke and vascular dementia in an ever increasing way, provision of such services warrants careful consideration by clinicians. Case studies are presented to highlight the concern that these services may not always be as 'joined-up' as they should.

Distinctions and overlap in vascular dementia and stroke

THERE IS considerable evidence emerging to suggest that vascular dementia and stroke overlap in terms of underlying aetiology, with a substantial body of research suggesting that they are in fact different aspects of the same condition (for example, de Leeuw & van Gijn, 2003; Gamaldo et al., 2006; Moorhouse & Rockwood, 2008; Béjot et al., 2011). To some extent, both vascular dementia and stroke may be considered as clinical conditions that predominantly affect later life, (though statistics suggest that this may be less exclusively the case for stroke, as indicated below). Despite the overlap in underlying aetiology and affected populations, however, there exist a number of gaps between the two, from how they are approached diagnostically, to how patients may be managed in respective services.

Considering further underlying aetiology, independent literature searches for either 'vascular dementia and aetiology' or 'stroke and aetiology' generate, as might be expected, a quantity of information that is almost impossible to manage. Searching instead for 'vascular dementia and stroke and aetiology' narrows the material, and more importantly, highlights the emerging overlap which is considered to exist between the two conditions. Of particular interest, though not from a scientific data source, is information provided by the Alzheimer's Society under the banner of 'vascular dementia' (Alzheimer's Society website, 2011). This information (summarised in Table 1), to which patients and their carers will readily have access, highlights the aetiological overlap between vascular dementia and stroke that is now believed to exist, reporting that the former may be caused by a single stroke (single infarct dementia) or a series of small strokes (multi-infarct dementia). In the case of a single stroke, the suggestion is that the symptoms a person experiences depend on which area of the brain has been affected; so that discreet and localised tissue damage in the language areas or motor cortex may result in clearly defined deficits that perhaps more readily fulfil clinical criteria for stroke, rather than vascular dementia. If brain areas associated with memory are affected instead, or the damage is more diffuse, the clinical picture that emerges is more in line with the clinical presentation of vascular dementia. If the two are so closely related aetiologically, the question is raised as to whether the clinical view that vascular dementia is progressive in nature while stroke consists more of a discreet, single episode is in fact misleading.

Considering next the possible late onset of vascular dementia and stroke, data for the latter is easier to uncover (for example, see

Table 1: The nature of the underlying aetiology.
Information from the Alzheimer's Society website (2011).

- 'There are two main types of vascular dementia: one caused by stroke and one caused by small vessel disease.'
- 'Stroke is the term used to describe permanent brain damage caused by an interruption in the supply of blood to specific parts of the brain. The symptoms that a person experiences…depend on which area of the brain has been damaged…particular areas can cause the symptoms of dementia.'
- 'When vascular dementia is caused by a single stroke, it is sometimes called single-infarct dementia. Vascular dementia can also be caused by a series of small strokes. These can be so tiny that the person might not notice any symptoms, or the symptoms may only be temporary. This is called multi-infarct dementia.'
- 'Small vessel disease-related dementia…also known as sub-cortical vascular dementia or, in a severe form, Binswanger's disease, is caused by damage to tiny blood vessels that lie deep in the brain.'

Table 2; Carroll et al., 2001), perhaps because stroke is seen, to a greater extent, as a single, discreet clinical entity. In contrast, in terms of statistics collation, vascular dementia prevalence may be masked as it is often subsumed under the overall banner of dementia, as for the data presented in Table 3 (Hofman et al., 1991; Alzheimer Europe, 2009). There exist many estimates however, of the proportion of dementia overall which is specifically vascular dementia (for example, Francis, 2010), and so referring again to Table 3, it is a reasonable assumption that the prevalence of vascular dementia increases steadily in the ageing population. As suggested above, while older adults (in the UK at least) account for a considerable proportion of the overall occurrence of stroke, as indicated in Table 2, this is far from exclusively the case, with a large number of men and women experiencing stroke during their working years. It may be for this reason, that is, that vascular dementia would appear to be rather more exclusively a condition of later life while stroke is seen to impact across the life span, that services managing the two conditions have historically emerged as distinct entities.

Indeed, in terms of service organisation, stroke is considered to be a Neurology specialty, one that is not age-specific, and stroke services operate under a medical model, with neurology subsumed under the Royal College of Physicians. Such services are often located within acute hospital trusts, not necessarily community-based, and psychology may not be involved. In contrast, vascular dementia is considered a specialty of Older Adult Psychiatry, and is thus located

Table 2: Stroke as a condition of later life?
Information from National Statistics Health Statistics Quarterly (Carroll et al., 2001): 'proportion of population by age…who consulted with a first ever or recurrent stroke during the year.'

