B

Babcock, Harriet (1807–1952)

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Major Appointments

- Manhattan State Hospital, New York, 1923–1925
- New York University, New York, 1931–1952

Major Honors and Awards

- Babcock was elected to the New York Academy of Science and was a Diplomate of the American Board of Examiners in Professional Psychology.

Landmark Clinical, Scientific, and Professional Contributions

- In the 1930s, Babcock began a longitudinal study of syphilitic patients, a project that was less notable for its outcomes (many of which were not subsequently replicated) than for its methodology. Classic neurological studies from the time of Paul Broca and Karl Wernicke were centered around clinical case observation. In a departure from this classic tradition, Babcock adopted the methods of scientific psychology to study the cognitive effects of neurological disease. Her research methods were well-characterized and repeatable, she utilized standardized psychometric measures, and she incorporated normal control comparison groups in her research. Anticipating later batteries of neuropsychological tests, Babcock attempted to quantify deficits in discrete mental abilities and used an "efficiency index" to summarize the overall functioning of her patients.
- Babcock based her efficiency index on the idea that intellectual function varies over time. More specifically, people may exhibit a higher level of intellectual function while healthy and in the prime of life, than they do after suffering neurological or psychiatric disorders. Babcock believed that mental disorders do not affect tests of prior knowledge acquisition (e.g., vocabulary). She further identified a number of tests that she thought were sensitive to mental disorders, including tests familiar to contemporary neuropsychologists (e.g., reverse digit span and various reasoning tasks). Babcock quantified mental efficiency by contrasting performance on these two kinds of tests, a forerunner of the hold–don't hold test comparison (▶ Hold–Don't Hold Tests).
- Babcock's contemporary influence is also evident in her story memory format. In this format, a story is initially presented and recall is tested. The story is presented a second time followed by 10 min of interpolated activity and a final recall test. This format has been adopted in some contemporary memory batteries and has the advantage of allowing the neuropsychologist to test both immediate and delayed recall, as well as learning with repetition. Although the original Babcock Story is rarely used today, some current memory batteries incorporate its format (▶ Wechsler Memory Scale). In this and other respects, Babcock's work continues to influence clinical and scientific neuropsychology.

Short Biography

Little has been written concerning Babcock's personal life. She was born in 1877 in Westerly, Rhode Island. She began her career late in life, earning her doctoral degree in her 50s. Prior to this, she lived a traditional life as a homemaker. She initially gained experience working in psychiatric facilities, but after earning her doctorate, she spent the balance of her career on the faculty at New York University. Despite her late beginning, Babcock's work was an important forerunner to the emergence of neuropsychology as a scientific field. Babcock died on December 12, 1952.

Cross References

▶ Hold–Don't Hold Tests
▶ Intelligence

Jeffrey S. Kreutzer, John DeLuca, Bruce Caplan (eds.), *Encyclopedia of Clinical Neuropsychology*, DOI 10.1007/978-0-387-79948-3,

► Intelligence Quotient
► Wechsler Memory Scale

References and Readings

Hartman, D. E. (1991). Reply to reitan: Unexamined premises and the evolution of clinical neuropsychology. *Archives of Clinical Neuropsychology, 6*, 147–165.

Stringer, A. Y., & Cooley, E. L. (2002). Neuropsychology: A twentieth-century science. In A. Y. Stringer, E. L. Cooley, & A.-L. Christensen (Eds.), *Pathways to prominence in neuropsychology: Reflections of twentieth century pioneers* (pp. 3–26). New York: Psychology Press.

Babinksi Reflex

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Synonyms

Long tract sign; Plantar reflex; Upper motor neuron sign

Definition

The Babinksi reflex or sign is elicited by making contact along the lateral side of the plantar foot with a blunt implement and not causing pain, discomfort or injury to the skin; the implement is run from the heel along a curve to the metatarsal pads. There are three responses possible:

- Extensor (positive or pathological): hallux (great toe) extension and the other toes abduct
- Flexor (negative or normal): all toes flex and the foot everts
- Indifferent: no response

Current Knowledge

A positive response signifies pathology in the upper motor neuron pathways, either in the spinal cord and/or brain, such as in multiple sclerosis, stroke, traumatic brain injury, or spinal cord injury. It may be the sole sign of upper motor neuron damage and is the most popular reflex that checks these pathways for the lower limbs. All infants exhibit an extensor response from birth, which converts to a flexor response during ages 12–18 months given normal development; developmental delay may result in a persistent positive response. Indifferent responses may be found in normal individuals but may also indicate the presence of a lower motor neuron or other peripheral nervous system injury that interferes with the expression of a flexor response.

Cross References

► Developmental Delay
► Multiple Sclerosis
► Spinal Cord Injury
► Stroke
► Traumatic Brain Injury

References and Readings

Babinski, J. (1896). Sur le reflexe cutane plantaire dans certaines affections organiques du systeme nerveux central. *Comptes rendus des séances de la Société de biologie et de ses filiales, 48*, 207–208.

Campbell, W. W. (2005). *DeJong's the neurological examination* (pp. 324, 331, 339). Philadelphia: Lippincott, Williams & Wilkins.

