Neuropsychology can be defined as the study of the relationship between brain structure and behaviour. As an experimental science, it bridges the disciplines of neurology and experimental psychology and seeks to understand how behaviour, emotion, cognition and perception may be related to, and underpinned by, the chemical, electrophysiological, anatomical and integrative functions of the central nervous system (CNS). Neuropsychology itself may be subdivided into subdisciplines.

Experimental neuropsychology is concerned with the purely academic aim of analysing the intricate relationships between normal brain structure and action, and is, perhaps, interested in the clinical effects of brain damage to the extent that studies of individuals with definable organic impairment may throw light on normal brain processes.

Cognitive neuropsychology seeks to understand the neuroanatomical and neurofunctional correlates of discrete cognitive processes and to analyse the microprocesses involved in everyday cognitive functions. It differs from experimental neuropsychology in its overt aim of dividing large-scale cognitive functions into ever smaller levels of processing, and it draws heavily on in-depth analyses of case studies of individuals with well defined CNS lesions.

Clinical neuropsychology has, as its broad aim, the assessment and rehabilitation of people with disturbed function consequent upon brain injury, illness or trauma.

While distinctions between these broad subdisciplines are not absolutely clear-cut, the focus of this chapter is on the methods of clinical neuropsychology.

Neuropsychology and psychiatry

Neuropsychology and psychiatry are both fundamentally concerned with the effects of alteration in cognition and emotion. In practice, neuropsychology is generally thought to be only concerned with services to brain-damaged people, while in principle it is applicable to all phenomena which result in alterations in mental function.
At both a theoretical and a clinical level, neuropsychology complements psychiatry by virtue of the clear observation that psychopathology, almost by definition, results in disturbance of cognition, emotion and behaviour.

Methodologically, neuropsychology offers a robust system for the measurement and quantification of cognitive function, emotional state and behavioural repertoire through standardised testing, questionnaire and observation, and it can therefore be used as a complementary system of analysis to psychiatry in the documentation and understanding of psychopathology. While it is not an alternative to psychiatric examination, neuropsychological assessment may prove useful as an aid to psychiatric diagnosis, appropriate placement, and in therapy and rehabilitation.

Furthermore, neuropsychology and psychiatry may be seen as complementary in the field of neuropsychiatry, which is broadly concerned with the understanding and treatment of psychological disturbance as a direct consequence of CNS trauma, injury or disease.

The methods of neuropsychology and psychiatry are clearly quite different, and the initial emphasis of this chapter is to understand the nature of neuropsychological methods. To understand this in context, however, it is necessary first to review briefly the historic antecedents of modern neuropsychology.

**History of neuropsychology**

Neuropsychology has its origin as far back as the early to mid-19th century and is exemplified by studies such as those of Broca, Wernicke and Dax, who observed a clear relationship between specific disturbances of language and loss of tissue at discrete sites in the CNS. We still think of language disturbances (that is, the aphasias) as distinct entities associated with localisable brain damage, for example Broca’s aphasia associated with lesions of the third frontal convolution of the dominant (usually left) hemisphere. The approach and studies of such individuals led to the fashionable view that most, if not all, cognitive functions could be clearly localised to discrete cortical tissue.

This ‘localisationist’ view of brain function still influences current theoretical views and clinical practice in its endeavour to assess brain damage. This paradigm is evident in the structure and interpretational guidelines of most clinical test batteries in common use to detect the presence and nature of brain damage (the Halstead-Reitan Battery (HRNB), the Wechsler Scales of Intelligence and Memory, Raven’s matrices, etc.) (Incagnoli et al., 1986).

Batteries such as the Halstead-Reitan have their genesis at theoretical level (that is determining the key cortical sites responsible for intelligence) and practical level (namely assessing the impact of penetrating missile wounds on the individual). This battery stands as a representative of the psychometric tradition in neuropsychology, an approach which may be seen to have a number of specific elements.
(1) Tests or test items are administered in an inflexible and rigid fashion to standardise administration across subjects and examiners. Thus the format of the test and the instructions and questions are expected to be rigidly adhered to.

(2) The results of each test or item (be this verbal, written, or drawn) are scored according to specific criteria.

(3) The overall results of a test are interpreted in two specific ways:
   (a) by recourse to overall population statistics which provide cut-off points or ranges of ability (i.e. normative data derived from a range of clinical populations such as normal versus brain damaged; right versus left frontal damage)
   (b) by, in some instances, comparing intra-individual variability across test procedures or subtests to determine whether the variability is normal or abnormal.

This approach additionally permeates research into the differences in cognitive, sensory, motor and perceptual function between the right and left cerebral hemispheres.

This is not to imply that the localisation of brain function or dysfunction is misguided, since there are statistically and clinically determined relationships between specific cognitive disturbances and damage to discrete cortical and subcortical areas. Where, perhaps, such an approach has its limitations is in its attempt to 'locate' complex cognitions, such as reasoning and abstraction, to single sites of action while ignoring the possibility that such complexity is likely to reflect integrated function across separate processing sites. This integrational approach to brain-behaviour relationships is best exemplified by the approach of the pre-eminent Soviet neuropsychologist, A. R. Luria (1966).

Luria's model is essentially three-dimensional in that, more explicitly than other approaches, it acknowledges the interaction between cortical and subcortical systems. It additionally relies on the concept of functional systems. Thus Luria envisaged that complex processes arise from the concerted and integrated action of separate and autonomous cortical and subcortical processing sites. Thus, for example, language comprehension may be viewed as a complex interaction of more discrete cognitive faculties such as phoneme and grapheme discrimination, visuoperceptual and auditory-perceptual function, syntactic manipulation, semantic processing and recognition and recall abilities. Each cognitive process may reflect the operation of autonomous and encapsulated processing units. When, however, their action is integrated in a related and concerted fashion, the functional result is that of language comprehension. This integrated action of independent units constitutes the functional system.

Accordingly, the understanding of cognitive processes and their dysfunction relies on analysis of individual processing units, the networked actions of such units, and the nature of communication between such units.
Clinically, these two approaches give rise to very different methods for determining the integrity of brain function. On the one hand, therefore, clinical neuropsychology uses highly standardised or psychometric procedures (such as the revised Wechsler Adult Intelligence Scale (WAIS - R) and Wechsler Memory Scale (WMS-R)) whereby the administration and scoring of tests is a structured, inflexible process. The interpretation of such tests is, almost without exception, based upon normative statistical data and hence a statistical process of analysing a person’s ability against population norms. On the other hand, the approach expounded by Luria results in an idiosyncratic and flexible administration of procedures designed to assess specific cognitive processes. Test items may be modified or manipulated by the examiner to determine the individual’s strengths and weaknesses, and there is a greater emphasis on how people approach the task and why they fail, rather than just whether or not they fail. Test results are not necessarily interpreted on the basis of quantifiable scores but more qualitatively, and the interpretation ultimately reflects the skill of the examiner and his/her approach to, or model of, brain function. This latter point emphasises that neuropsychological tests do not, of themselves, measure brain function or damage. Rather they measure aspects of cognitive function which are interpreted in relation to models of brain function and damage.

Clinical assessment is ultimately not an ‘either-or’ process in terms of these methods, and they should not be seen as mutually exclusive. To exemplify this, the original clinical procedures of Luria have been modified and revised by Charles Golden and colleagues at the University of Nebraska. This has resulted in the development of a standardised administration, scoring and interpretational process while still retaining the elements of flexibility espoused by Luria. The resultant test - the Standardised Luria-Nebraska Battery - may in a real sense be seen as a merger of widely differing practices and principles.

The process of neuropsychological assessment

Neuropsychological assessment may be used clinically:

1. to determine the presence or absence of organic pathology
2. to determine, within the individual, the interaction between organic and non-organic processes which lead to pathology
3. to determine change of function over time, for example as a consequence of treatment or spontaneous recovery, or alternatively to monitor deterioration
4. to plan cognitive rehabilitation.

Assessment comprises initially a comprehensive interview covering medical, psychological, social, educational and vocational history in relation to the emergence and course of disturbed cognition, emotion and behaviour.
In addition it is clearly desirable to obtain a detailed account of the manifestation of cognitive disturbance within the person’s natural environment. More often than not this constitutes an extended interview with the client and relatives.

On the basis of such interviews, an assessment is instigated, the precise nature of which undoubtedly varies across practitioners. One approach is the application of a comprehensive, standardised battery such as the Halstead-Reitan or Luria-Nebraska battery (see Appendices 2 and 3 to this chapter). These have the advantage of being psychometrically robust, highly reliable discriminators, with clear normative data. Their disadvantages are the lengthy administration time and the need for lengthy training and experience to avoid misinterpretation. Their accuracy, in the diagnostic sense, does however make them desirable tools, their sensitivity to the presence of brain damage being of the order of 95%.

