General Introduction: What Is the Relevance of Neuropsychology for Clinical Psychology Practice?

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WHY STUDY CLINICAL NEUROPSYCHOLOGY?

At this early stage in the 21st century, clinical neuropsychology is rightly finding its feet as a well-delineated and expanding clinical specialty within Clinical Psychology. It has moved away from the purely diagnostic role it acquired after the Second World War, to one in which the characterization of a person's functional strengths and weaknesses and the explanation of their behaviour have become central in extending the range of meaningful questions that can be posed about an individual patient's presentation. Clinical neuropsychology is now very much valued as not simply involving the assessment of cognitive abilities in patients with cerebral pathology, but also as playing a major role in the rehabilitation of such people. It is also contributing to the understanding of the impact on cognitive functioning of disorders hitherto conceptualized as psychiatric or 'functional' (rather than 'organic')—for example, depression or schizophrenia—and is being used to understand and hence possibly conceptualize in neuropsychological terms a variety of antisocial or maladaptive behaviours. Neuropsychology has expanded its area of enquiry beyond the testing room and into the implications of cognitive impairment for everyday life, with a range of tests that are striving to be more ecologically valid (e.g., Wilson et al., 1996) as well as environmentally based (Shallice & Burgess, 1991; Alderman et al., 2003).

It is therefore important that all clinical psychologists, and not just those working in specialist neuropsychological settings, have a basic grounding in neuropsychology. Perhaps the simplest way of illustrating the widespread application of neuropsychological skills comes from the types of questions that clinical psychologists might need to answer about their patients. Thus a clinical psychologist working in a primary care setting, being the first person to undertake a formal assessment of a patient, might need to determine whether their patient's complaint of poor memory represents a condition that merits referral for further investigation

by a neurologist or is likely to represent the consequences of anxiety or depression. In an adult mental health setting, just as in a neuropsychiatry service, there may be the need to decide whether a newly developed memory disorder is psychogenically determined, perhaps even characteristic of factitious disorder or malingering. A clinical psychologist working with people with learning disabilities might need to be able to assess whether their patient's cognitive profile is indeed characteristic of a particular disorder (e.g., Down's syndrome), whether it represents the likely onset of the dementia that is often found in older adults with Down's syndrome or points to the impact of some additional, acquired neuropathology (e.g., a recent head injury). In a forensic setting the question for the clinical psychologist to address may well take the form of whether the person's offending behaviour could be accounted for by a previous head injury leading to impulsive behaviour characteristic of executive dysfunction. Working with older adults, the clinical psychologist may not only be trying to clarify whether the person's cognitive decline is representative of dementia rather than affective disorder, but may also need to detail the precise nature of any dementia. (e.g., Alzheimer's disease or frontotemporal dementia). In an alcohol abuse service the evaluation of a person's memory and executive dysfunction may have implications for their future treatment or placement. In child psychology settings the need may well be to clarify the impact of developmental as well as acquired neuropathology on educational and social development.

In all of these settings, a good grounding in the principles of neuropsychological assessment and test interpretation (see Chapter 6) will contribute to the delivery of an effective and professional service. This grounding may also, given service constraints, permit the formulation of appropriate interventions designed to ameliorate the cognitive difficulties delineated by means of the assessment, as well as through observations of the patient's everyday behaviour. In all such instances the clinical psychologist should be seeking to act as a scientist–practitioner, using the ever-growing neuropsychological literature on which to base hypotheses for their assessment and gathering information from as wide a range of sources as possible. As Walsh and Darby (1999) indicate, the clinical (neuro)psychologist may be setting out to confirm that certain features of the patient's presentation are consistent with a particular disorder or syndrome, to generate and then test their own hypotheses about the nature of the patient's deficits, or to decide between competing hypotheses about the person's deficits and their causes, often in a medico-legal setting of either a criminal or civil nature.

One of the main reasons that the clinical neuropsychologist's role has moved away from a strictly diagnostic one is the dramatic development in neuroimaging techniques that now offer markedly improved options for identifying structural and functional cerebral abnormalities (see Chapter 3). This has left clinical neuropsychologists free to develop a better understanding of the nature of different disorders and their neuropathological correlates. One example of this development is the careful study of different types of dementia, whereby distinctions have been made between Alzheimer's disease, vascular dementia, and frontotemporal dementias (and their variants), based both on formal neuropsychological test batteries and newly developed behavioural rating scales (e.g., Bathgate et al.,

2001; Grace & Malloy, 2001; Hodges, 2000; Hodges & Patterson, 1996; Hodges et al., 1992, 1999; Kertesz et al., 2000; Snowden et al., 2001; see pp. 12–15 and Chapter 4) as well as between dementias related to other neurodegenerative diseases (e.g., Hodges, 2000; Morris & Worsley, in press). There is now also much better understanding of how to assess psychogenically determined as opposed to organic memory impairment (see, for example, Chapter 7), which has implications both for interventions and medico-legal work, an area where clinical neuropsychologists can assume a very high profile (see Chapter 15).