Sex	Age Group	Population
Female	65 and over	42,930 (256,882)
Male	65 and over	29,054 (245,600)

Table 3: Vascular dementia as a condition of later life?
EURODEM prevalence rates of diagnosed and undiagnosed dementia in the UK (Hofman et al., 1991; Alzheimer Europe, 2009).

Age group, years	Male	Female
30–59	0.16%	0.09%
60–64	1.58%	0.47%
65–69	2.17%	1.10%
70–74	4.61%	3.86%
75–79	5.04%	6.67%
80–84	12.12%	13.50%
85–89	18.45%	22.76%
90–94	32.10%	32.25%
95–99	31.58%	36.00%

in mental health services, usually with a mainly community-based remit. For dementia, psychology is more likely to be involved, especially in memory clinic services. Considering one difference that emerges from this distinction, stroke is much more likely to be diagnosed *via* medical assessment using the techniques outlined in Table 4 (Bradley et al., 2004; BMJ Evidence Centre, 2011), while vascular dementia is more likely to be diagnosed on the basis of clinical presentation and observable changes in cognitive abilities, as suggested in the following quote:

> *'As a recently appointed consultant in old age psychiatry (having been trained in the 'old' way about diagnosing vascular dementia, i.e. sudden onset, stepwise deterioration, history of vascular risk factors, etc.), I started noticing a very different presentation of vascular dementia… These cases commonly present with a range of frontal executive function deficits, with functional psychiatric symptoms of anxiety and depression and sometimes with progressive aphasia, and do not necessarily have the classical history of vascular dementia as described in textbooks.'* (Sikdar, 2002)

Given the distinction that exists between dementia and stroke services, with each having its basis in a different medical specialty, there is a risk that there may be limited contact between the two sets of services, with the possibility that patients may not, therefore, receive as full a service as possible. Three clinical cases are presented below, to highlight this risk further.

When vascular dementia does not progress: The case of Mr A

Mr A was 68-years-old when his GP made a referral into the local memory clinic, because the gentleman reported problems with concentration and mild non-specific memory difficulties. At that time, the patient was otherwise reasonably well. To begin with, he was initially seen at home by the specialist nurse from the memory clinic for screening (which consisted of a structured interview with both the patient and his spouse, and a formal assessment that included the MMSE, HADS, and Bayer ADL Scale). The specialist nurse recommended that the patient progress through the memory clinic pathway; so he was subsequently seen by both the psychiatrist and clinical psychologist in the service, for in-depth assessment with a view to diagnosis.

The psychologist's assessment indicated mild cognitive difficulties only, with a possible vascular aetiology. Accordingly to standard procedure in the memory clinic,

Table 4: Diagnostic techniques for stroke.
(From Bradley et al., 2004; BMJ Evidence Centre, 2011)

- CT (computed tomography) scan: to ascertain what kind of stroke and extent of damage; also used to exclude other illnesses with similar symptoms – should be done within seven days of stroke, ideally within 48 hours.
- Magnetic resonance imaging (MRI) scan: more detailed than CT scan.
- Doppler or duplex ultrasound scan: to ascertain whether there is a narrowing of carotid arteries supplying blood to the brain, a major cause of stroke.
- Blood tests: to check for conditions that may contribute to stroke such as diabetes or problems with blood clotting.
- Chest x-ray: for underlying conditions such as heart or chest complaints that may contribute to stroke.
- Electrocardiogram (ECG): for irregular heart rhythms can cause stroke & need to be treated.
- Echocardiogram (or cardiac ultrasound scan): to look for heart problems that may contribute to stroke.

the patient was invited back for follow-up, but he was unable to attend on two occasions as (according to his spouse's report) he was 'unwell'. The nature of his illness was not revealed to staff in the memory clinic at this stage, but Mr A was seen instead by neurology services at the regional hospital (admitted via A&E), where he remained as an inpatient under neurology for one week. During admission, a right-sided weakness and slurred speech were noted, and this gentleman was considered to be at severe risk of aspiration by the speech and language therapists. His clinical condition was investigated via a CT head scan, carotid dopplers, echoCG, and a chest X-ray; mild carotid plaques, heart chamber dilatation and impairment, and heart value regurgitation were revealed. Mr A was discharged home when medically stable, with advice to his GP regarding medication (aspirin and warfarin), and a cardiology follow-up was recommended. On discharge, the patient was noted to be well.

