NEUROLOGICAL INVESTIGATIONS

Neuropsychological assessment

Lisa Cipolotti, Elizabeth K Warrington

Patients with brain damage may present with impairments of memory, language, perception, thought, action, and other functions. These cognitive deficits can occur both in multiple domains or as highly selective impairments. In the 19th century and early 20th century, neurologists investigated cognitive impairments in patients with neurological disease by clinical and descriptive methods. These methods provided new insights and allowed the isolation of distinct syndromes—for example, aphasia,^{1,2} alexia and agraphia,³ acalculia,⁴ visual agnosia,⁵ and amnesia.⁶ Indeed, these discoveries formed the basis for the development of a new discipline, “neuropsychology”, devoted to the study of the relation between the brain and cognitive functions. The clinical and descriptive methods, however, provided a poor standard of description of the cognitive impairments in these syndromes. They were “. . . little more than the bald statement of the clinical opinion of the investigator. . .”.⁷

To deal with this lack, neuropsychologists developed principled techniques for the measurement of cognitive functioning. In the early days, psychometric tests, originally developed for the measurement of either scholastic attainment or occupational guidance, were used. In particular, tests for the measurement of intellectual and memory functions became available to the clinician.^{8–11} Gradually, over the past four decades an increasing number of measurement tools have been specifically designed for investigating the cognitive functions of patients with suspected or confirmed cerebral disease. Neuropsychological assessment involves the use of a series of tests that are “reliable”—in the same circumstances they produce the same result—and “valid”—they measure what they are designed to measure.

The aim of this paper is to provide an overview of the main methods for the assessment of cognitive function and an outline of what may prompt a neuropsychological assessment (see also Lezak,¹² Crawford *et al*,¹³ and Hodges¹⁴). Before approaching a neuropsychological assessment it is necessary to have a general theoretical structure on which to base and interpret the different levels of disturbance that can arise as a result of cerebral damage. (for a similar view see Hodges¹⁴) In the next three sections we discuss our gen-

eral theoretical schema; the methods of assessment of cognitive function; and the purposes of a neuropsychological examination.

General theoretical schema

Our approach makes the assumption that impairments in cognitive function can best be studied and understood by (a) assuming that there is a high degree of functional specialisation in the cerebral cortex; (b) by undertaking a modularity approach to the analysis of complex cognitive skills; and (c) by assuming that brain damage can selectively disrupt some components of a cognitive system.¹⁵ The extent to which these assumptions have a direct anatomical substrate is less established.

The idea that the human brain is highly differentiated in terms of its functional organisation is not new. The phrenologists in the early 19th century were already speculating that the convoluted surface of the brain reflected the juxtaposition of a large number of discrete cerebral organs each subserving a particular psychological faculty.¹⁶ Several years after these accounts, neurologists began to study and record impairments of the higher cortical functions and their accompanying cerebral lesions. Aphasic disorders were extensively studied and the specialised language functions of the left hemisphere were recognised.^{1,2,17} Subsequently, after the pioneering work of Jackson,¹⁸ the specialised visuo-perceptual functions of the right hemisphere were also recognised. These early workers not only localised a number of specialised functions in the brain but they also discussed their findings within a theoretical framework. For example, Lichtheim¹⁹ produced a complex diagram of the various sub-components of the language system by incorporating and expanding on Wernicke's² original scheme. In his diagram, the various sub-components of the language functions are represented as a series of “centres” (for example, the concept centre, the centre of the motor images of the words, the centre of the auditory images of the words), each of which was thought to be located in a specific area of the brain. These different functional centres were thought to be connected with each other through sets of fibre tracts. This approach—those adopting it were termed the “diagram makers”—has some resemblance to that of

Psychology Department, National Hospital for Neurology and Neurosurgery, Queen Square, London WC1N 3BG, UK
L Cipolotti
E K Warrington
Correspondence to:
Professor E K Warrington.

modern cognitive neuropsychology theorists.

Despite this, the idea that cognitive skills such as language could consist of multicomponents and be localised in different, highly specialised areas of the brain came under attack from the "global theorists".²⁰⁻²³ Of particular relevance here is the development of "mass action" theories. These theories proposed that there was no differentiation in the cortex for specific cognitive functions; rather, that it was equipotential with respect to cognitive abilities.²⁴ According to such a view, any form of neurological damage would deplete by a greater or lesser extent the available amount of some general cognitive resource and not specific cognitive functions. The amount of damage to the general cognitive resource, also termed intellect or abstract attitude, would depend on the extent of the brain damage and not on the site of the damage.

The notion that different brain regions are specialised for different cognitive functions regained popularity in the 1950s and the modern revolution in imaging techniques has made it possible to visualise these structures in the living human brain. The idea that there are cognitive processing systems that involve only specialised brain regions is now accepted "... as one of the cornerstones of modern brain science ...".²⁵ For about 95% of right handers and 70% of left handers major language, literacy (reading, writing, and calculation), verbal short term memory, verbal long term memory, semantic memory, and praxis are represented in the left hemisphere. The right hemisphere is involved in non-verbal processing such as the analysis of perceptual and spatial stimuli, spatial short term memory, visual long term memory, spatially directed attention, face recognition, topographical knowledge, and in some prosodic components of language. For those few people who do not have normal lateralisation this pattern seems to be reversed, although a very small proportion of subjects may have bilateral organisation of some cognitive skills. The anterior parts of both hemispheres have been accepted as being implicated in problem solving processes that are required in a wide range of situations including practical routines and social interactions as well as abstract reasoning tasks.⁷ The most posterior parts of both hemispheres are involved in early visual processing. Subcortical, as well as cortical, brain regions, are involved in attention and alertness. Subcortical brain regions are also involved in episodic memory, in some aspects of long term memory, and in the motor control of language.¹²

A modularity approach to the analysis of cognitive skills implies that each complex cognitive process can be thought of as consisting of a series of functionally independent specialised subprocesses.^{7 26-29} The interaction of these subprocesses results in the complex cognitive skills. The way in which the cognitive processes are organised is often characterised, similarly to the "diagram maker" approach, in terms of flow diagrams that

attempt to detail the way that the different subprocesses are brought together to perform a specific task. Empirical support for the modularity approach can be obtained at various levels including the neurophysiological, neuroanatomical, and neuropsychological.^{30 31} For example, numeracy has been fractionated into several independent components: cognitive mechanisms for number comprehension, number production, arithmetical fact retrieval, and arithmetical procedures.³²

The idea that complex cognitive skills are carried out by distinct subprocesses combined with the idea that there are highly specialised areas in the brain, has led many cognitive neuropsychologists to assume that a cerebral lesion can damage only some subprocesses within complex cognitive skills. Indeed, cognitive neuropsychologists have succeeded in showing many dissociations between the subcomponents of cognitive skills that allow valid conclusions about the nature and functions of the impaired processing components to be drawn.