It is inevitable that clinicians will develop differing approaches to the assessment and documentation of (and also interventions to deal with) their patients' cognitive impairments. This will arise through differing training experiences and both pre- and post-qualification clinical service constraints. In the following section, however, we will outline some of the principles we consider to be essential to the development of personal competence in the delivery of a service that is able to answer neuropsychological questions about patients. We will be focusing in large part on the assessment and interpretation of neuropsychological impairment.

COMMON ISSUES ACROSS DIFFERENT ASSESSMENTS

Irrespective of the specific referral, there are certain types of information that must be collected prior to the assessment in order for clinical psychologists to maximize their opportunity for collecting meaningful data. Here we will expand on, and add to, some of the very helpful suggestions made by Powell and Wilson (1994). Thus information should be collected on:

- *The intended purpose of investigation.* It is important to clarify with the referrer what information is being sought from an assessment, and it may well be necessary to reframe the referrer's question into one that is neuropsychologically meaningful and possible to answer, as neuropsychological assessments are time-intensive and should not be seen as 'trawling' exercises.
- *The patient's demographic variables* (e.g., age, handedness, education/qualifications, current/previous profession, cultural background), in order to set the context for the interpretation of current test performance. Additional information concerning developmental stage reached will be particularly important in the case of children (see Chapter 13).
- *The patient's previous as well as current medical history*, as this may also be relevant to the development of cognitive impairment, and also their history of alcohol and/or substance abuse.
- *The results of previous investigations* (e.g., neurological investigations, EEGs, CT/MRI or functional brain scans, X-rays, biochemical tests—see Chapter 3 for a description of neurological investigations), and previous (as well as current) psychiatric diagnoses, all of which can assist in the formation of hypotheses about the patient's likely deficits, and so guide the assessment and its interpretation.

- *The results of previous neuropsychological assessments*—these can guide the choice of current tests and permit evaluation of change.
- *The history of the person's lesion/disorder* (e.g., site of trauma, age at and time since injury or onset of illness, history of epilepsy [either predating injury or post-traumatic] if relevant, whether or not anoxic episodes were associated with injury, length of post-traumatic amnesia [PTA] and retrograde amnesia, length of loss of consciousness, Glasgow Coma Scale scores and operation reports), since again these will assist in the formulation of hypotheses about the aetiology, nature and severity of the deficits that may be revealed by the examination.
- *Factors that might affect testing* (e.g., drug types and levels [see Chapter 5], the timing of the assessment in relation to drug ingestion, which may have a direct effect on whether or not the person can be assessed [e.g., in the case of drugs used to treat Parkinson's disease, where 'off' periods at the end of the drug's effectiveness may make assessment extremely difficult or impossible], recent epileptic seizure activity [if relevant], mood and motivation [see Chapter 4], motor/ speech/visual problems [which may determine which tests are feasible to administer] and the patient's likely distractibility).
- *Informants' views of the person, their deficits and if/how they have changed*—many patients with acquired brain injury will have little insight into the reason for their referral for assessment/treatment, and the nature and/or extent of their own cognitive deficits. Thus informants may provide important information about the areas to be explored in the neuropsychological assessment (see Chapter 9).
- *The context in which the assessment takes place* (i.e., whether there are relevant compensation or other medico-legal factors that might affect the person's motivation during the assessment).

While not all of the information will be available in every case, it is important to gather as much information as possible prior to seeing the patient since, as indicated with respect to medico-legal work in Chapter 15, this also permits clarification with the patient of inconsistencies in the history and allows what may be a limited time in which to undertake an assessment to be used to cover the most important areas of that person's cognitive function.

The selection of the tests to be administered then needs to be based on:

- predictions of the likely range of deficits to be found, given what is known about the person's history, neurological investigations, presenting complaints and the neuropsychological profile of that particular disorder and other relevant disorders that may form part of a 'differential diagnosis';
- the time available in which to undertake the assessment (e.g., it may be practical to assess an inpatient on more than one occasion, but only one session may be possible, albeit less than desirable, for someone living at a great distance from the clinical setting) and the patient's likely tolerance of testing;
- the suitability of the test in terms of its standardization when compared with the patient (i.e., whether or not the patient is similar to the standardization sample in terms of IQ, age, etc.);
- the potential adaptability of the test to overcome problems posed by the patient's

motor/speech/sensory deficits and how this might affect interpretation of the results that are obtained;

- the need for an interpreter where the patient's first language is not the same as that of the psychologist or that in which the test is published/standardized;
- the tests that have previously been administered (i.e., one may need to use parallel forms of tests if they are available and consider the possibility that practice effects may be present on other measures, serving to mask deterioration);
- whether the patient is part of a research cohort (e.g., evaluating a neurosurgical intervention for epilepsy, deciding upon the suitability of the patient for pharmacological treatment of dementia—see Chapter 19), in which case a fixed protocol may be required for the assessment;
- whether it will be particularly important to use tests that are statistically inter-related (e.g., the Wechsler Test of Adult Reading, the Wechsler Adult Intelligence Scale—3rd edition [WAIS-III] and the Wechsler Memory Scale—3rd edition [WMS-III], see Chapters 6, 7 and 14) or whether this would pose too taxing an assessment load for the patient to yield interpretable data, in which case other tests might be more suitable;
- what is then found during the assessment (i.e., one may wish to follow up on specific findings with further standardized tests or the development of more idiosyncratic measures using a single case design).