Mr A's case was tracked, by chance, by the clinical psychologist who worked across the two specialties at the time, being responsible for the gentleman's neuropsychological assessment when attending the memory clinic, and also for neuropsychological input while he was an inpatient at the regional hospital. A few months post discharge, Mr A was invited again for a follow-up at the memory clinic, and on this occasion he was able to attend. At that point in time, both Mr A and his wife indicated that he was doing well, specifically that the previously noted cognitive difficulties had eased, and they felt that he had benefitted from the medical treatment he had received. The decision was made by psychiatry within the memory clinic, however, to retain Mr A under that service, and to monitor him on a longer-term basis. Mr A and his wife seemed puzzled by this decision, and while this has may have been the best clinical judgment on the basis of growing knowledge of the association between stroke and vascular dementia, the message to the patient was unclear. There was no discussion to consider whether Mr A had a progressive organic disorder or not, nor were alternatives to remaining under the care of dementia services explored, for example, the issue of whether Mr A should have been offered care under stroke services, with possible rehabilitation input.

By way of contrast to the above case, where progression of the vascular disorder, in cognitive terms at least, should perhaps not

have been the only focus of clinical input, the following two cases highlight risks that may emerge when progression is not considered and is not addressed with the patient.

When stroke progresses (1): The case of Mrs B

Mrs B was 64-years-old when she was admitted to an acute neurology ward in a regional hospital because of a right main cerebral artery infarct and atrial fibrillation. When she was medically stable, she was transferred to a neurorehabilitation bed, prior to which she had received only minimal physiotherapy input, with no psychology or occupational therapy input. On transfer, a left-sided weakness only was reported, but the neurorehabilitation occupational therapist (OT) queried low mood and reduced motivation for the patient, and she requested psychology input.

On interview, the psychologist noted the patient to have good language skills (for both receptive and expressive language) and good social skills. The psychologist completed a cognitive screening (CLQT), on which language was scored as 'within normal limits' but all other domains (attention, memory, executive functions, and visuospatial skills) were scored as 'moderate impairment.' A left visual field deficit was also apparent on cognitive screening, something which had not been previously noted by staff or reported by the patient herself.

The above areas of cognitive difficulty were discussed with the patient, then with her family and with staff, something which was useful as it helped to inform the rehabilitation process. The psychologist provided further input by advising occupational therapy and physiotherapy as 'motivation' continued to be an issue, and the psychologist also supported the patient emotionally. The rehabilitation process was slow, and inconsistencies began to emerge on a day-to-day basis in the patient's self-report, reflecting memory problems which were perhaps previously masked by her good language and social skills.

Seven weeks after transfer to neurorehabilitation, the patient became acutely unwell: vascular problems were suspected, cardiology became involved in the lady's case, and rehabilitation was halted. When Mrs B was medically stable again, occupational therapy and physiotherapy staff noted reductions in her language functioning, memory, and concentration. Some discussions then took place regarding this lady's rehabilitation potential, but the issue of possible further decline was not addressed, either directly with the patient or within the staff group.

When stroke progresses (2): The case of Mr C

Mr C was 66-years-old when he was admitted to an acute neurology ward in the regional hospital, following a road traffic accident. The patient was alone in his car at the time of the accident and no other vehicle was involved; so the possibility that he had a stroke while driving was raised. When medically stable, Mr C was transferred to a neurorehabilitation bed, prior to which time he had received minimal physiotherapy input only, with no psychology or occupational therapy input. On transfer for rehabilitation, a left-sided weakness only was reported for this gentleman, but the neurorehabilitation OT raised concerns regarding issues of safety, feeling that the patient was not sufficiently aware of his own limitations. The OT also noted that the patient was emotionally labile, and she requested psychology input for cognitive assessment in particular.

On interview, the psychologist noted that the patient had good language (both receptively and expressively), but that his humour seemed somewhat inappropriate. The psychologist completed a cognitive screening (CLQT), for which language and memory were scored as 'within normal limits,' but other domains (attention, executive functions, and visuospatial skills) were scored as 'mild impairment.' A left visual field deficit was observed on screening, which had not previously been noted by staff

or reported by Mr C himself. These areas of mild cognitive difficulty were discussed with the patient, then with his family and with staff members, and the psychologist continued to provide input by advising and liaising with the OT in particular, as safety and self-awareness continued to be issues of concern. Rehabilitation progress was slow, and the OT remained concerned about memory problems, feeling that Mr C was deteriorating in this respect, and that he and his family were unaware of the extent of his difficulties.

Two-and-a-half months after transfer to neurorehabilitation, Mr C became acutely unwell; a pulmonary embolism was detected, and rehabilitation was halted. When medically stable again, the psychologist repeated the CLQT with Mr C, with exactly the same results obtained. Both the psychologist and the OT, however, felt that the patient was deteriorating cognitively so the psychologist completed further neuropsychological assessment (WTAR, RBANS, Key Search Test and Zoo Test from the BADS); results suggested deficits consistent with a vascular aetiology, and on the RBANS, a profile suggesting vascular dementia clearly emerged. The patient's difficulties became more apparent on a day-to-day basis, something which was discussed with both Mr C and his family, and his family became more aware of this gentleman's limitations and issues of concern regarding safety. Some discussions regarding rehabilitation potential took place, as such potential was thought to be limited by the multidisciplinary team; however, the issue of possible further decline was not addressed, and Mr C was discharged home with no care package as he had been living with his son previously, and intended to continue doing so. Rehabilitation follow-up was also uncertain because of the location of Mr C's home.