An alternative (but again not mutually exclusive) approach would be a stepwise process such as follows:

1. assessment of fundamental problem areas (using specific procedures such as an aphasia battery, memory test or tests of perceptual disturbance); the aim here is to obtain a precise understanding of the degree of disturbance of a specific function
2. use of general organic screening procedures (e.g. tests such as the Stroop, Category Test and Trail Making Test are highly sensitive to the presence of brain damage but tell us little, when used in isolation, in respect of possible localisation); the use of organic screens helps to determine whether the primary problems reflect organic impairment (brain damage) or functional (psychogenic) disturbance
3. flexible assessment to determine precise cognitive strengths and weaknesses, thereby gaining a more complete understanding of the specificity of disturbance as well as documenting intact functions or retained abilities
4. further testing, determined by hypothesis testing (namely a hypothesis of localisation); such an approach may be termed ‘hypothesis driven’.

In addition to specific cognitive testing, use may be made of self-reporting questionnaires of emotional state such as anxiety, depression, hypomania, anger or hostility, and so on.

The interpretation of a neuropsychological profile, particularly for differential diagnosis, must be made on the basis of a convergent analysis of the test results and the consistency between test results and symptoms. Thus:

1. The presence or absence of brain damage ought in most cases to be made upon confirmation from a number of organic screens rather than a single item.
2. It is necessary to determine the precise reasons why an individual fails on a particular test. Thus the Trail Making Test is an example of an
organic screening procedure which assesses visual scanning, number and letter sequencing, rote memory, visuomotor coordination, psychomotor speed, and ability to alternate between conceptual categories. Practically, this test comprises two components. On the first, the examinee is required to draw a continuous trail sequentially between the numbers 1 to 25 (presented in a random spatial arrangement). In the second part the examinee connects the numbers 1 to 13 and letters A to L in an ascending but alternating fashion (1-A-2-B-3-C, etc.). While failure on this test may indicate acquired brain damage, it may not be clear why the individual failed or which specific cognitive process is impaired. Thus failure could arise from numeracy difficulties, problems in scanning, ordering or sequencing, poor planning, psychomotor disturbance or inability to shift mentally between categories. In practice, the precise determinants of failure can be ascertained by further administration of more discrete and subtler tests of each process.

(3) Similarly the determination of reduced concentration as, for example, by simple digit span procedures, may tell us little about the facets of attentional disturbance, and more exacting procedures ought to be used. It should also be noted, and is often forgotten, that digit span assesses auditory/verbal concentration and is, therefore, an incomplete and perhaps simplistic examination of concentration abilities.

(4) If interpretation involves localisation of brain damage, then clearly that interpretation ought to account for all observed areas of retained and disturbed function.

(5) Disturbance of function as detected by assessment ought, in the final analysis, to complement and explain the described symptoms.

As for most investigative processes, neuropsychological interpretation is not 100% accurate in its sensitivity to brain damage, and cannot be used as a 'stand alone' system. In practice neuropsychological assessment is part of a multidisciplinary process in which neurological, neurophysiological, neuro-radiological and neuropsychological data are examined and analysed for consistency by the skilled practitioner in the formulation of diagnosis. However, assessment batteries such as the Halstead-Reitan boast a discriminative accuracy in the region of 85-95% (Incagnoli et al, 1986) - such tests will accurately classify an individual as normal or brain damaged on 85-95% of occasions. This compares extremely favourably with electroencephalography (Chapter 8), computerised tomography, or magnetic resonance imaging (Chapter 9).

There are, therefore, major strengths with neuropsychological test procedures. Their reliability and accuracy make them powerful tools for diagnosis, descriptive interpretation and rehabilitation. Their major weaknesses fall within the area of cost-effectiveness – they are time consuming and hence relatively expensive.
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<thead>
<tr>
<th>Site of damage; cognitive disturbance</th>
<th>Test</th>
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<td>Frontal lobe</td>
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<td>Expressive language disorders</td>
<td>Benton Verbal Fluency Test</td>
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<td>Disorders of planning, programming, execution, monitoring</td>
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<td>and regulation of behaviour</td>
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<td>Disturbance of reasoning, abstraction and logical analysis</td>
<td>Trail Making Test</td>
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<td>Rigidity of cognitive processing</td>
<td>Wisconsin Card Sorting Test and Halstead Category Test</td>
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<td>Disorganisation of complex motor actions</td>
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<td>Disturbance of focused attention</td>
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<td>Memory disturbance</td>
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<td>Dysregulation of mood</td>
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<td>Personality disturbance</td>
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<td>Temporal lobes</td>
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<td>Auditory-perceptual defects</td>
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<td>Visual field disturbances</td>
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<td>Disorders of visual organisation</td>
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<td>Short-term memory deficits</td>
<td>Benton Visual Retention Test, Complex Figure of Rey, and Revised</td>
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<td>Wechsler Memory Scale</td>
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<td>Amnesic syndrome</td>
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<td>Receptive language disorders</td>
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<td>Sensory and perceptual disturbance</td>
<td>Left-Right Disorientation Test</td>
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<td>Disorders of intersensory association</td>
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<td>Spatial disorientation</td>
<td>Complex Figure of Rey, Block Design and Object Assembly of the</td>
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<td>Impaired location and topographical memory</td>
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<td>Constructional apraxia</td>
<td>Arithmetic Subtest of the WAIS-R and Arithmetic Scale of the</td>
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<td>Spatial dyslexia and dyscalculia</td>
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<td>Unilateral spatial neglect</td>
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<td>Spatial agnosias</td>
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<td>Receptive language disorders</td>
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<td>Occipital lobe</td>
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<td>Cortical blindness</td>
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<td>Visual agnosias</td>
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<td>Pure word blindness (agnosic alexia)</td>
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Ultimately there are a number of approaches to neuropsychological testing and innumerable tests which can be used to assess cognitive function. To document the nature, characteristics and functions of each of the major tests is beyond the scope of this chapter. However, Appendix 1 of this chapter describes and details commonly used and widely known procedures in terms of their aims, uses and key characteristics, while Table 5.1 summarises tests which may be of particular value in localising brain damage. For a detailed review of neuropsychological procedures, the reader is referred to Lezak (1983).

Additionally, to facilitate awareness of commonly used neuropsychological test batteries, Appendix 2 and Appendix 3 detail respectively the Halstead-Reitan and Luria-Nebraska tests. It is worth stating again that the provision of such batteries should not convey the impression that they are either easy to administer or unproblematic to analyse - they should not be considered 'assembly kit’ approaches to neuropsychological diagnosis.

In summary, neuropsychological assessment may be seen as an approach to the quantification of cognitive function. In the interpretation of neuropsychological data, inferences as to the integrity of brain function and the localisation of brain damage are made on the basis of the pattern of cognitive strengths and weaknesses within the individual. It must be borne in mind, however, that neuropsychological interpretation rests only partly on numerical scores obtained from performance tasks.

As important, and in some instances more so, is a more qualitative understanding of the nature of and changes in cognitive, emotional and behavioural status reported by the individual.

Since neuropsychological interpretation in the determination of brain damage is an inferential process, it is not strictly equivalent to or an alternative to neurological diagnostic procedures such as radiological scanning, and it is extremely rare that interpretation of disease process can be made from neuropsychological assessment in isolation. This brings to the fore the need to view neuropsychological assessment as an investigative adjunct to other clinical examinations. It is only, perhaps, in the realm of cognitive rehabilitation that neuropsychological assessment may be viewed as operating largely independently from neurological examination.

**Neuropsychological testing in clinical practice**

Cognitive, behavioural and emotional function may be disturbed in a variety of conditions in an apparently similar fashion. Certain psychiatric disturbances such as reactive and endogenous depression, intractable anxiety and psychosis may be associated with disturbance in specific cognitive abilities such as concentration, short-term memory, learning ability, and intellect. The process then of discriminating between cognitive disturbance as a consequence of organic or psychiatric processes is extremely difficult as is, for example, discriminating discrete cognitive change as a consequence of specific types of organic syndrome.
The following sections summarise the neuropsychological data relevant to a number of organic and psychiatric conditions. The aim is not to provide an exhaustive review, but to summarise the key neuropsychological findings associated with particular disorders, in order to aid the clinician’s diagnosis.