Methods of assessment of cognitive functions

One of the fundamental principles underlying neuropsychological assessment is to establish whether the subject is still functioning at their premorbid optimal level or whether there has been a deterioration. Therefore, the methods used for assessing cognitive functioning in neuropsychology need to be able to provide: (a) an indirect measure of the premorbid skills of a person and (b) a measure of the present cognitive state of that person. Once the two types of measures are obtained they can be compared. This should indicate: firstly, whether the functioning has changed from the premorbid state; and secondly whether this reflects organic or functional impairment. If the results indicate organic impairment then an attempt will be made to establish the extent of the change. It is not only useful to know that a change in cognitive functioning has occurred; it is also useful to know whether the change can be characterised as global or focal. If the cognitive impairment is focal, neuropsychological measures can be used to specify more precisely the cognitive impairments: whether the impairment is indicative of lateralised dysfunction or confined to the anterior or posterior regions of the brain. In exceptional cases, it is possible to document highly selective cognitive impairments with a known and relatively precise anatomical localisation.

A comprehensive neuropsychological examination would include the assessment of: (a) premorbid ability; (b) general intellectual level; (c) memory; (d) language; (e) calculation; (f) problem solving; (g) alertness and attention; (h) visual and space perception. Ideally a cognitive profile would be constructed from performance on tests of proved validity and comparable difficulty. A long term aim for the neuropsychologist is to achieve a level of measurement for all

Example of a circle diagram of cognitive skills. Sectors of the circle have been apportioned to each of the cognitive skills examined. The concentric circles represent the level of functioning in terms of percentile score. The levels of the subject's test performance are indicated by the degree of eccentricity within a sector. In this patient the most prominent feature was a global memory impairment more pronounced for verbal than visual material. (Diagram courtesy of Newman *et al.*³³)

cognitive skills that permit comparison across tasks and is sensitive to change. The method described by Newman *et al* in their study that monitored subjects at risk for Alzheimer's disease exploited this methodology (figure).³³

In the next section we do not attempt to describe all the tests and techniques available to the neuropsychologists for investigation of all these different areas of cognition. Rather we focus on three main areas of cognitive function: intelligence, memory, and language functions. These serve to illustrate the range of techniques and procedures available for the investigation of cognitive impairments.³³

ASSESSMENT OF PREMORBID ABILITY

Various procedures are adopted for obtaining an indirect measure of a subject's premorbid skills, which can then be compared with his current level of performance. These procedures can be divided into two main types: methods that use demographic data such as age, sex, race, education, and occupation; and methods that use tests considered to be relatively resistant to neurological and psychiatric disorders. The first type of method is based on the known relation between a number of demographic variables and measured IQ*.³⁴ Not only may educational and occupational records be used as a rough estimate of a subject's optimal or premorbid level of functioning; they may also, through the use of various types of regression equations, provide a more precise and objective estimate.³⁵ One of the principle limitations of this type of technique, however, is that educational and occupational histories may not always be readily available and they may be incomplete, uninformative, or anomalous.

The second method involves the measurement of a cognitive skill that is known to be highly correlated with intellectual factors and resistant to brain damage. This method is obviously not reliant on preexisting data. Some of the first methods of this type involved the use of vocabulary.³⁶ These methods were based on the finding that patients with brain disease retained old, well established verbal skills, such as those implicated

in the verbal definition of words, long after other cognitive skills were impaired. The application of the same principle led to the development of various Wechsler deterioration indices.¹²

More recently, a measure of premorbid optimal level of functioning has been based on the overlearned skill of reading. Nelson and McKenna³⁷ first established that word reading skill, as measured by the Schonell graded word reading test³⁸ was highly correlated with general intelligence in a normal population. Nelson and O'Connell³⁹ then established that the reading of irregular words, such as "heir" or "chord", which cannot be pronounced correctly by applying the usual rules that map spelling on to phonology, were better indicators of premorbid intelligence (IQ) in demented subjects than estimates based on reading regular words. Nelson subsequently developed the National adult reading test (NART), which consists of 50 irregular words. Indeed, the NART has become one of the most commonly used measures of premorbid intelligence.⁴¹ An American version of this test is also available.⁴² One of the major limitations of the NART test is that it cannot be used with those who have poor literacy skills or in patients with obvious impairments of speech production or problems associated with dyslexia. In addition, there have been claims that patients with dementing disorders may not present with preserved irregular word reading.⁴³ Consequently these patients present with difficulties in reading the NART words, and this would result in erroneous low estimates of their premorbid IQ. In the cases of early dementia when language skills are relatively unimpaired, however, it has been shown that the NART remains stable over time and can be used as a predictor of the premorbid optimal level (Paque and Warrington, unpublished data).

ASSESSMENT OF GENERAL INTELLECTUAL LEVEL

Historically, intelligence has been defined in many different ways. For example, Spearman,⁴⁴ although he himself avoided the term intelligence, proposed the existence of a central intellectual ability, which he referred to as "*g*". Although he never actually defined what *g* was he thought that it involved "the eduction of relations and correlates".⁴⁴ In his formulation *g* referred to the determinant of shared variance among various tests of intellectual ability. An alternative view, associated with Thurstone⁴⁵ and Guilford,⁴⁶ involved the application of the term intelligence to a large set of diverse mental abilities (or factors). These included not only reasoning and problem solving on new data but also

*The term IQ was first introduced by Stern⁴⁰ to describe a method of comparing one child's score on the Binet intelligence scale with the performance of average children of the same age. It is nowadays used to indicate intellectual level by comparing a subject's performance with the average scores attained by members of the same age group.

specialised knowledge derived from prior schooling or experience. In line with this, Cattell⁴⁷ distinguished between fluid intelligence, the ability to deal with novelty and to adapt one's thinking to a new cognitive problem, and crystallised intelligence, which reflects a knowledge base and skills that have been previously acquired through learning and experience.

The available measures of intelligence reflect these different formulations of the abilities underlying intelligence. For example, Raven's test, including the coloured progressive matrices,¹¹ the standard progressive matrices,¹⁰ and the advanced progressive matrices (sets I and II)⁴⁸, are widely used for the clinical assessment of general intelligence. The various versions of this test are believed to weigh heavily on *g* and measure processes that are central to the definition of fluid intelligence (more recently also termed analytic intelligence⁴⁹). Indeed, they require abstract reasoning, induction of relations, and education.⁴⁹ The test was developed as a "culture fair" measure of general intellectual ability and because of its non-verbal format, its ease of use, and its speed (especially the coloured progressive matrices), it has gained wide use in both clinical and research settings. This may be overoptimistic as educational level has subsequently been shown to have a major effect on the normal subject's performance.⁵⁰

The Wechsler adult intelligence scale (WAIS)^{51,52} is considered to be one of the core measures for evaluating general intellectual ability. It involves six verbal and five non-verbal subtests that sample various skills. These subtests are thought to measure various mental abilities as would follow from Thurstone's⁴⁵ and Guilford's⁴⁶ views, including both the explicit knowledge base derived from educational and previous experience and the ability to deal with and solve new cognitive problems. Verbal and performance IQs are determined from the use of the Wechsler scales. Much research has focused on discrepancies between verbal and performance IQ as a means of differentiating between left and right hemisphere impairment⁵³ although this has not resulted in a general consensus.¹² Indeed, Warrington *et al*⁵⁴ suggested that such scales have little value as regards the localisation of a lesion or, for that matter, the identification of specific cognitive deficits. Nevertheless, the Wechsler adult intelligence scale-revised (WAIS-R), the successor of the WAIS is the most often used psychological test of intellectual functioning and is a cornerstone for most neuropsychological test batteries. It is also widely used with geriatric patients, and recently, normative data for people who are 75 or older have become available.⁵⁵ Numerous studies are based on the WAIS-R.