Final considerations

The above cases are presented to highlight gaps in service, or at least lack of overlap, that can occur for vascular dementia and stroke: what other discussions and communication could have taken place for patients, families, and staff? In the absence of such, the outcome for the patient can be poor prognosis, poor information giving, and poor preparation for discharge and/or future care. There exists the risk that patients may find themselves in the 'wrong' service in the longer term, or perhaps without follow-up where this would in fact be appropriate.

If vascular dementia and stroke are linked to the extent that is now believed to be the case, the distinction between respective services that continues to exist does not seem to adequately reflect clinical need. While merging services is unlikely to be practical, or indeed ultimately useful given the fact that single, discreet stroke is more likely to affect across the life span, lack of communication between the two is a cause for concern. Such reduced communication may reflect the historical distinction separating the two medical specialties in which stroke and vascular dementia find themselves; however, clinical psychologists, who are not subject to such medical boundaries, may find themselves in an ideal position to foster essential communication. It is hoped in future that this will be the case.

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Glossary

Bayer ADL Scale:
Bayer Activities of Daily Living Scale

CLQT:
Cognitive Linguistic Quick Test

HADS:
Hospital Anxiety & Depression Scale

MMSE:
Mini-Mental State Examination

RBANS:
Repeatable Battery for the Assessment of Neuropsychological Status

WTAR UK:
Wechsler Test of Adult Reading – UK

Dual tasking and executive functioning in healthy ageing, Alzheimer's and Parkinson's disease

Jennifer A. Foley

Our understanding of the executive system has moved away from its initial conceptualisation as a unitary system towards assuming that there are several different executive functions. However, the precise nature and number of these executive functions remains unclear. This paper discusses various putative frameworks of the executive system and illustrates how the different executive functions are affected by healthy ageing, Alzheimer's and Parkinson's disease.

The executive system

MAKING BREAKFAST in the morning, attending appointments on time and buying the right groceries at the supermarket are all complex activities thought to be supported by the 'executive system'. The 'executive system' is a term used to denote a range of higher-order cognitive processes, which are used to control and direct attention, and allow us to cope with the demands of everyday life (Baddeley, 1996; Evans, 2009). Damage to the executive system can lead to a multitude of impairments, such as increased distractibility, difficulty making or following plans, impaired judgement, and behavioural disinhibition.

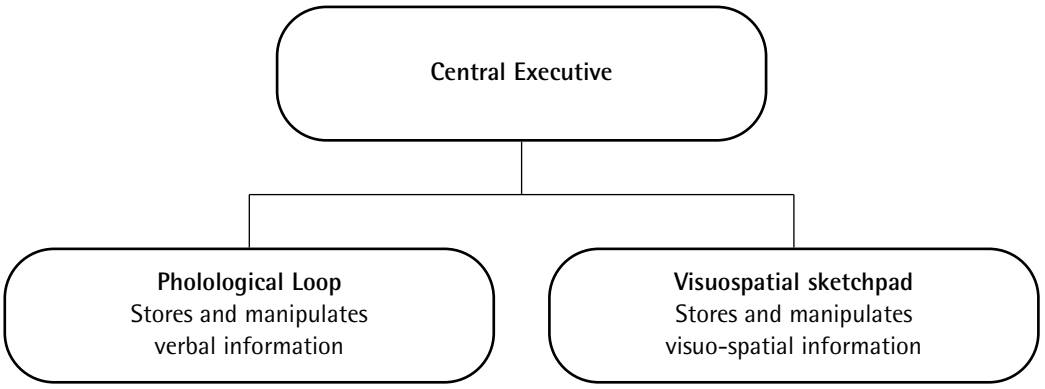
Such deficits were initially thought of as reflecting the functioning of the frontal cortex, and were subsumed under the label of 'frontal lobe disorder'. However, it was later recognised that so-called frontal symptoms could arise from damage to other parts of the brain and that damage to the frontal cortex did not always lead to frontal symptoms (Alvarez & Emory, 2006; Stuss & Benson, 1984). Thus, Baddeley (1986) suggested this disorder be re-labelled as the 'dysexecutive syndrome' to minimise any assumptions about the location of the brain damage.

Models of the executive system

One of the first theoretical accounts of the executive system was that given by Baddeley and Hitch (1974) in their model of working memory (see Figure 1). In this model, they suggest that the short-term storage and processing of information is supported by two domain-specific systems: the phonological store and the visual sketchpad, responsible for verbal and visuo-spatial information respectively, which are co-ordinated and monitored by a domain-general 'central executive'.