It will not be uncommon for a clinician to develop greater familiarity with some tests than with others (see also Chapter 9), but clinicians should remain open to the need to be flexible in their choice of tests when this enables them better to answer the clinical question being posed in an individual case. It is also important to remain up to date with the development of new neuropsychological tests and to be aware of the psychometric implications of changing between older and newer versions of similar tests for the interpretation of between-assessment results. An important example of this is the difference in IQ scores yielded by different versions of the Wechsler Adult Intelligence Scale.

There is also a clear balance to be drawn between undertaking an adequate assessment and over-assessing a patient. It is a frequent mistake for inexperienced clinical neuropsychologists to suppose that the more tests given the better. It is also not uncommon to see reports where patients have been subjected to hours and hours of testing. This is rarely necessary. If after several hours of testing one is still unsure of what to conclude, it will normally be more informative to gather other types of data such as direct behavioural observation or to interview staff or relatives of the patient rather than to reach for yet another standardized test. However, considerable importance also needs to be attached to the overall scope of the assessment in being able to rule out the presence of cognitive impairment. Thus one should always be aware that deficits, for which the patient has not been assessed, cannot be ruled out definitively. Teuber's widely cited view (see, for example, Walsh & Darby, 1999) that 'absence of evidence is not evidence of absence (of impairment)' continues to be an important reminder that generalizations cannot be made from limited assessments. Clinicians should always specify

the factors limiting their interpretations, making it clear to the reader exactly what tests were undertaken as part of their assessment of the patient.

We will now address a number of other important issues, which also arise when interpreting and reporting the results of a neuropsychological assessment:

- One should not over-interpret minor discrepancies between test scores. It is common to see in reports that a patient who scores at the 10th percentile on one measure is then felt to be significantly relatively impaired on other measures, on which they score at the 5th or 2nd percentile. The difference in reliability of different tests means that such small differences may not necessarily be interpretable in terms of trying to identify specific deficits (see also Chapter 9). In addition, likely premorbid levels of functioning need to be taken into account when trying to decide whether a currently average level score represents intact performance or evidence of change following acquired neuropathology (see Chapter 6 for a further discussion of important psychometric concepts to consider in test interpretation).
- It is important not to rely on test scores alone when deciding whether an impairment is present. Any clinical neuropsychological report should make some reference to the behaviour of the patient during testing and the manner in which they go about solving the tasks and should give at least some brief details of difficulties the patient is reporting in everyday life. Shallice and Burgess (1991) described three patients with strategy application disorder who performed normally on traditional neuropsychological tests including many measures of executive functions, but nevertheless had profound difficulties in everyday life (see Chapter 9 for further details). These cases illustrated the potential danger in over-reliance on formal test performance in deciding whether the patients did or did not have neuropsychological deficits.
- Similarly, a diagnosis should never be made purely on the basis of neuropsychological test results. There are many different reasons that patients may fail tests, so it is never sufficient to rely purely on test performance. If a patient shows a pattern of performance that would be consistent with a particular disorder, then the most that can be concluded is that their performance is *consistent with* that disorder, not that they *have* the disorder (see also Chapter 9). Similarly, neuropsychological tests results should never be used by a clinical (neuro)psychologist to make a diagnosis for which there is no *a priori* medical basis, as they will be acting outside their area of expertise and place themselves at risk of disciplinary and other action.
- One should not be afraid to conclude that test performance cannot determine what the causal factors are in a patient's current problems. For example, in the case of a patient with a history of psychosis and current cognitive problems or learning difficulties who then has a moderate head injury, it may not be possible to tease out to what extent current problems existed premorbidly or were recently acquired, apart from by relying on the reports of relatives, friends or staff who knew the patient before the head injury. Similarly, medication effects (see Chapter 5) may exaggerate or obscure certain deficits. There is nothing distinctive about

neuropsychological tests that means they are *only* failed by patients with some kind of acquired brain injury.

- It is also important to acknowledge that more than one assessment may be necessary in order to arrive at an accurate interpretation of a patient's difficulties; this is often the case when attempting to distinguish, for example, between a developing dementia and depression. Here a further assessment, once an affective disorder has been treated effectively, may permit clarification of whether the person is demonstrating a progressive, neurodegenerative condition (see Chapters 4 and 14 for further discussion).

- It is not uncommon to see reports that conclude that, because a patient passes tests of malingering, they cannot be faking a bad performance; this is incorrect. If the tests of malingering were developed by asking normal controls to fake a bad performance, then it does not necessarily follow that a patient with a mild injury who is trying to accentuate a deficit on formal testing will perform in the same way.