Getz, C. (1985). Introduction to the motor systems. In E. R. Kandel & J. H. Schwartz (Ed.), *Principles of neural science* (pp. 429–442). New York: Elsevier.

Landau, W. M., & Clare, M. H. (1959). The plantar reflex in man, with special reference to some conditions where the extensor response is unexpectedly absent. *Brain, 82*, 321–335.

Background Information

► History (Medical, Social, Psychological)

Backward Digit Task

► Digit Span

Backward Masking

► Temporal Inhibition

BADS

► Behavioral Assessment of the Dysexecutive Syndrome

BAER

► Brainstem Auditory Evoked Responses

BAI

► Beck Anxiety Inventory

Balance Disorders

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Definition

Normal balance requires the integration of three sensory systems: visual, vestibular (found in the inner ear), and somatosensory (sensations from the skin, muscles, tendons, and joints) – in addition to muscle strength. When these systems are impaired, individuals may experience episodes of spinning, light-headedness, trouble focusing their eyes, and/or poor balance or falls.

Categorization

Balance may be affected by disturbances of strength in the trunk or legs, sensation deficits, or difficulties with coordination. Multiple systems may be affected. A detailed history and neurological examination may help detect the affected area. Balance may be impaired after a focal event such as a stroke or may develop during the course of a neurodegenerative disease such as Parkinson's disease. Medications and infections of the brain or inner ear may also contribute to balance difficulties.

Epidemiology

Aging may also affect balance. Approximately 40% of people older than age 65 suffer falls each year. Vertigo is the most common form of dizziness.

Natural History

Balance disorders associated with neurodegenerative diseases tend to be progressive.

Neuropsychology and Psychology of Balance

Neurodegenerative disorders associated with balance that affect the cortex can also be associated with cognitive difficulties.

Evaluation

The history and physical examination often lead to a diagnosis. At times, laboratory tests and imaging are obtained for confirmation or to rule out harmful diagnoses. If a reversible cause is found and treated, significant recovery may occur. However, if the balance problem is due to a permanent or progressive neurological deficit, the patient may need training to manage their gait and balance difficulties.

Treatment

Physical therapy and vestibular rehabilitation may be useful in appropriate cases. They may improve current functioning and potentially decrease the potential for progression of deficits and complications from falls.

Cross References

► Ataxia
► Parkinson's Disease

References and Readings

Ackley, S., Newell Decker, T., & Limb, C. J. (2007). *An essential guide to hearing and balance disorders.* Psychology Press.

Balint, R. (Rezso (Rudolf) Balint) (1874–1929)

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Major Appointments

- University of Budapest, Budapest, Hungary, 1910–1929

Major Honors and Awards

- Balint's work was honored by the later naming of his "triple-syndrome complex" as "Balint's Syndrome" by Hecaen and Ajuriaguerra (Hecaen & Ajuriaguerra, 1954).

Landmark Clinical, Scientific, and Professional Contributions

- Hungarian physician Rezso Balint's first writings, published while he was still a medical student, were case studies examining muscular atrophy in hemiplegia. He went on to study tabes dorsalis and the treatment of epilepsy. In 1907, Dr. Balint recorded his observations of a patient who suffered from a unique constellation of neurologic symptoms including fixation of gaze, neglect of objects in his periphery, and misreaching for target objects. The patient was noted to first experience these symptoms following damage to the posterior parietal lobes. This "triple-syndrome complex" was later named "Balint's Syndrome."

Short Biography

Rezso Balint was born in 1874 to a German-Jewish family in Budapest, Hungary. He attended the University of Budapest, where he received his degree in medicine in 1897. Balint was a student of Friedrich von Koranyi. He was employed as a Lecturer at the University of Budapest in 1910 and was promoted to Professor of Internal Medicine in 1917.

At the onset of World War I, Dr. Balint turned his research focus from neurology to tuberculosis and metabolism and the treatment of diabetes. He is most well-known in his home country of Hungary for the treatment of gastric ulcer with the use of alkali.

Rezso Balint died of thyroid cancer in 1929 at the age of 56.

Cross References

► Neglect
► Optic Ataxia

References and Readings

Hecaen, H., & Ajuriaguerra, J. (1954). Balint Syndrome (psychic paralysis of visual fixation) and its minor forms. *Brain, 77*, 373–400.
Husain, M., & Stein, J. (1988). Reszo Balint and his most celebrated case. *Archives of Neurology, 45*, 89–93.
Rezso Balint (physician). From Wikipedia: http://en.wikipedia.org/wiki/rezs. Retrieved January 7, 2009.

Balint's Syndrome

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Short Description or Definition

Balint's syndrome was first described by Rezső Bálint in 1909. It consists of three visuospatial abnormalities: simultanagnosia, optic ataxia, and ocular motor apraxia. The syndrome typically occurs in the absence of visual field deficits. Individuals with Balint's syndrome experience significant perceptual limitations. Patients with this syndrome cannot perceive more than one object at a time. They experience great impairments in their ability to explore visual space: they have difficulty navigating through their environment, they get lost easily, and they experience difficulty reaching for or grasping items in need.