Testing of patients with psychiatric disorders

Schizophrenia

Our view of schizophrenia has gone through marked swings, from predominantly sociopsychological theories through to a firm determination that the basis of schizophrenia is biological in nature. Current thinking is probably consistent with an interactionist-type model, in which it may be argued that the vulnerability towards schizophrenia is genetically determined and may constitute some disturbance in brain function. The manifestation of schizophrenia as a complex disturbance of thought, emotion and behaviour may best be thought of as a consequence of an on-going interaction between constitutional vulnerability and environmental stressors (see Birchwood et al., 1988). Biological research also strongly supports the conclusion that schizophrenia is a heterogeneous disorder - that is, that there may be several distinct types of schizophrenic subsyndrome, each of which may have different aetiologies.

One such dichotomy which has been purported to reflect aetiological differences is that of type 1 versus type 2 schizophrenia. Type 1 refers to schizophrenic symptoms which may be considered positive additions to behaviour (e.g. the first-rank symptoms of Schneider). Type 2 schizophrenia, or negative schizophrenia, is characterised by losses of behaviour and emotion, such as withdrawal or flattening of affect. The separation of type 1 and 2 schizophrenia implies clearly distinguishable subtypes, each of which is argued to reflect distinct anomalies. The distinction also clearly implies that, clinically, individuals may only manifest type 1 or type 2 symptoms, when in reality they present with a mix of positive and negative symptoms. It is also evident that many of both the positive and negative symptoms once thought to be pathognomonic of schizophrenia are in fact found in other forms of psychosis, such as mania and manic-depression.

The search for distinctive CNS substrates of schizophrenia is unlikely to be clear cut; however, elucidating distinct mechanisms which may be associated with specific types of psychotic symptoms is both theoretically and clinically important. One can perhaps support this view by drawing parallels with neuropsychological research into traumatic head injury. Thus, an understanding of the cognitive correlates of, for example, diffuse axonal injury, concussion, shear damage, orbitofrontal damage and localised ischaemic changes is of clear
clinical importance in being able to understand the precise needs of the head-injured patient. This clinical and research database is of additional value in neuropsychiatry, medicolegal assessment and in determining within the individual the relative influence of organic and non-organic factors. It is partly because of this database that the neuropsychologist is able to formulate inferences as to the presence and type of brain damage from patterns of cognitive impairment.

The important point in the present discussion is that such inferences are possible despite the fact that head-injured patients rarely present with local damage or single symptoms. Rather, patients will present with mixed symptoms underpinned and produced by the operation of multiple organic mechanisms, with the acknowledgement that particular symptoms arise by virtue of specific mechanisms.

In the same manner it is theoretically possible to examine the neuropsychological and neuroanatomical correlates of specific schizophrenic symptoms in the hope of determining neurobiological determinants of schizophrenia, despite the fact that schizophrenic patients present with mixed symptoms. With this in mind it remains instructive to review evidence suggesting distinct neurobiological correlates of negative and positive symptoms.

**Negative symptoms**

The evidence from biological studies indicates that the negative symptoms of psychosis may reflect a more generalised form of brain damage. Studies on schizophrenic populations presenting with predominantly negative symptoms (usually chronic patients) indicate that, relative to normal subjects, there is evidence of a significantly higher ratio of the size of the ventricles to the size of the brain; schizophrenic patients presenting with a predominance of positive symptoms do not have a higher ratio. While the specific significance of this increased ventricle:brain ratio are not known, there is evidence that it reflects cortical atrophy, including neuronal loss, rather than raised intracranial pressure.

Neuropsychological studies indicate that negative schizophrenia is characterised by overall deterioration in IQ, disturbances in short-term memory, and generalised neuropsychological performance deficits (such as sensory and motor disturbance, perceptual difficulties, and deficits in higher order reasoning). These deficits are similar to those associated with certain global types of brain damage such as dementia or damage by virtue of long-term alcohol abuse.

**Positive symptoms**

The symptoms classified as positive are in themselves unusual and include specific types of auditory hallucination, feelings of passivity (as if someone
else is controlling one's behaviour) and specific types of delusions of significance. These types of symptoms were originally thought to be unique to schizophrenia, although it is now clear that they are evident in other forms of psychosis as well as in specific organic states such as temporal lobe epilepsy and in tumours and necrosis of the corpus callosum.

The evidence for an anatomical correlate of positive symptoms does not suggest global changes in neuronal density. Thus in the normal brain there is evidence to indicate that the left planum temporale is significantly wider than the right planum temporale, this asymmetry apparently correlating with the fact that the left hemisphere is usually the one dominant for language comprehension. Additionally, in normal brains, the right frontal plane is significantly wider than the left frontal plane (although the significance of this is not clearly understood). In contrast, schizophrenic patients with predominantly positive symptoms tend to show the reverse pattern of asymmetry. It has been argued that this reversal forms the basis of abnormalities in cognitive processing which may result in such symptoms as auditory hallucinations and feelings of passivity (see Birchwood et al, 1988).

**The unilateralist model**

The best way to summarise the neuropsychological findings is in relation to current theoretical modelling. At present it may be argued that there are a few core hypotheses as to the nature of the brain deficit associated with positive symptoms. The first of these may be considered as a unilateralist model, in which it is argued that the primary disturbance of function in schizophrenia arises as a consequence of subtle damage or impairment to the dominant frontotemporal circuitry (e.g. Flor Henry, 1969), a hypothesis derived from the observation of similarities between the symptoms of schizophrenia and of temporal lobe epilepsy where the epileptic focus is on the left and not the right side.

At a neuropsychological level, there is also consistent evidence of a left temporofrontal disturbance. Thus schizophrenics tend to perform poorly on neuropsychological tests that may be considered to assess frontal function and temporal function (such as the Category and Wisconsin Card Sorting tests, and the Speech Sounds Perception test respectively). Thus, by way of example, schizophrenic people tend to show poor performance on Trails B and not Trails A of the Halstead-Reitan battery (Trails B specifically assesses the ability to shift flexibly between cognitive sets, inflexibility being most clearly associated with frontal lobe dysfunction). In addition they show a weakening of dominance on the right hand in comparison with the left, as manifested by reduced finger tapping speed. On tests such as the Category Test and Wisconsin Card Sorting Test (which assess higher-order reasoning), schizophrenic people again do far more poorly than either controls or other psychiatric controls.

In addition there is evidence to indicate specific disturbances of short-term memory, in particular verbal short-term memory (Kapur, 1988). The precise
characteristics of this memory disturbance are again not clearly understood, but it is thought that they may reflect both specific features of concentration as well as encoding and storage of information.

If we view specific test procedures as assessing functions which are in principle localisable to specific areas of the nervous system, then there does seem to be an abundance of evidence to indicate that those tests which assess temporal or frontal lobe function are more poorly carried out by schizophrenic populations than by others. It must be borne in mind, however, that the differences between groups on such tests are statistically derived and it is not necessarily the case that the magnitude of deficit on a particular test is of clinical significance. We must therefore be very careful when interpreting purely statistical differences between groups as being of diagnostic utility as opposed to clinically meaningful disturbances of function.

In the final analysis, the value of neuropsychological discriminations relies on the fact that there is a definable entity that can be termed 'schizophrenia'. As schizophrenia is not a unitary disorder, we have to ask what type of schizophrenia we are trying to discriminate from 'traditional' organic syndromes.

Having pointed out these difficulties, it is the case that standard batteries such as the Halstead-Reitan and Luria-Nebraska batteries contain guidelines for the clinician for discriminating schizophrenia from organic states, which suggests that there may be some use in using cognitive assessment as an adjunct to psychiatric interview and neurophysiological assessment to assist in the diagnostic process.

*The integrationist model*

The second model which has been put forward may be envisaged as an integrationist approach, and argues that the primary deficit in schizophrenia lies not within a single hemisphere but arises as a consequence of faulty communication between the two cerebral systems (termed 'interhemispheric transfer deficit'). The hypothesis is derived from investigations of the split brain condition and much of the method used to assess this process is drawn from experimental neuropsychology (see Hallett (1987) for a detailed review).

The model has been put forward to explain the fact that certain positive symptoms of schizophrenia and the neuropsychological correlates of faulty interhemispheric communication are not found in research populations presenting with predominantly negative symptoms. While this model is somewhat in its infancy and requires more thorough investigation, Green (1986) and Hallett (1987) have shown its value in clinical diagnosis.

*Clinical implications*

Many of the commonly used general neuropsychological tests, such as the Halstead-Reitan Battery, Wisconsin Card Sorting Test and most memory scales,
contain population norms for discriminating schizophrenia from other psychiatric disorders and from neurological disease. However, it is clearly not the case that schizophrenia can be solely diagnosed on the basis of neuropsychological testing, nor is it being suggested that this should be the eventual aim. Rather, neuropsychological assessment may be of value to the psychiatric profession theoretically as well as clinically.