ASSESSMENT OF MEMORY

Memory is not a unitary function but rather a collection of distinct and independent components, each of which is associated with different brain structures. A broad distinction is

generally made between short and long term memory. Short term memory is considered to be responsible for the immediate retention of a limited amount of information; this information will decay in a matter of seconds if it is not refreshed. Long term memory retains larger amounts of information for longer periods—depending on the salience—which may be for minutes, days, and years. Short and long term memory functions can be further divided into verbal and visual memory according to whether they retain verbal or non-verbal information.⁵⁶ Long term memory is also subdivided into implicit (or procedural) and explicit (or declarative) memory.⁵⁷ Implicit memory retains information that affects behaviour but it is not available for conscious recollection (for example, motor skills, conditioned reflexes, priming). A further example is the three letter word stem completion task, which can be performed by guessing rather than by conscious recall.⁵⁸ In this task patients are presented with a list of words and their retention is tested either by standard recall and recognition techniques or by presenting the first three letters of the target item in a word completion task (for example, "cha"—chair). Explicit memory retains information that can be consciously accessed. It is subdivided into episodic and semantic memory.⁵⁹ Episodic memory contains information about temporally dated episodes or events and temporospatial relations among them (for instance, this can be for both autobiographical memories and memories of an artificial event such as a word list or short stories). Semantic memory contains our organised knowledge of concepts and facts as well as words and their meanings (for example, encyclopaedic memories). Most clinical assessments focus on three main types of memory functions: short term memory, episodic memory, and semantic memory.

Assessment of short term memory

The assessment of verbal short term memory usually requires the repetition of a progressively lengthening string of digits (digit span), letters, and words. The normal range of digits is five to nine. Spatial short term memory can be assessed with the Corsi block tapping test.⁶⁰ This requires the subject to tap a progressively lengthening sequence of blocks.

Assessment of episodic memory

Many tests and batteries are available for the assessment of episodic memory.⁶¹ These use either a recall or recognition paradigm and typically assess the anterograde (the ability to acquire new information) rather than the retrograde component (the ability to recall previously learnt material). One of the oldest batteries used is the Wechsler memory scale (and the Wechsler memory scale-revised), which requires the recall of both complex verbal material (for example, short stories) and visual material (for example, reproduction of geometrical designs). Some of its subtests are not dependent on memory itself but rather on attentional processes (for example, mental

control and orientation). Unfortunately all the subtests contribute to the final memory quotient. A more recently developed test for the assessment of long term verbal memory is the adult memory and information processing battery,⁶² which has many similarities to the Wechsler memory scale. The Rivermead behavioural memory tests consist of a series of tests held to have ecological validity.⁶³ A task that is also very often used for assessing verbal anterograde recall is word list learning (for example, the auditory-verbal learning test⁶⁴). For the assessment of non-verbal anterograde recall, the two most commonly used tests are the Rey-Osterreith complex figure test⁶⁵ and the Benton revised visual retention test.⁶⁶ Both require the recall of geometric figures.

Warrington⁶⁷ developed a test that used a recognition rather than a recall paradigm (the recognition memory test). The recognition paradigm was chosen because it is possible to have comparable tests of verbal and visual memory. This test incorporates the verbal and non-verbal dichotomy by having separate subtests with word and face stimuli. Age corrected percentile scores of a large standardisation sample are available. Validation of this test has shown that patients with right hemispheric lesions are impaired on the visual version and patients with left hemispheric lesions are impaired on the verbal version. It has also been shown that this test can detect minor degrees of memory deficit.³³ Clegg and Warrington⁶⁸ have also recently standardised and validated four "easy" memory tests (three recognition memory tests and a word paired associate learning test) for older adults (64 and older) that are recommended for patients in whom memory impairment is suspected but whose mental state (for example, poor attention, anxiety, or agitation) precludes longer or more demanding tests.

Most tests of retrograde verbal and visual recall have been devised for research rather than clinical purposes. They normally test recall and recognition of famous names and famous faces. Perhaps because they so quickly become dated their standardisation and validation are problematic. A relatively new test assessing autobiographical memory is an exception to this rule (autobiographical memory interview⁶⁹). This test requires the recall of personal remote facts and incidents from three epochs: childhood, early adult life, and recent experience.

Assessment of semantic memory

Patients with a semantic memory disorder present a general loss of knowledge, including object and word meaning. This deficit can manifest itself as an inability to comprehend words and identify pictures and objects. The classic syndromes of transcortical sensory aphasia and visual associative agnosia have been identified with the impairment of semantic memory.⁷⁰ There are no standardised batteries for the assessment of semantic memory because, unlike episodic memory, it has only been studied in the past 20 years,

after the seminal paper of Tulving.⁵⁹ Semantic memory can, however, be assessed through tests devised for other domains (mainly tests also used for the assessment of language disorders). To evaluate the difficulties in word definition some verbal subtests of the Wechsler scales, such as vocabulary and information, can be used. Naming tests can be used as indirect evidence of semantic memory impairment (see language section later). The pyramids and palm tree test⁷¹ was developed specifically to evaluate impairments in the understanding of concepts. There is a verbal and a pictorial version of this task devised for assessing conceptual relations. Limited normative data are available. A further test, the British picture vocabulary test,⁷² which uses a word picture matching technique, was first developed for the assessment of language developments between the ages 2 and 18. More recently it has been standardised in a normal healthy elderly population (Clegg and Warrington, unpublished data).

ASSESSMENT OF LANGUAGE

Language is not a unitary function. The most useful dichotomy is to consider spoken and written language separately.

Spoken language can be characterised as a collection of independent components, each of which is associated with different brain structures. The three main central linguistic components are phonology, syntax, and semantics.⁷³ Phonological processing analyses the constituent sounds of words. Syntactic processing analyses the grammatical aspects of language—for example, the ordering of the individual words in the sentence. Semantic processing analyses the referential meaning of words. In addition to these three components there are more specialised peripheral systems subserving articulation and prosody. Furthermore, at least for phonology and syntax, receptive and expressive deficits can occur as selective impairments.^{74–76} Disruptions in phonological or semantic processing are found at the level of single words whereas disruptions in syntactic processing are found at the level of sentences.