This description of the central executive was criticised as being akin to a black box or modern-day homunculus, because of the poor specification of its component processes. In an attempt to define these processes, Baddeley (1986) suggested that the central executive may function similarly to Norman and Shallice's information processing model, the Supervisory Attentional System (SAS; Norman & Shallice, 1980). In this model, perceptual information triggers a database of specific action schemas or scripts, with the correctly-matching schema being selected by a contention scheduling system. When there is no clear matching schema (e.g. in situations that are novel or require planning) or there are several appropriate schemas (e.g. following interruption), the SAS exerts a top-down bias on contention scheduling, so the most

Figure 1: Model of working memory (Baddeley & Hitch, 1974).



appropriate schema is selected. The SAS is theorised as having at least five constituent processes: (1) energising schemata; (2) inhibiting schemata; (3) adjusting contention scheduling; (4) monitoring the level of activity in schemata; and (5) control of if-then logical processes. Importantly, this offers an account of the subprocesses of the SAS, but do these SAS subprocesses translate to specific executive processes?

Indeed, Burgess (1997) discusses a number or reasons for why it may be difficult to identify the component processes of the central executive. He argues that common measures of executive functioning tap a number of different executive and non-executive processes (such as language, processing speed, memory, visuo-spatial functioning), and furthermore, such executive processes may not function independently, but be serially dependent upon one another. Even if measures were able to isolate specific executive processes, using two similar indices of a specific executive process is likely to generate different performance profiles, as when the novelty of the situation is diminished, it should require less executive control.

Furthermore, some believe that the function of the central executive or SAS cannot be fractionated; that the executive system does not comprise constituent subprocesses, but reflects a single process, such as 'fluid' intelligence (Duncan, 2005; Duncan, Burgess & Emslie, 1995; Duncan et al., 1996). Despite this, and the aforementioned methodological issues, converging evidence supports the existence of some separable and independent executive processes (Baddeley, 1996; Burgess et al., 1998; Friedman et al., 2006, 2008; Stuss et al., 1995).

Separable executive processes

Stuss et al. (1995) used lesion, behavioural and neurophysiological evidence in an attempt to separate out different executive processes. They found evidence for seven distinct processes, which are: (1) sustaining attention; (2) concentrating; (3) sharing attention; (4) suppressing; (5) switching; (6) preparing; and (7) setting. Furthermore, they overlapped the SAS subprocesses onto these seven executive processes, and hypothesised that the several different SAS subprocesses were involved in each one of these executive processes.

Similarly, Baddeley (1996) described behavioural evidence for at least four executive processes. These are: (1) dual tasking (which refers to the 'sharing attention' process identified by Stuss et al., 1995); (2) random number generation, or the switching of retrieval strategies; (3) selective

attention (which can be thought of as overlapping with both concentration and/or suppressing attention described by Stuss et al., 1995); and (4) activation of long-term memory or working memory.

Burgess et al. (1998) explored the putative fractionation of the central executive by performing a factor analysis on carer-ratings of people with dysexecutive symptoms. Using this methodology, they found evidence of three main factors: (1) inhibition; (2) intentionality; and (3) executive memory (similar to Baddeley's working memory).

Miyake et al. (2000) explored executive processes by assessing healthy young adults on a number of basic measures of executive functioning, and then, using factor analysis and structural equation modelling, identified the factorial structure of the executive system. They found evidence of three executive functions, namely: (1) inhibition; (2) updating; and (3) shifting. Interestingly, although these executive functions did share some variance, they were also independent of each other, clearly disagreeing with single process theories of executive functioning, such as that of Duncan and colleagues (c.f., Duncan et al., 1995). They explored how such 'basic' executive functions were associated with performance on more 'complex' tests of executive functioning, such as the Wisconsin Card Sorting Test and dual tasks. They found that although the basic executive functions were related to performance on most of the complex executive tests, no basic executive function was associated with performance on the dual task measure, lending further support to both Stuss et al. (1995) and Baddeley et al. (1996), that dual tasking reflects an independent and separable executive function. This hypothesis is further supported by Friedman et al. (2008), who identify updating, task switching and inhibition as executive functions but also note that dual tasking is an additional and important executive function, which has received much less attention from the research community.

Thus, there seems to be accumulating evidence of a putative taxonomy of the executive system, involving (but not necessarily exclusively) the commonly identified executive functions of:

1. Inhibition

Inhibition is identified as a separable executive function by Stuss et al. (1995), Burgess et al. (1998), Miyake et al. (2000) and Friedman et al. (2008). There is also overlap with the concept of 'inhibition' and Baddeley's (1996) 'selective attention', as selectively attending can be seen to require inhibition of attention to irrelevant stimuli. Inhibition is commonly assessed using interference paradigms, such as the Stroop task (Miyake et al., 2000), which require inhibition of pre-potent responses.