At a theoretical level, neuropsychological studies support the contention that there may be distinct CNS substrates to certain types of symptoms. In conjunction with other methods, such studies contribute to a greater understanding of the disorder and ultimately to better treatment and management.

Clinically, neuropsychological assessment may assist the psychiatric practitioner:

(1) by providing detailed information on a cognitive state, which may help in placement and in rehabilitation
(2) by contributing to an understanding of the interaction between organic and non-organic factors in an individual’s presentation or by ruling out the presence of contributory but independent factors
(3) by assisting in the diagnosis and understanding of complex cases, in which an individual’s primary problems may be adversely influenced by, for example, long-standing learning difficulty or intellectual deficit.

Anxiety states and depression

It is widely acknowledged that anxiety may adversely influence performance on neuropsychological tests, and one of the preliminary roles of the clinician is to attempt to ensure that this test-related anxiety is minimised. Similarly, it is widely accepted that adverse effects on cognitive function may arise as a consequence of depressed affect.

Both anxiety and depression affect cognitive testing by virtue of their initial influence on concentration. Thus anxiety may often result in a state of distractibility by virtue of over-worrying thoughts and heightened autonomic arousal, which have the net effect of reducing concentration span. Depression may result in a similar end state by virtue of introspective and negative thinking and difficulty in orientation to the outside world. Principally, then, they may be seen to lessen cognitive effort and active attention. Seen in this way, both states may result in an overall flattening of scores on, for example, tests of short-term memory and new learning ability (e.g. the revised Wechsler Memory Scale) as well as reduced performance on tests of concentration (e.g. Digit Span).

In principle it could be argued that depression and anxiety may interfere with performance on any task which requires concentration or cognitive speed or which has a significant loading on short-term memory. Thus in cases of severe emotional disturbance, a general flattening of cognitive performance may be seen on IQ tests such as the Wechsler Adult Intelligence Scale. Anxious
and depressed subjects may under-perform on certain subtests such as Digit Span and Digit Symbol, and may generally do relatively poorly on the Performance scales, since these are timed.

Finally, both states may result in disturbance of motor function, as a consequence of muscular tremor and rigidity in anxiety and psychomotor retardation in depression.

Since such general reductions in cognitive function might also be associated with certain generalised forms of neurological impairment, it raises the question of their diagnostic value. The resolution to this lies in a number of areas. Firstly, cognitive test results are not interpreted in isolation from medical and psychological history, which often provide the important clues to aetiology.

Secondly, depression and anxiety can in principle be isolated by the degree of impaired performance across a range of cognitive procedures (where generally it may be assumed that such psychological disturbance will result in milder forms of disturbance than that attributable to neurological impairment).

Thirdly, differential diagnosis can be assisted by comparison of an individual’s performance between those tests considered highly sensitive to the effects of mood state and tests which are more resistant to mood state.

Finally, it should be emphasised that assessment is a convergent process across a range of general procedures. Thus, in an attempt to discern whether short-term memory disturbance is of psychological or organic origin, the clinician will have recourse to organic screening tests of good clinical discrimination. In addition, the use of standardised anxiety and depression scales provides an important adjunct to cognitive methods.

To complicate matters, there is growing evidence to indicate that depression, for example, is associated with a more pervasive disruption of cognitive processes deemed to be subserved by the non-dominant (usually right) cerebral hemisphere, and that certain forms of depression may be linked to specific cognitive processes such as the inability to recognise affective components in, for example, faces, and more generally may be linked to deficits in spatial processing. Research supporting this approach carries the same sorts of practical difficulties as that associated with schizophrenia and we are, in truth, a long way off from being confident enough in this database to use it routinely in clinical practice.

Testing of patients with neurological and neurosurgical conditions

Head injury

The use of neuropsychological procedures in cases of head injury is perhaps somewhat removed from standard diagnosis. Its foremost value is in terms
of documenting the degree of cognitive disturbance, and this may have several facets:

(1) to document the individual’s strengths and weaknesses in the planning of rehabilitation programmes
(2) to determine the degree and likelihood of continuing change over the initial months following head injury
(3) to determine the degree to which the cognitive and emotional changes consequent upon head injury may be inferred to reflect primary damage to the CNS, or may reflect psychological factors associated with the trauma of head injury (in particular post-traumatic stress disorder) or with the psychological difficulties individuals may experience in coming to terms with their disabilities.

Many people with a history of head injury may at some stage find themselves referred to a psychiatrist, and neuropsychological testing provides a further valuable role in determining the degree to which current disturbance of function may be related to historic organic insult.

Ultimately, the commonest type of head injury is likely to be closed head injury through road traffic accidents or industrial or sporting injury, and brain damage may arise from concussion, haemorrhage, the destruction of grey and white matter tissue, secondary damage by virtue of meningeal intrusion or infection, as well as oedema and ischaemia. This is discussed further in Chapter 6 (p. 218-219).

The cognitive, emotional and behavioural sequelae of head injury may be deemed multifarious and clearly dependent upon the site or sites of injury, as well as the actual mechanism of injury. It would be unproductive even to begin to review the precise neuropsychological attributes of this population, and the reader may wish to peruse Richardson (1990) for a detailed exposition. For a brief résumé of the commonly observed cognitive deficits associated with damage to specific brain regions, see Table 5.1 (p. 157).

It may rather be more instructive to address certain discrete issues which serve to illustrate the importance of neuropsychology in decision making and the management of head injury.

**Amnesia**

Amnesic difficulties commonly follow head injury, that is post-traumatic amnesia (PTA) and retrograde amnesia (RA).

Retrograde amnesia refers to a specific impairment of memory for events immediately before the injury. It is considered to arise directly from shear forces within the brain and rotational movement of the brain within the skull which, respectively, give rise to diffuse lesions and more specific lacerations and contusions of the frontal and temporal regions.
The length of RA broadly varies as a function of severity of head injury as judged by duration of unconsciousness, but the number of complications surrounding this rule cast doubt on its reliability. These complications include the following:

(1) Extended periods of retrograde amnesia can occur in head-injury victims who do not lose consciousness or suffer impairment of consciousness. Furthermore, extended periods of RA may be observed in instances where other objective measures (e.g., brain scan) deem the severity of head injury to be mild.

(2) The duration of RA normally shrinks as the individual recovers, raising difficulties in determining its precise length, or in deciding at which point in time during recovery the assessed period of amnesia is important for prognosis.

(3) RA is rarely a blanket period of complete memory loss but is often characterised by islands of memory which can be difficult to assess accurately in time and which, in themselves, increase with recovery.

(4) The duration of RA in some instances can be determined as such by emotional trauma arising out of the event leading to head injury. Inasmuch as emotional trauma is difficult to quantify, the interaction makes for a complex equation in terms of relating RA to severity of head injury.

Retrograde amnesia constitutes a disturbance of memory for personal events and experiences. Prior general information and knowledge may be only partially impaired and skilled behaviour unaffected. Assessing RA is therefore, in many instances, an imprecise process of determining the most recent event or experience an individual can remember before head injury from the patient's own reconstruction of events.

The assessment of RA is useful in understanding the nature of the memory deficits responsible for memory loss and may clinically offer insights into the effects of the psychological trauma experienced, but it must be generally considered that RA is an unreliable index of injury severity and prognosis.

By contrast, PTA is usually considered to vary directly with length of coma and is widely used as a clinical guide to severity of head injury and prognosis. Additionally, length of PTA may also be used as a practical estimate of the severity of cognitive disturbance following head injury.

In principle, PTA refers to the period subsequent to head injury for which the individual has no memory and is taken to end at the time which the patient can give a clear and consecutive account of what is happening to him/her. The end of PTA, determined as a return of continuous memories, highlights the fact that PTA constitutes two separable disturbances - the first being anterograde amnesia (namely the inability to remember the continuous flow
of new experiences) and disorientation (inability to locate oneself in time and place). Thus only when both factors have normalised can it be said that PTA is ended. Estimating the duration of PTA can thus be problematic and can lead to inappropriate clinical statements if both elements are not monitored. Furthermore it is important to determine continuity of memory over a reasonable period since islands of recall can occur within an on-going dense PTA.

In addition, PTA may be intuitively seen to begin once the individual has regained consciousness. Contemporary practice, however, measures PTA from the actual time of the head injury. As such the total duration of PTA includes not only the period of anterograde amnesia and disorientation but also the duration of coma.

The above factors raise the risk of underestimating the total time of PTA and hence underestimating the severity of injury and may possibly lead to an inaccurate prognosis.