Assessment of spoken language

There are several traditional clinical taxonomies of the acquired aphasias principally inherited from the earliest scientific papers on language disorder.^{1–3 19} These taxonomies, based on mixed functional, anatomical, and pathological terms, have inspired the development of classic aphasia batteries. The most widely used are the Boston diagnostic aphasia examination,⁷⁶ the western aphasia battery,⁷⁷ the Porch index of communicative ability,⁷⁸ and the Aachen aphasia test.⁷⁹ The traditional taxonomies that form their basis have been questioned in so far as they failed to capture the multidimensional pattern of language breakdown, they are not useful for guiding therapy or for the detailed analysis and understanding of language disorders.⁸⁰ In this section we provide a brief account of the core

tests that could provide a framework for the more detailed assessment of a patient's language impairment. We discuss only two areas of language dysfunction: word and sentence comprehension and word and sentence retrieval.

Word and sentence comprehension—Word comprehension deficits can occur as a result of an impairment in auditory perception or as a result of an impairment in word meaning. An auditory word perception deficit can be identified in patients that have a deficit in word repetition that cannot be attributed to a more general articulatory deficit.⁸¹ It can be assessed through phonological discrimination tasks that are usually included in most of the traditional aphasia batteries. Impairment of word meaning is one component of the semantic memory disorders (see earlier) and, as the word retrieval deficit (see later), can be category specific. For example, selective deficits for abstract and concrete concepts and within the concrete domain animate or inanimate reference and even specific word class effects have all been reported.⁸² One of the most direct tests of word meaning are synonyms tests (for example, “*timid*” means “*afraid*” or “*quiet*”). Coughlan and Warrington⁸³ have offered a modest standardisation of one such test. Word meaning comprehension can also be tested by using word-picture matching tests such as the pyramids and palm tree test⁷¹ and the British picture vocabulary test,⁷² described in the semantic memory section. In addition, the recent psycholinguistic assessment of language processing in aphasia⁸⁴ is a useful research tool for assessing comprehension in the domains of verbal and visual knowledge.

One of the earliest and most commonly used test of sentence comprehension is the token test devised by De Renzi and Vignolo.⁸⁵ This test uses tokens of different shapes, sizes, and colours and the patient is given an oral instruction in progressively more complex non-redundant sentences (for example, “put the red circle between the yellow square and the green square”). There have been various modifications of the test including a shortened version by De Renzi and Faglioni and a very abbreviated version by Coughlan and Warrington.⁸³ Educationally standardised normative data are available. Parisi and Pizzamiglio⁸⁶ devised a test specifically for testing syntactic comprehension (for an English version see Lesser⁸⁷). Another test for grammatical comprehension is the test for reception of grammar.⁸⁸ This test was developed for the assessment of language developments and has been used also in the context of acquired aphasia investigations. It should be acknowledged that some normative data are available for the sentence comprehension test reviewed here and are undoubtedly very useful for in depth assessment of a patient's aphasic deficit.

Word and sentence retrieval—Word retrieval difficulties are exemplified by the syndrome of amnesic or nominal dysphasia and are often present in other aphasic syndromes and

in cortical degenerative conditions. They can be specific for particular categories such as letters, colours, body parts, proper names, and fruits and vegetables.³¹ To evaluate word retrieval difficulties naming from verbal description (for example, “what is the name of the large grey animal with a trunk”) and picture naming tests can be used. The graded naming test⁸⁹ was developed to identify very mild degrees of anomia. It comprises items of low frequency and it has been standardised in a normal population and validated in patients with unilateral lesions. The Boston naming test⁹⁰ comprises line drawings of objects and has been widely used in aphasia studies. Only a limited standardisation is available.

Spontaneous language is often elicited by complex picture description. The cookie jar theft picture from the Boston diagnostic aphasia examination is widely used for this purpose. De Renzi and Ferrari⁹¹ devised the reporter test requiring the patient to act as a reporter of the performance carried out by the examiner who acts in accordance with the commands of the token test described earlier. This test is particularly useful for the identification of impairments in grammatical sentence construction, although there are only limited normative data at present.

WRITTEN LANGUAGE

In the past 30 years cognitive neuropsychologists have investigated reading and writing disorders in detail and depth. This has resulted in the identification of new syndromes that take the description of reading and writing difficulties well beyond the classic syndrome described by Dejerine³: dyslexia with dysgraphia and dyslexia without dysgraphia. Each of these different dyslexic and dysgraphic syndromes corresponds to an identifiable impairment in a subcomponent or subcomponents of the reading and writing process. Shallice and Warrington⁹² have proposed a distinction between peripheral and central dyslexic syndromes and this dichotomy applies equally well to the dysgraphia syndromes. Peripheral dyslexias and dysgraphias result from damage to processes responsible for the categorisation of a string of letters or phonemes as orthographic or phonological entities. Central dyslexias and dysgraphias are due to impairment in the comprehension and production of a target stimulus. The study of central dyslexias and dysgraphias has provided evidence that there are at least two parallel forms of processing for reading and writing: one phonologically based and one semantic based. Phonological processing utilises a set of rules for translating print to sound or sound to print. It is used for reading or writing unfamiliar words or non-words. Semantic processing accesses meaningful representations of the words that are in the subject's vocabulary. These two types of processing can break down independently to produce different types of reading and writing impairments.

Assessment of written language

Following the seminal work of Marshall and Newcombe⁹³ a psycholinguistic method of assessment of written language disorders has gained wide popularity. This method involves the presentation of lists of words that sample contrasting psycholinguistic properties. It is thought that the data on the effect of the psycholinguistic and visual (length, script, and displays) variables coupled with an errors analysis allow conclusions to be drawn about the likely origin of dysfunction within the reading system. In this section we provide a description of some of the standardised and validated formal tests for the assessment of reading and spelling disorders.

Reading—Any assessment of reading skills should include an evaluation of a patient's ability to read both single words and text. In some peripheral dyslexias the ability to read text can be impaired whereas the ability to read single words can be spared. (for example, attentional dyslexia⁹⁴) The Neale test⁹⁵ for assessment of prose reading in children is useful in this context. In addition, most of the standard aphasia batteries include a subtest for text reading (for example, Boston diagnostic aphasia examination). For the formal assessment of single words, the two most widely used tests are the NART and the Schonell graded word reading test. Both tests are graded in difficulty and are measures of reading skills; an estimate of premorbid optimal level of functioning can also be obtained (see earlier). When assessing reading skills it is important to evaluate the patient's ability to read aloud non-words. This ability can be selectively impaired despite good word reading as in the case of phonological dyslexia.⁹⁶ No formal standardised tests for non-word reading are available; however, several lists have been devised for research purposes.