- A consistently perfect correspondence between CT/MRI scan results and performance on formal neuropsychological tests does not exist (which is not surprising as they are measuring very different things—see Chapter 9—and different types of scan may be more sensitive to particular types of neuropathology than others—see Chapter 3). It is therefore possible to find patients who have normal structural brain scans with significant cognitive deficits, or the converse pattern of a patient with an abnormality on brain scanning but intact performance on formal cognitive tests. Evidence from a brain scan should not be used to confirm or disconfirm the validity of observed cognitive deficits, but rather to offer possible hypotheses as to why the observed deficits may be occurring; test sensitivity and premorbid levels of ability may be factors that need to be considered in interpreting the correspondence or otherwise between different forms of assessment.

- Although many neuropsychological tests are now supposedly ecologically valid, very few provide any formal evidence to support this claim (with the notable exception of some of the Thames Valley Test Company tests such as the Behavioural Assessment of Dysexecutive Syndrome [BADS; Wilson et al., 1996] and the Rivermead Behavioural Memory Test [Wilson et al., 1985]). Great care should therefore be taken before drawing conclusions about how a patient will function in everyday life based on neuropsychological test performance alone. For example, it may be reasonable to expect that perceptual or executive deficits may impact on someone's driving abilities, but a decision as to whether someone is competent to drive should never be taken purely on neuropsychological test performance.

- Remember that a clinical (neuro)psychologist is an independent professional in their own right, with a responsibility to the patients they assess and treat. One should not be afraid to question the appropriateness of referrals. It is always best to take responsibility for providing feedback of one's test results oneself since other professions are far less likely to have the in-depth understanding of neuropsychological tests, although it should be *medical* practitioners who deliver *medical* diagnoses if multi-disciplinary feedback sessions are not possible. Issues relevant to the provision of feedback to people with neuropsychological impairments are discussed by Gass and Brown (1992).

• Interpretation of neuropsychological assessments will be enhanced in certain cases by a good working knowledge of psychiatric disorders, their presentation and diagnosis.

It would be difficult to illustrate the relevance of all the above suggestions for clinical practice in such a short chapter. What follows is a selection of case examples highlighting a number of the points we have made above and demonstrating the diverse issues that assessments may raise.

CASE EXAMPLES

Social Problems or the Consequences of a Previous Head Injury? The Importance of a Good History

Ms Y, a 32-year-old, right-handed single mother, was referred for a neuropsychological assessment as a preliminary part of care proceedings being undertaken by the local authority in connection with her three children whose behaviour she was having difficulty controlling. Despite considerable social services input she was unable to manage her household affairs. Another clinical psychologist had wondered whether a neuropsychological assessment might be warranted by Ms Y's apparently disorganized behaviour and had heard that Ms Y had sustained a head injury many years previously. Little other information was available about her history, so Ms Y gave consent for her GP records to be obtained. It was these records that provided some of the information indicated in Chapter 3 as being very important in understanding her presentation.

Although Ms Y's GP queried the value of the release of her medical records since 'all her problems were social ones', her records indicated that, at the age of 17 years, she had sustained a significant head injury as a pedestrian and was unconscious for 10 days. Only after a further 10 days or so could she begin to co-operate with instructions and speak short sentences. Her physical progress was good, but she retained some facial asymmetry and slurred speech. At the time an IQ assessment (test unspecified) yielded an IQ of 110, but she was reported to show a marked emotional deficit, lack of drive and initiative, an increased tendency toward immature behaviour and dependence and two years later was still felt to have very little insight into her deficits. She was unable to continue to train as a secretary as she was slow and forgetful and showed insufficient initiative. Her social life was severely curtailed following the accident. There were, therefore, sufficient behavioural descriptions from her medical records to suggest residual, significant deficits resulting from what was an apparently severe head injury.

Although no scanning information was available to demonstrate any long-term neuropathological sequelae of her apparently severe head injury, the requested neuropsychological assessment was undertaken with a view to determining whether deficits could be elicited that would be consistent with a dysexecutive syndrome (see Chapter 9). Her behaviour during testing was rather distractible,

and she was unable to pick up on social cues to indicate that she should cease chatting and continue with the tests.

On the WAIS-R, Ms Y obtained a Verbal IQ (VIQ) of 101 and a Performance IQ (PIQ) of 96. Her premorbid estimated WAIS-R IQ on the basis of her reading ability on the National Adult Reading Test was 115 for Verbal IQ and 116 for Performance IQ. There was therefore some slight suggestion of an overall reduction in her general level of intellectual ability.

Given the early descriptions of Ms Y's memory difficulties, and the common association between acquired memory impairment and head injury, a number of memory tests were administered. Ms Y's ability to recall a short story from the Adult Memory and Information Processing Battery (AMIPB) was above average (between the 75th and 90th percentile) for both immediate and delayed recall. However, learning of a list of 15 words revealed no consistent strategy for encoding the words, and overall learning was only at the 10th percentile. Immediate and delayed recall of a complex geometric figure were at the upper end of the average range, but learning of an abstract design fell between the 2nd and 10th percentile and was characterized by frequent errors and perseverations of previously incorrect lines.