There is also a theory that inhibition may underlie performance on other types of executive tasks (e.g. Hasher & Zacks, 1988), but there is sufficient evidence to consider it as a distinct, and separable, executive function (Friedman et al., 2008; Miyake et al., 2000).

2. Switching

Task switching, or set-shifting, is also commonly identified as an executive function by Stuss et al. (1995), Baddeley (1996), Miyake et al. (2000), and Friedman et al. (2008). Shifting is indexed by performance on measures requiring the alternate performance of two similar tasks, such as the plus/minus task (Jersild, 1927, as used by Miyake et al., 2000), and Local Global or Navon figure tasks (Miyake et al., 2000).

3. Updating

Updating (Friedman et al., 2008; Miyake et al, 2000) is another commonly identified executive function, which requires the short-term storage, processing and revision of information. This overlaps with the concept of activation of long-term memory or working memory (Baddeley, 1996) and executive memory (Burgess et al., 1998), as well as sustained attention and concentration

(Stuss et al., 1995). Updating can be assessed using measures of working memory span (Baddeley, 1996), and sustained attention (e.g. tone monitoring, Miyake et al., 2000).

4. Dual tasking

Sharing attention (Stuss et al., 1995) or dual tasking (Baddeley, 1996; Friedman et al., 2008; Miyake et al., 2000) requires the simultaneous performance of two tasks that involve separate cognitive resources. It has been recognised that accurate assessment of dual tasking ability should ensure that the difficulty of the two individual tasks are equated across individual, so that the difference between single and dual task performance can be directly compared (Della Sala et al., 2010). Further, the change in performance observed on each task from single to dual task conditions should be combined to give an overall measure of dual tasking ability, to account for any trade-off that may occur between tasks (Baddeley et al., 1997; Della Sala et al., 2010; Miyake et al., 2000).

Della Sala et al. (2010) presented a new test of dual tasking ability, which complies with these various methodological issues. The test involves performing two tasks: listening to numbers and repeating them back (with level of difficulty calibrated to each participant's own level of ability), and using a pencil to trace a path around a sheet of paper. Performance is assessed on each of these tasks when performed alone and then together. The combined performance score is then used as an index of dual tasking ability.

Executive functioning in healthy ageing

Performance on all of these functions appears to improve with age in the first two decades (Huizinga, Dolan & van der Molen, 2006; Lehto et al., 2003), with updating continuing to improve into young adulthood (Huizinga et al., 2006), but the picture is less clear at the other end of the age spectrum.

Performance on many, but not all, aspects of executive functioning is thought to deteriorate with age (de Frias, Dixon & Strauss, 2006; Fisk & Sharp, 2004; Johnson, Logie & Brockmole, 2010; Lamar & Resnick, 2004; MacPherson, Philips & Della Sala, 2002). Age-related decrements have been found on measures of shifting (Verhaeghen & Cerella, 2002; Verhaeghen et al., 2005), such as the CANTAB ID/ED attention set-shifting paradigm (De Luca et al., 1996), the Wisconsin Card Sorting Test (Parkin & Walter, 1992; Spencer & Raz, 1994) and category switching fluency (Henry & Phillips, 2006).

Similarly, performance on measures of updating, such as digit span and other spans (Fisk & Sharp, 2004; Johansson & Berg, 1989) and prospective memory (Logie & Maylor, 2009), has been found to decline with age. Some studies have found age-related decrements on other aspects of updating, including verbal fluency (Parkin et al., 1995; Veroff, 1980; Whelihan & Lesher, 1985), but others have not (Daigneault, Braun & Whitaker, 1992; Henry & Phillips, 2006; Machado et al., 2009; Parkin & Walter, 1992; Treitz, Heyder & Daum, 2007).

Fewer studies have specifically examined the effect of age on inhibition, but those that do suggest that performance also deteriorates with age (Boone et al., 1990; Stoltzfus, Hasher & Zacks, 1996; Whelihan & Lesher, 1985).

Some studies have found that dual tasking also deteriorates with age (Anderson, Craik & Naveh-Benjamin, 1998; Craik et al., 1996; Craik & McDowd, 1987; Fernandes & Moscovitch, 2000; Hartley & Little, 1999; Lindenberger, Marsiske & Baltes, 2000; McDowd & Craik, 1988; Naveh-Benjamin et al., 2005). However, in these studies, the demands of the two individual tasks were not calibrated to the ability of each participant. As single task performance is not equated across age groups, any group difference in dual tasking ability may have arisen from differences in baseline ability to perform the two tasks. When the demands of the two tasks are calibrated to the ability of each individual, so that single task performance is

equated across groups, dual tasking ability appears unaffected by normal ageing (Baddeley et al., 1986; Belleville, Rouleau & Caza, 1998; Della Sala et al., 2010; Logie et al., 2004; Salthouse et al., 1995).