Oral and written spelling—Written and oral spelling are known to dissociate and therefore are assessed independently. For the assessment of written spelling the Schonell graded spelling test can be used. For the assessment of oral and written spelling, Baxter and Warrington⁹⁷ have recently standardised and validated a test that is sensitive to minor degrees of deficit in the general neurological population. This is a graded difficulty test; thus the raw scores can be converted into percentile scores. For patients whose poor eye sight precludes reading, spelling can provide a useful measure of premorbid abilities. The assessment of non-word spelling is also important because patients with phonological dysgraphia might present with some preserved word spelling despite impaired non-word spelling.⁹⁸ Several non-word spelling lists have been developed for research purposes.

The use of the standard word reading and spelling tests described combined with assessment of the patient's ability to read and write non-words and an analysis of the errors made by the patient identifies more than the presence of a reading or spelling disorder. It can also provide some preliminary indication of

the status of the peripheral and central processing involved in word reading and spelling. This, rather than the description of the presence or absence of a reading or spelling disorder, has a clear clinical and theoretical significance.

Purposes of a neuropsychological assessment

There are at least three main reasons for conducting a neuropsychological assessment: diagnosis, treatment and management, and research.

DIAGNOSIS

A neuropsychological assessment allows the description and evaluation of the major cognitive deficits incurred in neurological patients with possible brain disease. Furthermore, it can indicate possible neuroanatomical correlates of the cognitive impairments. A neuropsychological assessment can, at the very least, provide pointers as to whether there is unilateral, bilateral, or subcortical damage. This information can be useful in diagnosis. Neuropsychological assessment has a key role in differentiating between organic and functional disorders.^{14 99–101} There are other neurological conditions—for example, cortical atrophy, frontal and temporal lobe tumours, and undetected temporal lobe seizures—that may manifest themselves with symptoms that can be misinterpreted as functional.¹⁰² For example, patients with visual disorientation disorders due to bilateral occipital disease are often misdiagnosed on the grounds that their visual handicap seems to be disproportionate in the context of normal or near normal acuity.¹⁰³

On the other hand, patients with symptoms of pseudodementia, such as hysteria, malingering, Ganser syndrome, bipolar disorder, and other ill defined psychiatric disorders often present with an abrupt intellectual and memory failure that mimics true cognitive deficits. A neuropsychological assessment can distinguish between organic and functional disorders. It does this by highlighting discrepancies between subjective complaints and objective performance, usually detecting a number of inconsistencies in the patients' performance and a too obvious mismatch between objective performance and daily life activities. Also, the body of neuropsychological knowledge on the organisation and fractionation of cognitive skills is nowadays highly developed. Crucially, the way in which the cognitive functions can fractionate often diverges from the common sense opinion of how a cognitive function can break down. Thus the patient's pattern of performance can be interpreted as neuropsychologically convincing or unconvincing. To consider one example, a neuropsychological assessment is useful in differentiating organic and functional memory loss. Studies of patients with dense organic amnesia have shown that they can still learn new associative information provided that they are tested using implicit

learning tasks.¹⁰⁴ For example, they show savings with repeated presentations of fragmented or degraded stimuli (pictures or words) in increasing degrees of completeness. Even quite severely demented patients would show some learning with these tasks. Clearly a patient showing additional impairments on these implicit learning tasks makes no neuropsychological sense. It makes good common sense, however, to also be impaired on these tasks (I have poor memory, I can't remember things). Indeed, a poor performance on these tests may be considered indicative of functional memory loss. On the contrary, a relatively preserved performance on these tasks conforms to an organic pattern.

Another common differential diagnosis where neuropsychological assessment has a key role is between early dementia, anxiety or depressive disorders, or the normal aging process. The diagnosis of probable dementia is usually made by establishing whether there is an acquired deficit of cognition without histopathological evidence obtained from a biopsy or necropsy. Often in the early stages of a dementing illness, the clinical diagnosis cannot be supported by neuroimaging such as CT, MRI, or functional imaging. Patients with depression or anxiety may complain of intellectual or, more often, memory failure similar to the so called "worried well" patients. Usually depressive or anxiety pseudodementia should be suspected when the patient complains of the memory problem more than the carer.¹⁰⁵ In these cases recognition memory tests should be used to determine whether the failure is due to an organic condition or to anxiety or depression. It has been shown that recall tests of memory are vulnerable to the effect of anxiety and depression, whereas recognition memory tests are not.¹⁰⁶

The aging process itself is associated with cognitive and memory changes. Hence, it is often necessary to differentiate memory failure due to cognitive deterioration rather than benign senescent forgetfulness. In these cases performance in recall memory tests requiring the subjects to engage in elaborative encoding, as opposed to allowing them to devise their own encoding strategy, may discriminate those with brain damage from normal elderly subjects. For example, in word list learning the strategy of performing associations between successive words improves the overall level of recall in normal subjects.

Neuropsychological assessment can also have a central role in diagnosing presymptomatic cognitive impairments in familial neurodegenerative conditions. From the nature of the inheritance and the relatively constant ages of disease onset within a family, asymptomatic at risk subjects below the mean age of onset can be examined. Such studies have shown cognitive abnormalities in apparently asymptomatic subjects with Huntington's and Alzheimer's disease.^{33 107}

TREATMENT AND MANAGEMENT

The baselines of cognitive functioning provided by the neuropsychological examination

allow the monitoring of certain conditions. For example, successive neuropsychological examinations provide reliable indications of whether a pattern of cognitive deficit associated with head injury or stroke is changing and if so, how rapidly and in what way. This information is useful in planning the future medical and social care of the patient. Similarly, repeated neuropsychological testing of patients with degenerative disorders can provide information about their different rates of cognitive decline and thus help them and their family plan for their care. The results of a neuropsychological assessment can also be used in the evaluation of medical and surgical treatments such as those associated with subcortical pathology that is associated with cognitive slowing (for example, Parkinson's disease and hydrocephalus). For instance, obtaining repeated measures of a hydrocephalic patient's performance in a series of psychomotor tests can provide a reliable indication of whether the underlying neurological condition is improving or deteriorating. Psychomotor tests are simple verbal and non-verbal tests that involve verbal and visuomotor responses and the measurement of the patients' speed of responding.¹⁰⁸ Practice effects are minimal in these tests, which are at the same time sensitive to subtle changes in cognitive efficiency. Therefore they can be used at regular and short intervals for monitoring the patient's neurological state. Neuropsychological assessment is also particularly important in monitoring the various treatments for epilepsy.^{109 110}

The baselines of cognitive functioning provided by the neuropsychological examination can be important for planning and monitoring rehabilitation programmes. In particular, when planning such programmes, neuropsychological evaluation can provide answers to key questions such as "... what are realistic treatment goals and ... what is the patient's capacity to benefit from available treatment ...".¹² Moreover, repeated neuropsychological testing can be used to monitor the effects of the rehabilitation programme. Furthermore, the baselines of cognitive functioning provided by neuropsychological examination can be used to explain to patients and their families their relative cognitive problems so that they can both prepare and understand the type of difficulties the patient may face when he leaves the hospital.