In terms of her performance on measures of executive functioning, despite performance on the Controlled Oral Word Association Test (COWAT) consistent with that predicted on the basis of her reading ability (Crawford et al., 1992) and completion of Parts A and B of the Trail Making Test that fell in the 50th to 75th percentile range, completion of the Stroop Test was only at the 4th percentile and performance on a test of cognitive estimates was impaired relative to controls. She also failed several subtests from the BADS, showing poor planning, sequencing and rule-following ability.

Thus despite Ms Y's IQ remaining in the average range, her head injury was likely to have produced lasting impairment, particularly in the domain of executive functioning, whereby she had difficulty in organizing information to be remembered, in undertaking cognitive estimation tasks where checking the plausibility of a response is required when the person has to use everyday information to answer a question (Shallice, 1988), in inhibiting unwanted responses and in planning tasks that require a strategy for their effective completion. She had never received any rehabilitation after her head injury (indeed appropriate cognitive rehabilitation of the sort described in Chapter 9 would not have been available to her at that time), and it was likely that, in an unstructured and less predictable everyday situation, her deficits would have had more marked impact on her everyday functioning, making it more difficult for her to care effectively for her children. Understanding the nature of her head injury and its immediate sequelae had been particularly helpful in this case in terms of trying to identify the likely origin of her everyday difficulties, although, in the absence of neuroimaging data, it was not possible to say with certainty that she had sustained damage specifically to the frontal lobes (see Chapter 9).

Distinguishing between Dementias: Matching Test Results to Disorder Profiles

Mrs A was a 55-year-old, right-handed woman who had obtained a first-class degree in Classics and then a further degree in History at Oxford University. She had gone on to work as a journalist for two major broadsheet newspapers and had subsequently worked in radio broadcasting until the age of 51, when she retired due to stress-related difficulties that took the form of panic attacks. Her history did not permit evaluation of whether these might have been accounted for by incipient cognitive difficulties. Mrs A's husband had retired about two years earlier with physical health difficulties. There was no family history of dementia.

Mrs A was referred for assessment via her local community mental health team who were treating her for depression, as they were concerned that she was developing a semantic dementia. Her husband was reporting that he felt her intellect had become 'rather ordinary' in comparison to the superior level it had been. She had become more socially withdrawn, was apparently less good at conversation than previously and was complaining of word-finding difficulties.

Prior to her referral for a neuropsychological assessment, an MRI scan had shown 'global cerebral atrophy, with more volume loss in the left than the right cerebral hemisphere'. There was no history of procedural, episodic or topographic memory impairment although some episodes of confusion had been reported when playing card games; Mrs A was reported as not knowing which way round the table they were playing or who was to play next.

A particularly significant neuropsychological development in recent years has been the delineation of the cognitive profiles in different types of dementia. This has permitted discriminations between frontotemporal dementias and more posterior dementing processes (notably Alzheimer's disease), and within the frontotemporal dementias there are detailed studies of individuals with semantic dementia and progressive non-fluent aphasia. The main features of these dementia types are described in the literature and summarized briefly in Table 1.1.

Based on the descriptions of these different dementia types, and adopting a hypothesis-testing approach, Mrs A underwent a comprehensive neuropsychological assessment, which aimed to examine the characteristics of semantic dementia and determine the extent to which she was impaired on tests of the relevant functions. Many of the papers that have described these disorders have used experimentally derived test materials (e.g., Hodges & Patterson, 1996; Hodges et al., 1992, 1999), whereas here clinically available tests were used.

During conversation, word-finding difficulties and pauses were apparent, with some semantic circumlocutions. Nonetheless, Mrs A's speech was easily produced and well articulated with appropriate prosody. A number of other findings from the assessment were consistent with a possible diagnosis of semantic dementia. These included worse verbal than visuospatial memory (WMS-R Verbal Index = 59, Visual Memory Index = 110) and impaired naming (13/30 on the Graded Naming Test and 9/15 on Naming from Description; see Chapter 8 for further description of naming tests). In addition, Mrs A was impaired at category fluency to a greater extent than letter-based fluency. She produced disjointed prose when

Table 1.1. Key features of different types of dementia of relevance to Mrs A's diagnosis