Executive functioning in Alzheimer's disease

With the onset of dementia, all of these functions eventually deteriorate. Perry and Hodges (1999) stated that executive functioning is the first non-memory domain to be affected by the disease, occurring before deficits in language or visuo-spatial functioning emerge.

There have been considerably fewer studies investigating the relative decay of the different executive functions in Alzheimer's disease (AD), probably because by the time of diagnosis many people with AD will perform at floor on the common measures of executive functioning. However, Perry and Hodges (1999) describe how, in the initial stages, aspects of selective attention (or inhibition) and divided attention (dual tasking) are particularly affected, whilst sustained attention (updating) is relatively preserved. It is difficult to determine how much of the impairment in inhibition is specifically caused by AD, and how much is simply an exacerbation of the age-related decline. However, the deficit in dual tasking is more striking, considering its unique preservation in healthy ageing.

In line with the findings of Perry and Hodges (1999), several studies have found that people with AD demonstrate gross impairment in dual tasking ability (Baddeley et al., 1986, 1991; Della Sala et al., 1995; Holtzer, Burright & Donovick, 2004; Logie et al., 2004; MacPherson, Della Sala & Logie, 2004; MacPherson et al., 2007; Morris, 1986; Morris & Baddeley, 1988; Sebastian, Menor & Elosua, 2006). This dual task impairment has been shown to reflect a specific deficit in the ability to co-ordinate the performance of two tasks, and not simply the effect of overall cognitive demand on a damaged brain (Logie et al., 2004). Importantly, the dual task impairment is specific to AD, and is not present in other disorders that can be misdiagnosed as AD, such as chronic depression (Kaschel et al., 2009).

Mild Cognitive Impairment (MCI) is often considered to be the prodromal state of AD. Currently, MCI diagnosis contains both true positives (individuals who will convert to AD), and false positives (individuals whose deficits will remain stable or even improve), making it difficult to determine individual prognosis. When people with MCI are compared with people with AD and healthy older adults on the dual task, only the people with AD showed dual task impairment (Foley et al., 2011). However, a small number of the people with MCI demonstrated particularly low dual task performance, and other people have found that lower dual task performance is a predictor of increased conversion rates to AD (Robert et al., 2006). Thus, low dual task performance may not only be a specific marker of AD, but also identify those at increased risk of developing AD.

Executive functioning in Parkinson's disease

Parkinson's disease (PD) is another form of pathological ageing, affecting around four million people worldwide (Parkinson's Disease Foundation, 2010). The disease is caused by degeneration of dopamine producing cells, initially restricted to the substantia nigra pars compacta, but later progressing to the ventral striatum and prefrontal cortex. Its characteristic motor symptoms are slowed movement or 'bradykinesia', muscular rigidity, tremor and postural instability. The presence of non-motor symptoms is also increasingly recognised, including cognitive and emotional dysfunction.

Parkinson's disease dementia is thought to occur in around 30 to 40 per cent of people with PD. In those without dementia, studies tend to report mainly a dysexecutive profile. Importantly, these executive impairments are not simply a reflection of slower

processing and motor speed (Gilbert et al., 2005), but reflect a real deficit in the ability to control and direct attention.

These impairments are found on measures of shifting, such as the CANTAB ID/ED (Cools et al., 2009; Owen et al., 1993; Williams-Gray et al., 2008) and other task switching paradigms (Cameron et al., 2010; Kehagia et al., 2009). Impaired performance has also been found on measures of updating, such as digit span (Gabrieli et al., 1996), spatial span (Cools et al., 2002; Zgaljardic et al., 2006), Brown-Peterson paradigm (Marié et al., 2007), and letter and semantic fluency (see Henry, Crawford & Phillips, 2004, for a review). People with PD also demonstrate impairments in inhibition, with poorer performance on the Hayling (Bouquet, Bonnaud & Gil, 2003; Uekermann et al., 2004) and Stroop tests (McKinlay et al., 2009).

There is also some evidence that people with PD display dual task impairment. Dalrymple-Alford et al. (1994) asked eight people with PD and eight age-matched healthy control participants to perform two tasks alone and then together. These two tasks were tracking a moving square around a computer screen using a joystick, and listening to and repeating back sequences of digits. The PD patients demonstrated significantly lower dual task tracking performance than the healthy controls, but no difference in digit recall dual task performance.

However, the PD patients actually performed better (although not statistically) than the healthy controls on the digit recall task, and, therefore, it is possible that if dual task performance on these two measures was combined, this group difference might disappear.

Therefore, we assessed dual tasking in 13 people with mild PD and 50 healthy age-matched controls. Interestingly, the PD patients did not demonstrate impaired overall dual task performance, suggesting the relative preservation of this executive function in the early stages of PD.