Neuropsychological assessment has a central role in the medicolegal context. Neuropsychological data concerning the type and severity of a cognitive deficit, its prognostic value, and the implications for future care are central issues in the litigation over compensation awards.¹¹¹ In this context neuropsychological investigation is crucial in assessing the possibility of simulated disability that can sometimes occur before financial settlement.¹¹²

RESEARCH

There are two main neuropsychological research methodologies: the single case study

and the group study.⁷ For both, neuropsychological assessment procedures are a crucial element. The strength of novel findings and unexpected dissociations can only be evaluated by reference to performance of established tests of cognitive skills. In group studies that explore new hypotheses neuropsychological measures are necessary to obtain baseline data.

Neuropsychological research may consider applied clinical problems or be theoretically driven. In applied research, batteries of neuropsychological tests are commonly given to describe the cognitive profiles associated with particular neurological diseases. For example, specific cognitive profiles have been obtained for diseases such as Parkinson's disease and multiple sclerosis.¹¹³⁻¹¹⁴ Neuropsychological assessments are also used to identify specific patterns of cognitive deficits associated with Alzheimer's disease and other degenerative dementias.¹¹⁵⁻¹¹⁸ In particular, attempts have been made to differentiate various subgroups existing within a category of dementing disorders. The long term aim is to further the understanding of the disease, in particular with regard to early diagnosis and the possibility of pharmacological intervention.¹¹⁹⁻¹²⁰

Theoretically driven neuropsychological research has proved to be of fundamental importance in the study of the organisation of cerebral functions in the brain and especially in the understanding of normal cognitive functioning. Cognitive neuropsychological single case studies have been used as a legitimate type of evidence to support or criticise information processing models of normal cognition. Furthermore they can provide valuable findings that constrain the development of new theories. Over the years the medical literature has grown immensely. New cognitive deficits have been identified and important advances have been made in the understanding of the relations between components of complex cognitive skills and the loci of brain lesions. The neuropsychologist's strategy is to harness and incorporate the findings of research investigations to a clinical problem in the form of more specific and more sensitive tests of cognitive function. These improved quantitative techniques for clinical assessment can bring to light new phenomena that in turn promote further theoretical advances.

- 1 Broca P. Remarques sur le siege de la faculte du langage articule suivie d'une observation d'aphemie. *Bulletin de la Societe d'Anatomie Paris* 1861;6:330. (Translated in Herrnstein R, Boring EG. *A source book in the history of psychology*. Cambridge, MA: Harvard University Press, 1965.)
- 2 Wernicke K. Der aphasische Symptomenkomplex. *Breslau*. (Translated in: *Boston studies in philosophy of science* 1874;4:34-97.)
- 3 Dejerine J. Contribution a l'etude anatomoclinique et clinique des differentes varietes de cecite verbal. *CR Hebdomadaire des Seances et Memoires de la Societe de Biologie* 1892;4:61-90.
- 4 Henschen SE. *Klinische und anatomische Betrag zur Pathologie des Gehirns*. Stockholm: Nordiske Bokhandeln, 1920.
- 5 Lissauer H. Ein Fall von Seelenblindheit nebst einem Beitrag zur Theorie derselben. *Archiv fur Psychiatrie* 1890;21:222-270. (Edited and reprinted in translation by M Jackson Lissauer on agnosia. *Cognitive Neuropsychology* 1988;5:155-192.)
- 6 Korsakoff SS. Uber eine besondere Form psychischer Störung combinirt mit multipler Neuritis. *Archiv fur Psychiatrie und nervenkrankheiten* 1889;21:669-704.
- 7 Shallice T. *From neuropsychology to mental structure*. New York: Cambridge University Press, 1988.
- 8 Wechsler DA. *Wechsler-Bellevue intelligence scales*. New York: Psychological Corporation, 1939.
- 9 Wechsler DA. A standardized memory scale for clinical use. *J Psychol* 1945;19:87-95.
- 10 Raven JC. *Standard progressive matrices*. London: H K Lewis, 1938.
- 11 Raven JC. *Coloured progressive matrices*. London: H K Lewis, 1956.
- 12 Lezak MD. *Neuropsychological assessment*. 2nd ed. New York: Oxford University Press, 1983.
- 13 Crawford JR, Parker DM, McKinlay WW. *A handbook of neuropsychological assessment*. Hove, UK: Erlbaum, 1992.
- 14 Hodges JR. *Cognitive assessment for clinicians*. Oxford: Oxford University Press, 1994.
- 15 McKenna P, Warrington EK. The analytical approach to neuropsychological assessment. In: Grant I, Adams KM, eds. *Neuropsychological assessment of neuropsychiatric disorders*. New York: Oxford University Press, 1986:31-47.
- 16 Gall F, Spurzheim G. Research on the nervous system in general and on that of the brain in particular. 1809. (Reprinted in: Pribram K, ed. *Brain and behaviour: Vol 1. Mood states and mind*. Harmondsworth: Penguin Books, 1969.)
- 17 Bastian HC. *Aphasia and other speech defects*. London: Lewis, 1898.
- 18 Jackson JH. Case of large cerebral tumor without optic neuritis and with left hemiplegia and imperception. *Royal Ophthalmological Hospital Reports* 1876;8:434-44.
- 19 Lichtheim L. On aphasia. *Brain* 1885;7:433-84.
- 20 Freud S. *Zur Auffassung der Aphasien*. Vienna: Deuticke, 1891.
- 21 Marie P. Revision de la question de l'aphasie: la troisieme convolution frontale gauche ne joue aucun role speciale dans la fonction du langage. *Semaine Medicale* 1906;21:241-7. (Reprinted in Cole MF, Cole M, eds. *Pierre Marie's papers on speech disorders*. New York: Hafner, 1971.)
- 22 Head H. *Aphasia and kindred disorders of speech*. Cambridge: Cambridge University Press, 1926.
- 23 Goldstein K. *Language and language disturbances*. New York: Grune and Stratton, 1948.
- 24 Lashley KS. *Brain mechanisms and intelligence*. Chicago: University of Chicago Press, 1929.
- 25 Kandel ER, Schwartz JH, Jessell TM. *Principles of Neural Science*. 3rd ed. New York: Elsevier, 1991.
- 26 Marr D. *Vision*. San Francisco, CA: Freeman, 1982.
- 27 Chomsky N. Rules and representations. *Behavioral and Brain Sciences* 1980;3:1-61.
- 28 Morton J. The status of information processing models of language. *Philos Trans Roy Soc Lond Biol* 1981;295:387-96.
- 29 Fodor JA. *The modularity of mind*. Cambridge, MA: MIT Press, 1983.
- 30 Zeki S, Lamb M. The neurology of kinetic art. *Brain* 1994;117:607-36.
- 31 McCarthy RA, Warrington EK. *Cognitive neuropsychology*. New York: Academic Press, 1990.
- 32 McCloskey M. Cognitive mechanisms in numerical processing: evidence from acquired dyscalculia. *Cognition* 1992;44:107-57.
- 33 Newman SK, Warrington EK, Kennedy AM, Rossor MN. The earliest cognitive change in a person with familial Alzheimer's disease: presymptomatic neuropsychological features in a pedigree with familial Alzheimer's disease confirmed at necropsy. *J Neurol Neurosurg Psychiatry* 1994;57:967-72.
- 34 Matarazzi JD. *Wechsler's measurement and appraisal of adult intelligence*. 5th ed. Baltimore: Williams and Wilkins, 1972.
- 35 Crawford JR. Current and premorbid intelligence measures in neuropsychological assessment. In: Crawford JR, Parker DM, McKinlay WW, eds. *A handbook of neuropsychological assessment*. Hove, UK: Erlbaum, 1992:21-49.
- 36 Yates AJ. The use of vocabulary in the measurement of intellectual deterioration. *Journal of Mental Science* 1956;102:409-40.
- 37 Nelson HE, McKenna P. The use of current reading ability in the assessment of dementia. *Br J Soc Clin Psychol* 1975;14:259-67.
- 38 Schonell F. *Backwardness in the basic subjects*. London: Oliver and Boyd, 1942.
- 39 Nelson HE, O'Connell A. Dementia: the estimation of premorbid intelligence levels using the new adult reading test. *Cortex* 1978;14:234-44.
- 40 Stern W. *Psychologische Methoden der Intelligenz-Prufung*. Leipzig: Barth, 1912.
- 41 Nelson HE. *The national adult reading test*. Windsor: NFER-Nelson, 1991.
- 42 Grober E, Sliwinski M. Development and validation of a model for estimating premorbid verbal intelligence in the elderly. *J Clin Exp Neuropsychol* 1991;13:933-49.
- 43 Patterson K, Graham N, Hodges JR. Reading in Alzheimer's type dementia: a preserved ability? *Neuropsychology* 1994;8:395-402.
- 44 Spearman C. *The abilities of man*. New York: Macmillan, 1927.
- 45 Thurstone LL. *Primary mental abilities*. Chicago: University of Chicago Press, 1938.