It is also possible to overestimate the length of PTA, since in most cases of moderate to severe head injury there will also be quantifiable but more focal disturbances in memory function which continue beyond the end of actual PTA.

Thus, while PTA may be seen to be an accurate predictor of severity and prognosis, care must be taken to assess its duration accurately. In this respect several factors need to be borne in mind:

(1) The relation between severity, coma length and PTA is less reliable in older clients, where extremely short duration of coma may be followed by a disproportionately long period of PTA.

(2) PTA of many hours can occur in patients who do not lose consciousness and in some such instances PTA may not begin at the time of the actual injury but some time later - the individual apparently exhibiting normal orientation and consciousness before lapsing into PTA. This point is raised since, clinically, PTA, while used as an index of severity, is often only seen as of significance for patients who present initially unconscious or comatose. This raises the possibility that in instances of PTA in the absence of coma there may be a tendency to misdiagnose.

(3) The prognostic value of PTA is most clearly seen in closed or blunt head injury, and its value is weaker in crushing or penetrating injuries in which individuals may experience shortened coma and PTA.

Notwithstanding the above, the general rule as applied to PTA stands, and PTA duration is well recognised to predict recovery, and long-term neurological, psychological and occupational outcome.

The duration of PTA comprises the time between the actual head injury and full return to continuous memory. Care must be taken therefore in evaluating whether an individual is exhibiting a full return or an island of memory. Care must also be taken when assessing PTA retrospectively, because
a patient’s reports may often contain information about earlier stages of their recovery which have been given to them by carers and relatives.

Periods of less than one hour of PTA will be difficult to measure and it is perhaps arguable whether smaller subdivisions are clinically useful, since most classifications include a cut-off point at the one-hour period.

It is arguable that assessing PTA purely by interview is difficult given the above and as such quantifiable measures have been developed to assist in estimating duration. The Wechsler Memory Scale and its revised version have been used to assess PTA since they include measures of disorientation as well as more general measures of short-term memory. In truth, however, the orientation subtest (containing questions such as ‘What day is it?’; ‘What month is it?’) may well be the most reliable measure since other aspects of these scales are also sensitive to more focal deficits of memory which persist beyond PTA.

A specific test of PTA (see Richardson, 1990) is the Galveston Orientation and Amnesia Test (GOAT), which contains 12 questions assessing simple biographical memory, knowledge of circumstances of the injury, and temporal and spatial orientation. This test yields a global index with a maximum score of 100, with separate estimates of RA and PTA. Administered at least once per day, PTA is defined as a score of less than 75, and hence PTA may be considered to have ended if a score of more than 75 is reached on several consecutive administrations. Such scales as the GOAT have also been found to be good predictors of neuropsychological outcome for patients with prolonged PTA after no or limited coma.

The ultimate key in the neuropsychological assessment of PTA is not absolutely in terms of the complexity of tests used, but in terms of repetition of assessment to determine accurate as opposed to apparent termination of PTA.

Cognitive effects

There is an accumulation of research and clinical evidence to indicate that it is the cognitive and psychosocial deficits consequent upon head injury, rather than physical disabilities, which are the primary contributors to long-term disablement and interfere most with independence. As such, the determination of the extent and precise nature of these difficulties lies firmly within the neuropsychological sphere.

Broad assessment of disablement using, for example, intellectual and memory screens (such as the Wechsler scales) carries us only part way to documenting the needs of the individual in terms of rehabilitation, although they may be of value in documenting definable change of function over time. In addition, it is often easy to misinterpret the key disturbances if one solely uses broad screening devices.

Thus the revised Wechsler Adult Intelligence Scale may indicate disturbance of intellectual function, but may under-rate the contribution of slowed information processing and deficits in arousal and attention which are common sequelae of closed head injury. Procedures such as the Luria-Nebraska
Neurological Battery arguably provide a more detailed and comprehensive examination, in that the emphasis of the test is in the determination of individual strengths and weaknesses and in identifying discrete but significant disturbances in cognitive chains.

It is useful here to consider the findings concerning an individual with closed head injury whose performance on intellectual assessment indicated a significant disparity between verbal IQ (in the average range) and non-verbal IQ (within the mentally defective range). Closer investigation revealed a specific disturbance of information processing in the rotation of information in mental space, the effect of which manifested itself across a range of apparently disparate cognitive abilities including concentration span, arithmetical calculation, and constructional ability. While clearly this adversely influenced certain features of the client's intellectual function, it could not be justifiably inferred that the client suffered from an absolute deficit in intellect. As importantly, the differential interpretation between a global IQ deficit and a specific focal disturbance (which in itself adversely influences a range of independent abilities) would have entirely different implications for treatment.

A further issue relates to the correlation between anatomical and neuropsychological assessments of the degree of brain injury following head injury. In clinical practice, it may often be inferred that a normal computerised tomography (CT) scan carries with it the implication that any disturbance of cognitive function must relate to psychological sequelae. This may be especially important many years after initial head injury, when the causal link between historic head injury and current symptoms may be difficult to ascertain, particularly if medical evidence is tenuous. Wilson (1990) points out, however, that CT is most generally sensitive to accumulation of blood after head injury, but that in many patients there may be other indices of severe brain injury in the absence of an abnormal CT scan. Thus Snoek et al (1979) observed that of 60 head-injury patients without haematoma undergoing CT, some 38% had normal CT scans despite the fact that they had experienced coma lasting over six hours.

Other studies have shown that CT has a sensitivity of only 18% for non-haemorrhagic lesions, compared with a sensitivity of 93% for magnetic resonance imaging. Neuropsychological assessment is, by contrast, in the region of 90–95% sensitive to the presence of organic damage in the same type of cases, although perhaps less so to its location.

This raises the important issue that the use of neuropsychological assessment in common clinical practice is arguably of greater value than CT in determining the degree and nature of impairment following injury and, in particular, may be of greater value in evaluating cases of mild brain damage and concussion.

Concussion is of particular importance when assessing an individual some months after initial injury. Persisting symptoms at that stage may be interpreted as reflecting a 'post-concussional syndrome' (PCS). Many clinicians adopt the view that PCS largely reflects the operation of psychological factors, particularly if symptoms persist beyond 12 months. This view is bolstered by the fact that
in many such cases CT is likely to be normal. By contrast, in many cases of PCS, the neuropsychological profile is abnormal and consistent with disturbances in concentration and short-term memory which reflect residual and focal brain damage.

A final point is the value of neuropsychology in medicolegal assessment, in which it is arguable that cognitive assessment can provide a more detailed and sensitive assessment of the effects of traumatic head injury than is the case from the interpretation of routine CT scans. The convergent nature of neuropsychological interpretation associated with the ability to discriminate between neurological and psychological factors makes it a highly desirable tool for litigation purposes.

Cerebrovascular accidents

A cerebrovascular accident (CVA) may be taken to mean any disruption of brain function which arises as a consequence of pathology involving the blood supply, and hence this broad heading covers a range of disturbances arising from haemorrhage, infarction, clotting, or venous malformation.

As with head injury, cognitive and psychosocial factors appear to be decisive in determining outcome over the longer term, and yet there are few studies relating neuropsychological indices to the outcome of stroke.

The value of neuropsychological assessment again mainly lies in the area of rehabilitation (see later). In addition, however, cognitive assessment may be used to assist diagnosis of people who may have experienced a transient ischaemic attack (or ‘mini-stroke’) or in whom the use of more invasive exploratory techniques such as cerebral angiography are contraindicated.

Site of lesion

When considering the effects of CVA there are several factors which need to be taken into account, which include the site, size and depth of the lesion. In addition the type of disease or injury which led to the CVA needs to be considered, since this may imply dysfunction by virtue of injury or systemic illness. Despite this, the organisation of the cerebrovascular system is such that specific patterns of deficit may be characteristically associated with the occlusion of the major cerebral arteries. Thus disorders affecting the anterior cerebral arteries may be associated with disturbance of higher cognitive functions such as amnesia, aphasia and adynamia (see Walsh, 1985).

Adynamia refers to an unusual state characterised by apparent alertness while being behaviourally inert and verbally unresponsive and corresponds to the psychiatric label of stupor. This may be associated with disturbance of function of the deeper aspects of the frontal lobes, but additionally may arise as a function of damage to the anterior of the corpus callosum, which is thought to play a significant role in both the regulation and initiation of speech. This may well be associated with language difficulties such as reduced
verbal fluency and, in extreme cases, by akinetic mutism. These difficulties may reflect the disruption of the regulation of speech arising from more general disturbance in motor control.