Summary

There appears to be growing evidence for at least four independent executive functions: updating, switching, inhibition and dual tasking. All of these, except for dual tasking, appear to be negatively affected by ageing, and further impaired by Alzheimer's and Parkinson's disease. However, dual tasking remains stable in healthy ageing, and is only impaired by Alzheimer's disease. This specificity suggests that dual task measures may be particularly useful in the early and accurate diagnosis of AD.

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Using neuropsychological assessment to predict everyday functioning in Alzheimer's dementia

Linda Monaci & Robin Morris

The use of neuropsychological assessment beyond diagnosis is related partly to the extent to which it can indicate everyday function. This study investigates whether the associations between neuropsychological functioning, activities of daily living (ADL) and instrumental activities of daily living (IADL) change over an 18- to 24-month follow-up, exploring whether these change with dementia progression.

Thirty-four patients with probable Alzheimer's disease were assessed at baseline and again after between 18 and 24 months. Neuropsychological function was assessed using the revised Cambridge Cognitive Examination, which includes within it the Mini Mental State Examination and an executive function scale. ADL and IADL were also measured, together with background neuropsychiatric features by using the Neuropsychiatric Inventory.

Pearson correlations between the measures of daily functioning and cognitive abilities and neuropsychiatric symptoms showed that initially neuropsychological test results tended to correlate with IADL rather than ADL measures. Neuropsychiatric symptoms were not correlated whether IADL or ADL. At follow-up, none of the neuropsychological function measures correlated with IADL or ADL, but neuropsychiatric symptoms were correlated with IADL.

At baseline, neuropsychological function is associated with IADL but not ADL. At follow-up, the association between neuropsychological function and IADL diminishes, and associations between neuropsychiatric disturbances and IADL emerge.

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Notes for Contributors

The *PSIGE Newsletter* welcomes the following submissions for publication: articles, research updates, Letters to the Editor, book reviews. These can be on any aspect of psychological theory or practice with older people.

Articles
Articles form the bulk of contents submitted to the *Newsletter*. As the *Newsletter* aims to cover a broad, cross section of work with older people, we are happy to consider academic, descriptive, discursive, or review articles for publication. These can cover empirical investigations, pilot studies, descriptions of service developments, audits and evaluations. Articles should be submitted three months before publication (i.e. October for the January issue, January for the April issue, April for the July issue, and July for the October issue).

Articles of any length up to a maximum of 3000 words will be considered. Experimental reports should follow convention in terms of subheadings and sections: Abstract, Introduction, Method, Results, Discussion, References.

References should follow conventional format as in journals such as *Psychological Review*:
(1) Book reference:
Mischel, W. (1986). *Introduction to personality.* New York: CBS.
(2) Journal article:
Martin, A. & Fedio, P. (1983). Word production and comprehension in Alzheimer's disease: The breakdown of semantic knowledge. *Brain and Language, 19*, 124–141.
(3) Paper in a book:
Garrett, M. (1980). Levels of processing in sentence production In B. Butterworth (Ed.), *Language Production (Volume 1) Speech and Talk.* London: Academic Press.

Research Updates
The *Newsletter* is particularly keen to publish contributions concerning ongoing research. These can reflect any stage in the research process, for example, ideas for discussion or early stage results, which are not ready for formal publication. Try to keep these submissions below 500 words.

Letters to the Editor
The Editor welcomes correspondence which combines brevity with rational argument. Letters may be edited if more than 250 words in length.

Book reviews
Submissions up to 250 words reviewing a text of relevance and interest to the PSIGE membership will be considered. These submissions must include full details of the book (including publisher).

The Editorial Board reserves the right to make minor changes to any submissions. Where major editing is necessary, the authors will be informed.

Images
The *Newsletter* is published in black-and-white. It is not advisable to send complicated, colour diagrams. If you are unsure, try printing the image or photograph out on a mono laser printer to check for clarity.

Please send original image files (.tif, .jpg, .eps or the like), not simply a Word document with the pictures imported into it, as these do not print properly.

Submission Procedure
All submissions must be written in language that is inherently respectful to older people and consistent with the British Psychological Society's guidelines.

All contributions must be word processed. Formatting should be consistent with the British Psychological Society's guidelines.

Please submit articles as a Word file via e-mail to the Editor.

When submitting articles please send the following information:
Full name;
Affiliation (title, place of work);
Contact details (should you be willing to be contacted by the membership);
Acknowledgements (as appropriate).

Finally, all reports of research should indicate whether or not Ethics Committee approval was awarded, and by which Ethics Committee, or whether the work was carried out as an audit/service evaluation project.

All contributions should be sent to: **louisa.jackman@hotmail.co.uk**

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