- 46 Guilford JP. The structure of intellect. *Psychol Bull* 1956;53:267-93.
- 47 Cattell RB. The measurement of adult intelligence. *Psychol Bull* 1943;40:153-93.
- 48 Raven JC. *Advanced progressive matrices, sets I and II*. London: H K Lewis, 1965.
- 49 Carpenter PA, Just MA, Shell P. What one intelligence test measures: a theoretical account of the processing in the Raven progressive matrices test. *Psychol Rev* 1990;97:404-31.
- 50 Bolin BJ. A comparison of Raven's progressive matrices (1938) with the ACE psychological examination and the Otis gamma mental ability test. *J Consult Psychol* 1955;19:400.
- 51 Wechsler DA. *The Wechsler adult intelligence scale: manual*. New York: Psychological Corporation, 1955.
- 52 Wechsler DA. *The measurement and appraisal of adult intelligence*. 4th ed. Baltimore: Williams and Wilkins, 1958.
- 53 Bornstein RA, Matarazzo JD. Wechsler VIQ versus PIQ differences in cerebral dysfunction: a literature review with emphasis on sex differences. *Journal of Clinical Neuropsychology* 1982;4:319-34.
- 54 Warrington EK, James M, Maciejewski C. The WAIS as a lateralising and localising diagnostic instrument: a study of 656 patients with unilateral cerebral lesions. *Neuropsychologia* 1986;24:223-239.
- 55 Ryan JJ, Paolo AM, Brungardt TM. Standardization of the Wechsler adult intelligence scale-revised for persons 75 years and older. *Psychological Assessments: A Journal of Consulting and Clinical Psychology* 1990;2:408-11.
- 56 Baddeley AD. *Human memory theory and practice*. Hove, UK: Erlbaum, 1990.
- 57 Schacter DL. Implicit memory: history and current status. *J Exp Psychol Gen* 1987;113:501-18.
- 58 Warrington EK, Weiskrantz L. Amnesia: consolidation or retrieval? *Nature* 1970;228:628-30.
- 59 Tulving E. Episodic and semantic memory. In: Tulving E, Donaldson W, eds. *Organization of memory*. New York: Academic Press, 1972:382-404.
- 60 Corsi P. *Interhemispheric differences in the localization of psychological processes in man [thesis]*. University of Montreal, Montreal, 1972.
- 61 Gathercole SE, McCarthy RA. *Memory tests and techniques*. Hove, UK: Erlbaum, 1994.
- 62 Coughlan AK, Hollows SE. *The adult memory and information processing battery*. St James's University Hospital, Leeds: A K Coughlan, 1985.
- 63 Wilson BA, Cockburn J, Baddeley AD. *The Rivermead behavioural memory test*. Bury St Edmunds, UK: Thames Valley Test Company, 1985.
- 64 Rey A. *L'examen clinique en psychologie*. Paris: Presses Universitaires de France, 1964.
- 65 Osterreith PA. Le test de copie d'une figure complexe. *Archives de Psychologies* 1944;30:206-356.
- 66 Benton AL. *The revised visual retention test*. New York: Psychological Corporation, 1974.
- 67 Warrington EK. *Recognition memory test*. Windsor, UK: NFER-Nelson, 1984.
- 68 Clegg F, Warrington EK. Four easy memory tests for older adults. *Memory* 1994;2:167-182.
- 69 Kopelman MD, Wilson BA, Baddeley AD. The autobiographical memory interview: a new assessment of autobiographical and personal semantic memory in amnesic patients. *J Clin Exp Neuropsychol* 1989;11:724-44.
- 70 Warrington EK. The selective impairment of semantic memory. *Q J Exp Psychol* 1975;27:635-57.
- 71 Howard D, Patterson KE. *Pyramids and palm trees*. Bury St Edmunds, UK: Thames Valley Test Company, 1992.
- 72 Dunn LM, Dunn LM, Whetton C, Pintilie D. *The British picture vocabulary scale*. Windsor, UK: NFER-Nelson, 1982.
- 73 Lesser R. *Linguistic investigations of aphasia*. London: Arnold, 1978.
- 74 Gainotti G, Caltagirone C, Ibba A. Semantic and phonemic aspects of auditory language comprehension in aphasia. *Linguistics* 1975;154:155:15-29.
- 75 Miceli G, Gainotti G, Caltagirone C, Masullo C. Some aspects of phonological impairment in aphasia. *Brain Lang* 1980;11:159-169.
- 76 Goodglass H, Kaplan E. *The assessment of aphasia and related disorders*. Philadelphia, PA: Lea and Febiger, 1972.
- 77 Kertesz A. *Western aphasia battery*. San Antonio: The Psychological Corporation Inc, 1982.
- 78 Porch BE. The Porch index of communicative ability—theory and development. Palo Alto, USA: Consulting Psychologists Press, 1967 (revised 1971).
- 79 De Bleser R, Bayer J, Luzzatti C. Die cognitive Neuropsychologie der Schriftsprache. Ein Überblick zwei deutscher Fallbeschreibungen. In: Bayer J, ed. *Grammatik und Cognition. Linguistische Berichte, Suppl 1*. Wiesbaden: Westdeutscher Verlag, 1987:118-162.
- 80 Caramazza A. The logic of neuropsychological research and the problem of patient classification in aphasia. *Brain Lang* 1984;21:9-20.
- 81 Auerbach SH, Allard T, Naeser M, Alexander MP, Albert ML. Pure word deafness: an analysis of a case with bilateral lesions and a deficit at the pre-phonemic level. *Brain* 1982;105:271-300.
- 82 McCarthy RA, Warrington EK. Disorders of semantic memory. *Philos Trans Roy Soc Lond Biol* 1994;346:89-96.