The amnesia resulting from CVA has been likened to that associated with Korsakoff’s psychosis, and is characterised by confusion, temporospatial disorientation, and confabulation as well as anterograde amnesia (that is, inability to recall events or ‘lay down memories’ after injury) in the face of relatively intact attentional capacity.

Specific attention has been given to disturbances of memory following cerebrovascular disturbance of the anterior communicating artery. Disturbance ranging from global amnesia and severe confabulation to more discrete dysfunction in temporal ordering and recall have been reported. In discrete aneurysms of the anterior communicating artery two specific features of memory disturbance have been noted—susceptibility to interference and impaired temporal discrimination resulting, respectively, in disturbance in the contents of recalled information, and confusion in the ordering of facts or events. It is, however, unclear as to whether these cognitive disturbances reflect a compromising of frontal lobe function or ultimately reflect damage to underlying limbic structures.

In addition to the above, cerebrovascular disturbance of the anterior cerebral and anterior communicating artery may disrupt executive functions such as reasoning and abstraction skills, planning and monitoring of problem solving and flexibility of cognitive processing. Such disturbances reflect themselves in deficient performance across a wide range of procedures, such as the revised Wechsler Intelligence and Memory Scales, and tests sensitive to frontal lobe function such as the Wisconsin Card Sorting Test, Halstead Category Test and Trail Making Tests.

It may be noted here that certain of the observed disturbances associated with anterior artery CVA, such as aphasic and amnesic difficulties, pose certain theoretical problems in that the neural centres involved are not ones considered to be vital for expressive speech or memory function. Recently, however, it has been argued that disruption of such skills may not reflect focal disturbance of key processing sites responsible for such skills, but rather that these difficulties may arise as a consequence of disruption to over-riding executive or ‘supervisory’ systems which monitor and regulate these specific processes.

Disturbance of the middle cerebral arteries may produce a more diffuse and widespread pattern of cognitive disturbance by virtue of their widespread distribution to the lateral aspects of the frontal, occipital, parietal and temporal lobes, as well as to important subcortical sites such as the thalamus and internal capsule. Dysphasia appears to be a common sequelae, as do sensory disturbance and motor weakness. The precise nature of language disturbance reflects the site of the lesion. Thus disruption of the frontal branches may result in Broca’s or non-fluent aphasia, while receptive or fluent aphasia arises from disturbance of the temporal and parietotemporal branches. Disturbances in spatial orientation and recognition may additionally arise, and constructional apraxia
is a common effect. There has been surprisingly little work on memory disturbance following impaired flow in this artery, although confusional states and disturbance in selective attention have been observed.

Finally, disruption of the posterior arterial system may affect temporal and occipital lobe function in addition to having diverse effects within the midbrain.

The subcortical effects arising from such lesions include sensory disturbance, homonymous hemianopia and certain extrapyramidal disturbances.

Cortically, temporally centralised lesions may result in receptive dysphasia and disturbances in short-term memory. Extreme conditions such as cortical blindness may result from bilateral disturbance of the occipital lobes and smaller lesions arising from posterior disruption result in specific conditions such as alexia (i.e. word blindness).

Although by no means exhaustive, the above review points to the possible specificity of neuropsychological impairment as a function of disturbance of particular arterial flow. This raises the possibility that such assessment, in certain cases, may be profitably used as a valid adjunct to diagnosis.

**Dementia**

‘Dementia’ is a term applied to many brain disorders with variable clinical manifestations and aetiology. The term can often be abused to describe any condition in which there is progressive and at some stage pervasive loss of higher cognitive function which is deemed irreversible.

In reality the dementias may be subdivided into those which are primary and due to direct degenerative changes within the cerebrum, and those which are secondary and associated with either systemic or other neurological conditions. Thus we must discriminate between alcoholic dementia, multi-infarct dementia, normal-pressure hydrocephalus, Alzheimer’s disease, post-traumatic dementias, and those produced as a consequence of drug toxicity.

The ensuing review largely concentrates on those forms of dementia in which neuropsychological assessment is most commonly used.

**Alzheimer’s disease**

The most frequent discriminative issues surround Alzheimer’s disease, which, neuropsychologically, is characterised by a progressive deterioration in certain features of intellectual ability, particularly reasoning, inferential skills and conceptual and abstract thinking (assessed, for example, by the Similarities, Comprehension, Picture Arrangement, Block Design, Object Assembly and Arithmetic Scales of the revised Wechsler Adult Intelligence Scale and by the Raven’s Progressive Matrices).

In addition the disease is characterised by a disturbance of short-term memory predominantly associated with poor retention of semantic information as demonstrated by rapid forgetting on verbal recall tasks (see Kapur, 1988). Such patients perform particularly poorly on the delayed recall components
of such tests and they exhibit significant difficulties in new learning ability while retaining long-term memory and well learned skills. Fragmentation of personality, which is reflected in poor self-care and flattening of affective style, may also be noticed.

Clearly, the progressive nature of the disorder suggests that the neuropsychological discrimination of Alzheimer’s disease should be determined by repeated assessment, a few months apart, rather than by the interpretation of a single administration. Since well learned abilities and skills are relatively intact until the later stages, discrimination relies on subtle and careful administration of appropriate memory tests rather than on gross assessment. Simplistic batteries such as the Kendrick tests of dementia which rely on the administration of a simple psychomotor test and an immediate visual memory task are likely to be clinically misleading.

**Pseudodementia**

Neuropsychologists are often required to discriminate between dementia and depression or depressive pseudodementia. There is a close similarity of symptoms between depressive pseudodementia (behavioural and cognitive disturbances resembling those of degenerative dementia but attributable to functional disturbance) and the dementias of neurological origin, such that differential diagnosis is extremely difficult in the early stages of their clinical course. Clearly, however, such a distinction is of particular importance if it is the case that disorders such as depression, for which there are effective interventions, go untreated because of misdiagnosis. This is especially important since estimates of treatable, functional ‘pseudodementias’ may be 10–40% of elderly patients referred for diagnosis. From the neuropsychological viewpoint, it is tempting to use simple procedures to instigate this process. It should be borne in mind, however, that depressed subjects may underperform on both components of the Kendrick battery by virtue of psychomotor retardation and disturbance of spatial short-term memory.

More comprehensive tests do suggest that it may be possible to discriminate between these two processes. Thus it has been suggested that dementia and pseudodementia may be discriminated on tests of attentional span, semantic recall and, in particular, the rate of forgetting of learned information. The difficulty with such studies is that they are retrospective, that is, tests have been used on patients who have been confidently diagnosed by clinical means. There have been few studies which are either prospective or longitudinal, or which have assessed the sensitivity of neuropsychological tests to early cognitive changes in dementia.

One such study, by Jones et al (1992), does report neuropsychological data on patients referred for evaluation of suspected dementia and in which assessment took place over two periods separated by six months. Predictably, at the second test, neuropsychological data significantly discriminated between dementia and pseudodementia. However, this ability was at a stage when
clinical examination was confidently able to diagnose. Furthermore, at the first tests, 73% of subjects diagnosed as dementing on clinical grounds retained this diagnosis after six months. The significant predictors of dementia were poor performance on a Temporal Orientation Questionnaire (assessing knowledge of the day, date, month, year and time), poor Block Design performance on the revised Wechsler Adult Intelligence Scale (poor visuoconstructive ability) and poor performance on a spatial short-term memory test (reduced ability to reproduce designs from memory). Those subjects receiving an eventual diagnosis of dementia gave an impaired performance, while those confirmed as having pseudodementia performed within normal limits. This latter finding substantiates the view made earlier in this chapter, that while depressed subjects may under-perform on specific tests of cognitive function, their performance is not within organic limits.

While studies such as this provide evidence that neuropsychological test data may be sensitive to early cognitive disturbance in dementia and hence useful in early diagnosis, it remains to be seen whether neuropsychological results are any more sensitive than clinical examination. Clearly, however, there is the possibility that diagnosis achieved by conjoint assessment may be more sensitive than either used in isolation.

Alzheimer’s disease is most characterised by early changes in memory function, with more global deficits emerging later. Discriminating between this and, for example, multi-infarct dementia and normal-pressure hydrocephalus cannot necessarily be achieved by neuropsychological assessment alone, and this emphasises again that cognitive testing cannot be seen to be a ‘stand-alone’ system.

**Multi-infarct dementia and normal-pressure hydrocephalus**

Thus some of the characteristics of multi-infarct dementia are an abrupt disturbance of gait associated with motor slowness and dysarthria (Walsh, 1985), and clearly the determination of the presence of multiple sites of infarction more exclusively lies within the realm of anatomical and blood flow investigations.