	Frontal variant of fronto-temporal dementia	Temporal variant of fronto-temporal dementia (semantic dementia; progressive fluent aphasia)	Progressive non-fluent aphasia	Alzheimer's disease (see also Chapter 14)
Memory	Typically unaffected early in the disease. Early mild impairments likely to be in episodic memory with normal semantic memory	Episodic memory (recall of recent events, recent autobiographical information) relatively well preserved	Relatively well-preserved autobiographical (and day-to-day) episodic memory. Short-term auditory–verbal memory severely disrupted	Impaired or poor anterograde episodic memory often the first feature, semantic memory may be relatively unimpaired early on, but patients ultimately show impairment on tests of semantic memory. Memory for both verbal and visual material impaired in most cases
Language	Language dysfunction is an early, subtle sign. Decreased verbal output, with 'factually empty' speech' occurs. Worse letter fluency than category fluency. Mild anomia, but repetition is normal. Comprehension may eventually become impaired. Repetitive speech (spontaneous echolalia). Articulation, phonology and syntax usually preserved	Early word-finding difficulties lead to anomia. Initially, there are mild word-finding difficulties and circumlocutory discourse. As disease progresses, comprehension problems develop and speech becomes stereotyped. Syntax and phonology preserved. Reading irregular words impaired ('surface dyslexia'). Category fluency more impaired than letter fluency	Severe early impairment, with phonological errors, agrammatical sentence structure and disturbed articulation. Phonemic approximations occur when naming. Initially, normal single word comprehension, but there is markedly impaired syntactic comprehension. Letter fluency is more impaired than category fluency	Relatively well-preserved phonology and syntax, but syntax becomes simplified. Conversation initially circumlocutory and later becomes vague and meaningless. Irregular word reading is relatively well preserved until very advanced disease. Category fluency very sensitive to early Alzheimer's disease, while letter fluency is better preserved. Naming to verbal description may yield deficits. Ultimately mutism may occur
Non-verbal skills	Even late in the disease, visuospatial and visual perceptual functions remain normal	Well-preserved constructional, perceptual and visuospatial abilities. Non-verbal semantic knowledge impaired	Non-verbal semantic knowledge well preserved	Visuospatial and visual constructional deficits found

Source: Hodges (2000), Hodges & Patterson (1996), Hodges et al. (1992, 1996, 1999)

asked to describe the Cookie Theft Picture and her auditory comprehension, also assessed using the Boston Diagnostic Aphasia Battery, was impaired.

However, a number of results were not consistent with a diagnosis of semantic dementia. Thus her delayed visuospatial recall (20th percentile) was worse than immediate recall (99th percentile). With respect to IQ measures, there was no difference between PIQ and VIQ scores (PIQ = 95, VIQ = 96, whereas VIQ is thought to show more impairment than PIQ in semantic dementia); in addition, Information and Vocabulary were the best preserved subtests (age scaled scores of 12), while Digit Span and Arithmetic were the least well-preserved verbal subtests (age scaled subtests 6 and 7, respectively), which again is not an expected pattern for semantic dementia. Her PIQ age-scaled subtest scores ranged between 4 and 9. In addition, Mrs A was not impaired at reading the National Adult Reading Test words, making only three errors. Mrs A also passed a measure of non-verbal semantic knowledge, the pictures version of the Pyramids and Palm Trees test (score 51/52) and was able to match written and spoken words and pictures from the Psycholinguistic Assessment of Language Processing in Aphasia test (PALPA) (spoken 40/40; written 40/40). Mrs A was impaired on the Modified Token Test (scoring 11/15) and on the Test for Reception of Grammar (TROG) (passed only 16 blocks), and was poor at reading sentences and paragraphs; she made mistakes on more complex items requiring understanding of more complicated syntax. There was also evidence of executive dysfunction (Trails B was completed in a time that fell below the 10th percentile) and she was classified as 'Impaired' on the Brixton Test, yet was still at the 'Moderate Average' level on the Hayling Sentence Completion Test.

Clearly, the results of this assessment were not clear-cut and demonstrated features that might have been consistent with both semantic dementia and a more typical Alzheimer's disease, and it was therefore felt that at this stage no firm conclusion could be reached. A year later, Mrs A had demonstrated further decline in VIQ but not PIQ, but she had shown further decline in visuospatial memory. Speed of language processing had deteriorated, as had her verbal fluency (letter and category) and naming to description. Single word comprehension remained intact as did her ability to match words to pictures and her access to non-verbal semantic knowledge. Comprehension of verbal material was limited to simpler statements, and oral and written communication was lacking in structure with missing nouns, agrammatic errors and a paucity of content. Thus her profile continued not to fit entirely with any one form of dementia, and, while her anomia, category fluency deficits and diminishing vocabulary and verbal comprehension remained consistent with a semantic dementia diagnosis, it was felt that an atypical form of non-fluent progressive aphasia might have been more descriptive of her difficulties, given her increasing impairment of syntactic comprehension, agrammatism and anomia.

One approach that was not taken in this woman's assessment, but which has received increasing attention, is the use of behavioural profiles in distinguishing between dementia types (e.g., Bathgate et al., 2001; Snowden et al., 2001). These informant-interview-based measures permit discrimination between frontotemporal dementia, Alzheimer's disease and to some extent vascular dementia (Bathgate et

al., 2001) and between frontotemporal and semantic dementia (Snowden et al., 2001).

Post-concussional Syndrome or Post-traumatic Stress Disorder after a Mild Head Injury? The Importance of Knowing about Psychiatric as Well as Neurological Diagnoses

Mr P was a 25-year-old, right-handed man who was a passenger involved in a road traffic accident six months earlier. He had sustained a mild head injury with a short loss of consciousness (10–15 minutes) and orthopaedic injuries, which required an eight-week stay in hospital. The driver of the car, who was a close friend of Mr P, was killed in the accident.