- 83 Coughlan AK, Warrington EK. Word-comprehension and word-retrieval in patients with localised cerebral lesions. *Brain* 1978;101:163-85.
- 84 Kay J, Lesser R, Coltheart M. *PALPA psycholinguistic assessments of language processing in aphasia*. Hove, UK: Erlbaum, 1992.
- 85 De Renzi E, Vignolo LA. The token test: a sensitive test to detect receptive disturbances in aphasia. *Brain* 1962;85:665-78.
- 86 Parisi D, Pizzamiglio L. Syntactic comprehension in aphasia. *Cortex* 1970;6:204-15.
- 87 Lesser R. Verbal comprehension in aphasia: an English version of three Italian tests. *Cortex* 1974;10:247-63.
- 88 Bishop D. *Test for reception of grammar*. Oxford: Thomas Leach, 1982.
- 89 McKenna P, Warrington EK. *The graded naming test*. Windsor, UK: NFER-Nelson, 1980.
- 90 Kaplan E, Goodglass H, Weintraub S. *Boston naming test*. Philadelphia: Lea and Febiger, 1983.
- 91 De Renzi E, Ferrari C. The reporter's test: a sensitive test to detect expressive disturbances in aphasics. *Cortex* 1978;14:279-93.
- 92 Shallice T, Warrington EK. Single and multiple component central dyslexic syndromes. In: Coltheart M, Patterson KE, Marshall JC, eds. *Deep dyslexia*. London: Routledge and Kegan Paul, 1980:119-45.
- 93 Marshall JC, Newcombe F. Patterns of paralexia: a psycholinguistic approach. *J Psycholinguist Res* 1973;2:175-99.
- 94 Warrington EK, Cipolotti L, McNeil J. Attentional dyslexia: a single case study. *Neuropsychologia* 1993;31:871-85.
- 95 Neale MD. *The Neale analysis of reading ability*. 2nd ed. London: Macmillan, 1966.
- 96 Beauvois M-F, Derouesne J. Phonological alexia: three dissociations. *J Neurol Neurosurg Psychiatry* 1979;42:1115-24.
- 97 Baxter DM, Warrington EK. Measuring dysgraphia: a graded-difficulty spelling test (GDST). *Behavioural Neurology* 1994;7:107-16.
- 98 Shallice T. Phonological agraphia and the lexical route in writing. *Brain* 1981;104:413-29.
- 99 Miller E. Cognitive assessment of the older adult. In: Birren JE, Sloane RB, eds. *Handbook of mental health and ageing*. Engelwood Cliffs, NJ: Prentice-Hall, 1980.
- 100 Kendrick DC. *Kendrick cognitive tests for the elderly*. Windsor, UK: NFER-Nelson, 1985.
- 101 Caine ED. Pseudodementia: current concepts and future directions. *Arch Gen Psychiatry* 1981;38:1359-64.
- 102 Strub RL, Black FW. *Mental status examination in neurology*. 2nd ed. Philadelphia, PA: Davis, 1985.
- 103 Godwin-Austin RB. A case of visual disorientation. *J Neurol Neurosurg Psychiatry* 1965;28:453-8.
- 104 Warrington EK, Weiskrantz L. New method of testing long-term retention with special reference to amnesic patients. *Nature* 1968;277:972-4.
- 105 Rossor MN. Management of neurological disorders: dementia. *J Neurol Neurosurg Psychiatry* 1994;57:1451-6.
- 106 Coughlan AK, Hollows SE. The use of memory tests in differentiating organic disorder from depression. *Br J Psychol* 1984;145:164-7.
- 107 Bennett T, Curiel M. Early neuropsychological presentation of Huntington's disease. *Int J Clin Neuropsychol* 1989;11:90-5.
- 108 Willison JR, Warrington EK. Cognitive retardation in a patient with preservation of psychomotor speed. *Behavioural Neurology* 1992;5:113-6.
- 109 Goldstein H. Neuropsychological investigation of temporal lobe epilepsy. *J R Soc Med* 1991;84:460-5.
- 110 Jones-Gotman M. Localization of lesions by neuropsychological testing. *Epilepsia* 1991;32(suppl 5):S41-52.
- 111 McKinlay WW. Assessment of the head-injured for compensation. In: Crawford JR, Parker DM, McKinlay WW, eds. *A handbook of neuropsychological assessment*. Hove, UK: Erlbaum, 1992:381-92.
- 112 Miller H. Mental after effects of head injury. *Proceedings of the Royal Society of Medicine* 1966;59:257-61.
- 113 Lees J, Smith E. Cognitive deficits in the early stages of Parkinson's disease. *Brain* 1983;106:257-270.
- 114 Grant I, McDonald WI, Trimble MR, Smith E, Reed R. Deficient learning and memory in early and middle phases of multiple sclerosis. *J Neurol Neurosurg Psychiatry* 1984;47:250-5.
- 115 Cummings J, Benson D, Read S. Aphasia in dementia of the Alzheimer type. *Neurology* 1985;35:394-7.
- 116 Spinnler H, Della Sala S. The role of clinical neuropsychology in the neurological diagnosis of Alzheimer's disease. *J Neurol* 1988;235:258-71.
- 117 Haxby J, Grady C, Koss E, et al. Longitudinal study of cerebral metabolic asymmetries and associated neuropsychological patterns in early dementia of the Alzheimer-type. *Arch Neurol* 1990;47:753-60.
- 118 Swearer J, O'Donnell B, Drachman D, Woodward B. Neuropsychological features of familial Alzheimer's disease. *Ann Neurol* 1992;32:687-94.
- 119 Martin A. Neuropsychology of Alzheimer's disease: the case for subgroups. In: Schwartz M, ed. *Modular deficits in Alzheimer type dementia*. Cambridge, Mass: MIT Press, 1990:143-75.
- 120 Rossor MN, Kennedy AK, Newman S. Heterogeneity of familial Alzheimer's disease. In: Boller F, Forrester F, Khachaturian Z, eds. *Heterogeneity of Alzheimer's disease*. Berlin: Springer-Verlag, 1992:81-7.