Similar disturbances may be associated with normal-pressure hydrocephalus and in many instances the distinguishing factors are their neurological presentation, speed, and age at onset.

**Localisation**

In principle, it may be possible to determine the difference between global deficits resulting from generalised damage to the CNS and multiple sites of focal disturbance. Thus if we accept the premise of localisation of cognitive function within the caveats set out at the beginning of this chapter it should be possible ‘geographically’ to map the brain in terms of its function.
Inferentially it should then be possible, given the precise pattern of test results, to determine whether there is a global deficit in adjacent areas of neuronal tissue or whether functional disturbance is multifocal.

In practice, however, parsimony dictates that the utility of neuropsychological testing in isolation must be rather limited, and diagnosis must ultimately be a multidisciplinary process.

**Toxic and deficiency syndromes**

Most attention has been given to the neuropsychological consequences of Korsakoff's psychosis (see Chapter 6, pp. 207, 209). While attributable to a number of causes, frequent attention has been given to its genesis via the combined effects of chronic alcohol misuse and vitamin deficiency. Particular attention has been given to the effects this syndrome has in terms of memory function, and the evidence is consistent with a specific pattern of retained and disrupted abilities. Thus, in Korsakoff's psychosis, memory impairment is usually prominent on tests which exceed the individual's immediate memory span (i.e. tests in which the number of definable memory units is greater than the amount of information which can be held within working memory) or if the individual is required to retain information while engaged in distractor tasks. Overall, the deficits in memory may be summarised as a profound difficulty in acquiring new information (anterograde amnesia) in the face of normal or preserved immediate concentration and attentional processes. Many aspects of learned or skilled behaviour are preserved, but recent recall of events is often characterised by confabulation (i.e. the filling in of subjective memory gaps with extraneous information).

On tests such as the revised Wechsler Memory Scale, a sufferer's performance is typically characterised by good performance on tests of immediate concentration but with uniform deficits in immediate and delayed recall for both verbal and spatial information and by retarded learning and rapid forgetting of new information.

By contrast, for example, chronic alcohol abuse resulting in encephalopathy is associated with a general lowering of all aspects of short-term memory, but with particular deficits in visual memory as opposed to verbal recall (Parsons & Farr, 1981).

**Neuropsychological rehabilitation**

Most professionals associate the term 'neuropsychological rehabilitation' with traumatic CNS injuries such as may be incurred from a road traffic accident. There is however a role for 'neurotherapy' across a variety of CNS disturbances such as might arise from CVA, cerebral infection and degenerating conditions, and it may be argued that it also has a role for those experiencing cognitive difficulties as a consequence of functional illness.
Furthermore, neuropsychological rehabilitation is often seen as being synonymous with cognitive rehabilitation, when the latter is best seen as one element of the former. Thus neuropsychological rehabilitation may be seen to comprise the following:

(1) Informational care - the provision of information to enable the client and carers and relatives to understand and gain insight into their cognitive, behavioural and emotional difficulties. A key component of this aspect of care is that it should seek to facilitate understanding while also allaying anxieties and dispelling misconceptions.

(2) Psychotherapeutic support - to monitor and alleviate emotional suffering with good practice aimed at preventing further emotional difficulties. In many instances, psychotherapy is aimed at helping the individual work through and come to terms with the traumatic events which form the onset of the individual's current difficulties.

(3) Cognitive rehabilitation - specific procedural approaches aimed at the remediation of cognitive dysfunction, such as memory retraining.

(4) Social and vocational skills training - aimed at reintegration into the social milieu and worthwhile employment.

It would be fair to say that cognitive rehabilitation most intimately reflects the specialist skills of the neuropsychologist and relies heavily upon psychometric, qualitative and ecological assessment.

Less than ten years ago it was generally considered that following traumatic CNS injury any residual brain damage evident beyond the period of spontaneous recovery was permanent. It is now more readily acknowledged that cognitive rehabilitation may play a vital role in the restoration or remediation of cognitive disturbance following brain damage, both in respect of facilitating the potential for restoration during the period of spontaneous recovery and in facilitating reorganisation of brain function beyond this phase.

Current cognitive rehabilitation comprises three approaches, which may be described as: the use of compensation to enable the client to minimise or work round deficits; substitution, whereby alternative methods are used to solve cognitively mediated problems; and retraining methods to stimulate specific impaired functions. In many respects, comprehensive rehabilitation programmes may draw from all three approaches. The terms 'compensation' and 'substitution' may be taken to imply procedural or environmental manipulations and prostheses that may ameliorate the magnitude of cognitive deficit, and at one level this is certainly the case. There is however a higher-order meaning to these approaches which derives from theoretical neuropsychology, in which it is argued that an individual's deficits may be ameliorated by functional reorganisation. This concept is based upon the premise of localisation of function - that specific abilities are carried out by (or are manifestations of) the action of task-specific cortical and subcortical sites. When these 'sites' are damaged, the programmes of cognitive function
(like cerebral software) may be effectively transferred to adjacent areas of brain
tissue. This view accounts for the observation in head-injury sufferers with
definable brain damage of a gradual (though not necessarily total) restoration
of function over time despite the fact that the key processing sites have been
destroyed.

While this is an attractive hypothesis, there is little direct evidence to support
it, and an equally plausible hypothesis is one which rests on a networked
view of cognitive function whereby restoration of function arises from the fact
that complex and higher-order abilities are not carried out by single processing
sites.

Despite the theoretical dispute over the mechanisms of reorganisation, there
is ample evidence that restoration can be facilitated by the use of specific
procedures and learning techniques, in which the individual is taught to use
alternative functional skills and processes to carry out cognitive operations
which are dysfunctional. A general example of this would be to teach an
individual who experiences verbal short-term memory deficits to analyse,
translate and encode information on the basis of its visuospatial properties,
and hence to facilitate the use of cognitive strengths to ‘bypass’ a cognitive
weakness.

To achieve such an aim ultimately requires detailed and highly specific
neuropsychological assessment, whereby the aim of testing is to gain as full
an understanding as is possible of the individual’s cognitive strengths and
weaknesses, and as discrete as possible an understanding of the elements of
processing which are dysfunctional. This ‘process-specific’ approach to
rehabilitation relies on a hierarchical modelling of cognitive function (such
as that expounded by Luria (1966)), in which it may be assumed that large-

scale abilities such as reasoning, short-term memory, and so on can be divided
into smaller processing modules. The focus of rehabilitation then becomes
these identified dysfunctional modules (in a sense weak microlinks in a
cognitive chain) rather than more global functions, and it is both arguable
and demonstrable that this type of modelling and approach to rehabilitation
is more successful and powerful than more general approaches.

Such a ‘process-specific’ approach to both assessment and treatment is
considered by some to be one which is largely independent of other types
of investigation, such as CT and magnetic resonance imaging, although clearly
one should never seek for total independence, since in neuropsychological
rehabilitation the assessment of dysfunction still needs to be carried out with
an understanding of the biological mechanisms leading to it, and these can
only be ascertained by other means.

Conclusions

This chapter has highlighted some of the clinical uses of assessment, rather
than attempting to condense a vast area into such a small space. Inevitably
important topics have not been covered or have been sacrificed in order to provide a relatively clear description of the broad range of the specialty. Those requiring convincing evidence of the power and application of neuropsychological assessment across a variety of clinical settings can refer to Incagnoli et al (1986). The aim in this chapter has not been to suggest that neuropsychological assessment is an infallible tool.

Neuropsychological assessment is a time-consuming process which requires on the part of the practitioner an all-round knowledge of clinical psychology, psychological test theory and practice, a working knowledge of neuropsychology, and familiarity with neurosciences such as neurology, neuroanatomy, neuropathology and, in some settings, neurosurgery. Indeed, the findings of neuropsychological assessment can only be interpreted reliably in the light of evidence from all of these.

Clearly, neuropsychology is open to abuse: there may be a temptation to derive seductive inferences from minimal test data, and the interpretation of neuropsychological profiles is one which is open to frank misinterpretation. But in skilled hands, cognitive assessment is a powerful process which can be applied across a range of clinical settings. In some instances the neuropsychologist’s role is one of contributing a database for diagnosis, and this may be seen in the same light as requests for CT scans or electroencephalography.

Neuropsychological assessment has particular importance because of its ability to describe the functional strengths and weaknesses of the individual. This allows crucial rehabilitation strategies to be devised and coordinated, with the aim of ameliorating the effects of brain injury and impaired cognitive processing.

In all of its applications, the strength of neuropsychological assessment is its quantification of the cognitive correlates of structural alterations to the CNS, but one must always view such assessment as part of an integrated approach to diagnosis and treatment, incorporating all of the relevant neurological disciplines.