Prior to the accident, Mr P was working as an IT consultant. He had been off work for four months after the accident and had recently returned to work, but found he was encountering problems. He had been to see his GP because he was aware of difficulties with his memory and concentration, as well as increased irritability. He was not sleeping, was experiencing headaches and he reported feeling anxious. His GP had referred him for a neurological opinion and a CT scan, which was reported to be normal. The neurologist referred him for a neuropsychological opinion.

In view of the severity of the accident and the death of his close friend, the differential diagnosis was between post-traumatic stress disorder (PTSD) and post-concussional syndrome (PCS). The cardinal features of PTSD (e.g., DSM-IV, American Psychiatric Association, 1994) are intrusive thoughts or nightmares about the event and avoidance of situations related to it, combined with symptoms of heightened arousal. Some authors such as Sbordone and Liter (1995) have claimed that PTSD does not occur following mild traumatic brain injuries since there is usually amnesia for the precipitating event. However, more recent studies have suggested that PTSD can occur even with severe head injuries where there is amnesia for the event (McMillan, 1996; McNeil & Greenwood, 1996) (see also Chapter 3, this volume, p. 83).

PCS is a term used to refer to range of symptoms that may arise after mild traumatic brain injury (where loss of consciousness is less than 20 minutes and PTA less than 1 hour). These include headache, insomnia, sensitivity to noise, poor memory and concentration, irritability and anxiety and depression. For most cases these symptoms will resolve within a few weeks of the injury, but a minority of patients may still show symptoms several months later (e.g., Gronwall & Wrightson, 1980).

On interview, Mr P could not remember the accident itself, but he could remember arriving at his local Accident and Emergency department by ambulance and had reasonable memory for events after this, suggesting a PTA of less than one hour. He reported feeling very distressed about the death of his close friend. He was experiencing some anxiety when travelling as a passenger in a car, and he was more reluctant to travel by car than he had been before the accident. However, he did not report any re-experiencing phenomena such as nightmares,

'flashbacks' or intrusive thoughts about the accident. This lack of any re-experiencing symptoms would preclude a diagnosis of PTSD.

On formal neuropsychological assessment, Mr P performed a little below his estimated premorbid level of functioning on a shortened version of the WAIS-R (VIQ 99, PIQ 83, NART [National Adult Reading Test] IQ equivalent 112). He was found to have particular difficulty with strictly timed tasks, and he performed very poorly in terms of his backwards digit span.

Mr P was found to have mild memory problems on formal testing. He performed poorly on immediate recall of the story and figure from the AMIPB (AMIPB story recall <10th percentile, figure recall <2nd percentile), although he did not show any further loss of information after a 30-minute delay. He also had difficulties with the Doors and Names recognition subtests from the Doors and People test (Doors: between the 5th and 10th percentile; Names: at the 5th percentile).

His visual perceptual and visuospatial skills were satisfactory (Visual Object and Space Perception Battery [VOSP]) Object Decision 18/20, Position Discrimination 19/20), and he did not show any language difficulties in spontaneous speech or on naming to confrontation (Graded Naming Test, 23/30).

His performance on tests of executive functioning was poor. He obtained a poor score on the Brixton Test (error score = 26, scaled score = 2), and he had marked difficulties with the Hayling Sentence Completion Test (overall scaled score = 1). His verbal fluency was a little lower than that predicted by his reading ability (Crawford et al., 1992). He also had problems with tests of speed and concentration and was slow and inaccurate on the AMIPB Information Processing subtest.

Mr P did not rate himself as depressed on the Hospital Anxiety and Depression Scale (HADS), but he did rate himself as mildly anxious (HADS depression score = 5, anxiety score = 10).

Thus Mr P was exhibiting significant cognitive difficulties on formal testing with memory, concentration and executive impairments that confirmed the everyday problems he had reported. This degree of cognitive impairment would not be expected to be observed simply as a result of PTSD. He was exhibiting anxiety symptoms, and he was avoiding some situations related to the accident itself. However, as already indicated, the lack of re-experiencing phenomena such as nightmares, 'flashbacks' or intrusive thoughts would preclude a diagnosis of PTSD. He was therefore felt to be suffering from the residual effects of his mild brain injury combined with additional emotional problems arising from the death of his close friend. Mr P was given education about head injury and strategies for coping with his memory and attentional problems. He was also given advice about attempting a more gradual return to work and offered counselling sessions to address emotional issues. Jones (1974) found that only 1% of patients with mild head injuries showed persistent symptoms at one year. It was therefore recommended that he be reassessed after a further six months to ensure that his symptoms had disappeared.

CLINICAL NEUROPSYCHOLOGY AS A PROFESSIONAL SPECIALTY: WHO IS A CLINICAL NEUROPSYCHOLOGIST?

The discussion so far has dealt with issues relevant to the day-to-day practice of clinical neuropsychology, with respect to how one should go about one's work. However, given the growing specialization of clinical neuropsychology, the issue of professional competence and titles becomes increasingly important.