Appendix 1. Neuropsychological tests in common practice

**Intellectual function**

*Wechsler Adult Intelligence Scale - Revised*

**Areas assessed** Establishes current IQ by the assessment of general information, verbal and non-verbal reasoning, vocabulary, comprehension, auditory concentration, arithmetic, constructional ability and visual-verbal integration.

**Methods** Comprises 11 subtests predominantly in the form of question and answer for the verbal items. For non-verbal items instructions are given verbally, the answers comprising either the construction of three- and two-dimensional objects, rearrangement of visually presented story pictograms, or drawing.
Memory function

**Wechsler Memory Scale - Revised**

*Areas assessed* Establishes broad memory function by assessment of rote memory, orientation, recognition, concentration, verbal and non-verbal short-term memory, verbal and non-verbal learning ability.

*Methods* Consists of a simple orientation questionnaire, drawing of designs from memory, verbal recall of stories, learning of new word pairs and of pairs of patterns.

**Adult Memory and Information Processing Battery**

*Areas assessed* Assesses immediate and delayed verbal and non-verbal memory, verbal and non-verbal learning, cognitive processing speed, motor speed.

*Methods* Similar materials to the above scale. The test comprises recall of designs and stories, learning of word lists and of a complex design, cancellation tasks, and a simple motor speed task.

**Warrington Recognition Memory Test**

*Areas assessed* Forced-choice recognition of verbal (words) and spatial (faces) material.

*Methods* Subjects view 50 words and 50 faces. For both sets of stimuli the subject is then shown 50 word pairs or face pairs - the aim being to pick out the original words and faces.

**Rey Auditory Verbal Learning Test**

*Areas assessed* Assesses rate of learning and degree of forgetting, susceptibility to interference, primacy and recency.

*Methods* Subjects learn a list of words over a number of trials. They then have to learn a new list and then finally recall the original test.

**Rey-Osterreith Figure**

*Areas assessed* Test of immediate and delayed recall of complex spatial material. Also assesses planning and constructional ability.

*Methods* Subjects first copy a complex design, then attempt to draw it from memory.

Language function

**Boston Diagnostic Aphasia Examination**

*Areas assessed* Wide-ranging assessment of receptive and expressive language. Encapsulates interpretational profiles associated with the major aphasic syndromes.
Methods Multiple assessment method involving drawing, writing, spelling and verbal responses to a range of stimuli.

Revised Token Test

Areas assessed Pure assessment of receptive language skills. The use of a motor response makes this a useful tool with individuals with expressive language difficulties.

Methods Subjects carry out motor actions to verbal commands. 'Tokens' of different size, shape and colour are moved in response to commands of increasing difficulty.

Benton Verbal Fluency Test

Areas assessed A simple procedure assessing fluency and word-finding ability.

Methods Subjects are required to say as many words as they can beginning with specific letters of the alphabet in one minute.

Information processing tasks

These are often used to screen for organic impairment.

Stroop Colour-Word Interference Test

Areas assessed Speed of processing for simple verbal and non-verbal information. Also assesses cognitive flexibility.

Methods Comprises three components: (1) reading out rapidly an array of 100 words made up of a random presentation of the words RED, GREEN, BLUE; (2) reading out the colour hues RED, GREEN and BLUE; (3) the words RED, GREEN and BLUE are written in coloured ink such that colour hue does not match the colour word. Subjects read out the colour hue only.

Trail Making Test

Areas assessed See Appendix 2, p. 183.

Methods See Appendix 2, p. 183.

Visual Search Test

Areas assessed Assesses pattern recognition, visual scanning, speed of visuospatial processing.

Methods Subjects pick out a pattern from an array which correctly matches a central pattern.
Paced Auditory Serial Addition Test

*Areas assessed* Assess attentional processes, monitoring, tracking and sequencing, speed of computational processing.

*Methods* Subjects listen to a series of numbers presented at defined time intervals and have to add up successive pairs.

**Tests of laterality**

*Annett's Handedness Questionnaire*

*Areas assessed* Assesses handedness by the miming of actions.

*Methods* Involves miming the actions (e.g. writing, striking a match).

*Harris Test of Lateral Dominance*

*Areas assessed* Assesses handedness, eyedness and footedness.

*Methods* Involves miming a variety of actions.

**Dichotic Listening Tests**

*Areas assessed* Experimental procedures which assess auditory perceptual asymmetries for verbal and non-verbal material from which inferences of cerebral dominance can be made.

*Methods* Subjects are auditorily presented with groups of word pairs which are simultaneous and presented one word to each ear. After each group, subjects recall as many words as they heard.

**Neurological tests**

*Finger Oscillation*

*Areas assessed* Dominant and non-dominant hand motor speed.

*Methods* Involves the rapid depression of a manual counter with the right or left index finger, to determine the number of taps within 10 seconds. Scores are rated for each hand over several trials.

*Finger Tip Number Writing*

*Areas assessed* Finger pad graphesthesia.

*Methods* The examiner inscribes single digits on each finger pad while the examinee tries to identify the number written.
Finger Agnosia

Areas assessed Somatosensory recognition and naming.

Methods Subjects name which finger the examiner has touched.

Right-Left Orientation

Areas assessed Inter- and extra-personal spatial disorientation.

Methods Involves the execution of pointing or touching actions on command (e.g. ‘touch your right shoulder’; ‘point to my left hand with your right hand’).

Double Simultaneous Stimulation

Areas assessed Auditory, visual and manual suppressions.

Methods Unilateral or bilateral presentation of simple auditory, tactile or visual stimulus.

Appendix 2. The Halstead-Reitan Battery

This takes five to eight hours or more to administer. The asterisked items are useful independently for screening for organic impairment.

Wechsler Adult Intelligence Scale (revised)

Measures Intellectual function.

Method See Appendix 1.

*Category Test

Measures Hypothesis formation, rule learning, planning.

Method Subjects view a series of stimulus cards each with four definable patterns. For each card the aim is to find the odd man out by determining rules about similar and dissimilar features across the four patterns.

Speech Sounds Perception Test

Measures Phoneme discrimination, attention.

Method Subjects listen to 60 nonsense words. For each word they pick out what they think they have heard from a visual array of four options.
Seashore Rhythms Test

Measures  Rhythm and pitch discrimination, attention.

Method  Subjects listen to 30 pairs of tone sequences. For each pair they determine if the sequences are identical or different.

*Trail Making Test

Measures  Visual scanning, visuomotor coordination, psychomotor ability, sequencing, category alternation.

Method  Trail A involves drawing a single line sequentially connecting the numbers 1 to 25 (presented in random visual order). Trail B involves connecting numbers and letters in ascending but alternating fashion (1-A-2-B-3 etc.).

Finger Oscillation Test

Measures  Dominant and non-dominant hand motor speed.

Method  See Appendix 1.

Tactual Performance Task

Measures  Astereognosis, spatial memory, spatial learning, orientation, uni- and bimanual function.

Method  Subjects, while blindfold, place three-dimensional objects into their matching cut-outs. Trial 1 uses the right hand only; Trial 2 uses the left hand only; and Trial 3 is a bimanual trial. Following this, subjects have to draw from memory the shapes and their location.

Sensory Perceptual Examination

Measures  Finger agnosia, finger tip number writing, visual, auditory and manual suppression, astereognosis.

Method  See Appendix 1.

Aphasia Screen

Measures  Broad but brief assessment of language comprehension and production.

Method  Short standard aphasia screen including drawing of designs, writing to dictation, word repetition, spelling, etc.
Minnesota Multiphasic Personality Inventory

Measures  Personality.

Method  A lengthy questionnaire in which subjects respond ‘true’ or ‘false’ to each question.

Appendix 3. The Standardised Luria-Nebraska Battery

The LNNB is almost entirely in a ‘pencil and paper’ format. Its administration involves question and answer, drawing, copying and execution of motor movements, reading words and text, completion of two-dimensional puzzles, and so on. All scores are conveniently recorded in a single booklet and are then either analysed manually or by computer.

There are 279 test items in total, and the battery takes on average two to four hours to complete.

Test components

Clinical scales

These scales are empirically derived from the test items.

Motor
Rhythm
Tactile
Visual
Receptive speech
Expressive speech
Reading
Writing
Arithmetic
Memory
Intellectual Processes
Intermediate memory

Subsidiary scales

These profiles and scales are derived from a reordering of the original test items.

Pathognomic
Right hemisphere
Left hemisphere
Profile elevation
Impairment
Power
Speed
Interpretational profiles

These profiles and scales are derived from a reordering of the original test items.

Factor items
Localisation
Analysis of test items

References