In the USA there has existed for some time a clear definition of who is a clinical neuropsychologist (American Psychological Association [APA], Division of Clinical Neuropsychology, 1989). This emphasizes the doctoral level of didactic and experiential training that will have been undertaken in both neuropsychology and neuroscience at an accredited university, the acquisition of at least two years of appropriate, supervised training where the person is delivering clinical neuropsychological services, peer review of their competencies and the compliance with local requirements for licensing and certification in the state in which the person practises. The APA's Division of Clinical Neuropsychology indicates the value placed upon the acquisition of the American Board of Clinical Neuropsychology (ABCN)/American Board of Professional Neuropsychology (ABPN) Diploma in Clinical Neuropsychology as providing the clearest evidence that their criteria have been met.

A number of European countries have developed graduate and postgraduate training programmes (see, for example, Kaschel et al., 1994). In the UK, moves to professionalize clinical neuropsychology as a specialty have only relatively recently become this sophisticated. These have taken the form of the development of formal, post-qualification training for clinical psychologists who are keen, or who need, to be seen as experts within this field. The British Psychological Society's (BPS) Division of Neuropsychology has now developed a Practitioner Full Membership qualification for those who wish to be seen to have achieved a recognized level of competence in the field. A substantial number of people will have acquired this prior to the end of 2003 through 'grandparenting' clauses, which recognizes that they will have:

- been a Fellow, Associate Fellow or Graduate Member of the BPS;
- been eligible to be a Chartered Clinical Psychologist or be a Full Member of one of the BPS's other divisions (but not the Division of Teachers and Researchers in Psychology) with a background relevant to clinical neuropsychology; and have
- been engaged in clinical neuropsychological practice for a period of two years full-time or its equivalent part-time.

People trained overseas may, with a statement of equivalence, have satisfied these criteria if eligible to be a member of one of the BPS's divisions (see BPS Membership and Qualifications Board, 2002a). By the end of 2003, those who have not already met these criteria will have to follow a post-qualification training route leading to an advanced professional qualification in the field of clinical

neuropsychology. There will be two forms of this Membership qualification: (i) Adult Clinical Neuropsychology and (ii) Paediatric Clinical Neuropsychology. The full regulations and syllabus for these new Practitioner Full Membership Qualifications have been outlined (BPS Membership and Qualifications Board, 2002b), but essentially those aiming to possess these qualifications will have to:

- have acquired Graduate Basis for registration in the BPS and be registered as a Chartered Clinical Psychologist (in the case of the Adult qualification), or have acquired Graduate Basis for registration in the BPS and be registered as a Chartered Clinical Psychologist or as a Chartered Educational Psychologist (in the case of the Paediatric qualification);
- satisfy the BPS Board of Examiners that he or she can demonstrate possession of underpinning knowledge relevant to clinical neuropsychology usually, but not exclusively, by having completed a part-time, university-based accredited course (MSc or Postgraduate Diploma);
- submit a neuropsychological research portfolio comprising a clinical neuropsychological research report, a research log and supporting evidence in the form of research files (or satisfy the research component by being able to otherwise demonstrate their neuropsychological research skills, in specified ways); and
- submit a portfolio providing evidence of their clinical competence, comprising a case log, including a summary sheet, a clinical supervision log and six detailed case studies.

Obviously anyone seeking to enrol for this qualification should seek to obtain the most up-to-date guidelines available at that time, but it is clear that this qualification is being seen as a means of identifying both competent practitioners and the requirements of competent practice. Ultimately, it may permit regulation of the practice of clinical neuropsychology in the UK (BPS Membership and Qualifications Board, 2002b) and may clearly influence the principles of ethical practice by which clinical neuropsychologists work and against which they may face disciplinary action.

Indeed, the BPS's Division of Neuropsychology (2000) has already set out guidelines for professional practice in the field of clinical neuropsychology. While always subject to development and revision, these guidelines emphasize the importance of the level of competence and experience of the person wishing to offer clinical neuropsychological services and the need to seek supervision from a Practitioner Full Member of the Division of Neuropsychology where such experience might be lacking, both with respect to administration and interpretation of neuropsychological tests. In addition, eligibility for Practitioner Full Membership of the Division of Neuropsychology is seen as justification for the clinician to see himself or herself as competent to act as an expert witness in medico-legal matters (see also Chapter 15).

Of course, not everyone required to demonstrate neuropsychological knowledge in their clinical work in non-neuropsychology settings will wish (or find it possible) to acquire the Practitioner Full Membership qualification. However, this should certainly not dissuade clinical psychologists from acquiring sufficient skills in neuropsychological assessment and test interpretation in order to, within the

boundaries of their level of competence, undertake effective pieces of work with their patients and know when to refer on to more specialist services.

CONCLUSIONS

This is a particularly interesting time to be presenting an overview of key areas of clinical neuropsychological practice and its relevance to clinical psychology practice in general. This is because of the enormous development of the neuroscientific context in which neuropsychology is housed, the increasingly sophisticated neuro-psychological assessment techniques available and the considerable strides made in developing evidence-based treatments for patients with neuropsychological impairments. It is an area of clinical work in which clinical psychologists can see themselves as uniquely skilled and able to make an important contribution to the overall care of their patients.

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