Balint's syndrome is usually associated with large bilateral lesions in the dorsal occipitoparietal region, and is consequently rare. The most common causes of Balint's syndrome include ischemia (particularly watershed infarctions) and degenerative disorders (e.g., Alzheimer's disease, posterior cortical atrophy). Balint's syndrome can also result from trauma, tumors, leukoencephalopathies, and

prion disorders. In individuals with HIV-AIDS, Balint's syndrome can develop secondary to HIV encephalitis or progressive multifocal leukoencephalopathy. Transient symptoms of Balint's syndrome have been reported in association with migraine onset.

Natural History, Prognostic Factors, Outcomes

The prognosis for patients with Balint's syndrome varies depending on the etiology of the syndrome. Patients with posterior cortical atrophy usually experience a declining course, while some patients with acute infarction may demonstrate improved functioning with time.

Neuropsychology and Psychology of Balint's Syndrome

As noted above, individuals with Balint's syndrome display three classic symptoms, including simultanagnosia, optic ataxia, and ocular motor apraxia. Simultanagnosia is generally considered to be a disruption in spatial attention, which is associated with an inability to direct one's attention to more than one or a few objects at a time. It is not uncommon for patients with this syndrome to ignore or neglect all other objects once one object in the visual field has been fixated upon. Although patients can perceive and name individual objects regardless of the object's location within the visual field, they exhibit an inability to perceive and interpret the gestalt of the scene. The second symptom associated with Balint's syndrome is optic ataxia, which is defined as a deficit in reaching under visual guidance despite normal limb strength and position sense. As a result of this symptom, patients demonstrate an inability to manually respond to visual stimuli and they often make location errors when pointing to or grasping for visual targets. Some of the impairments noted on tests of reaching abilities include: increased action latency, poor control of hand trajectory, increased variability at the end of the reach, tendency to reach to one side, and dissociations of distance and direction control. The third symptom of Balint's syndrome includes ocular apraxia, which is manifested by an inability to voluntarily shift gaze toward a new visual target. The ability to make a saccade on command is significantly impaired and is next to impossible for patients with Balint's syndrome, whereas the ability to make reflexive saccades (e.g., those made to suddenly appearing visual objects or sudden noises) and random spontaneous saccades remains intact.

Evaluation

Before a diagnosis of Balint's syndrome can be made, more general cognitive dysfunction (e.g., hemineglect, visual impairments) should be ruled out. It is important that the patient's visual fields be assessed fully as some types of visual field abnormalities (e.g., extensive peripheral scotomata) can result in symptoms that are very similar to Balint's syndrome.

A typical method of assessing for simultanagnosia includes asking the patient to examine and describe the events depicted in a complex visual image (e.g., the Cookie Theft Picture from the Boston Diagnostic Aphasia Examination). In such a task, it is helpful if key elements of the image are presented in all four quadrants of the picture in order to assess visual attention more fully across the quadrants. Individuals with hemineglect may describe items on one side of the picture only. Patients with Balint's syndrome often are able to identify discrete items in the picture; however, they are frequently unable to integrate the various elements of the picture into a coherent story. Patients will show impairments on visual search and counting tasks. Letter identification and reading abilities may also be assessed for functional purposes.

In assessing for optic ataxia, one may place several items at different locations on a table and ask the patient to touch or grasp each of the items. It is important to assess whether the patient is able to grasp items within both hemifields with each hand independently. Patients with unilateral lesions typically demonstrate greater impairment when reaching for items located in the hemispace that is contralateral to the lesion, using the contralateral hand. Individuals with Balint's syndrome are impaired when reaching for visual targets in all locations within the visual field; however, some patients with Balint's syndrome may demonstrate reaching difficulties in which one arm is more affected than the other. Patients with this syndrome are noted to be clumsy when grasping items and they may often mislocate objects in space when reaching for or pointing to items. In contrast, reaching to somatosensory targets such as parts of the patient's own body (e.g., knee, shoulder) on command is frequently intact; however, patients with significant parietal spatial representation abnormalities may demonstrate impairments in both reaching for objects as well as reaching to somatosensory targets.

In assessing for ocular motor apraxia, the patient's ability to make saccadic eye movements to targets on command can be compared to their ability to make reflexive saccades to targets that appear suddenly in their field of vision. The former can be tested by asking the patient to saccade between the clinician's left and right

index fingers, spaced far apart and held at various locations across the patient's visual field. The latter can be tested in response to a person passing by or to a loud unexpected noise occurring in the periphery.

Treatment

Relatively little is known about treatment of patients with Balint's syndrome. Rehabilitation often utilizes a functional approach in which the patient's strengths are used to offset impairments. There is some evidence to suggest that cognitive and perceptual rehabilitation approaches using verbal cues and organizational search strategies can improve visual function and reaching abilities (Perez, Tunkel, Lachmann, & Nagler, 1996).

Cross References

- ▶ Hemiinattention
- ▶ Simultanagnosia
- ▶ Visual Field Deficit

References and Readings

Perez, F. M., Tunkel, R. S., Lachmann, E. A., & Nagler, W. (1996). Balint's syndrome arising from bilateral posterior cortical atrophy or infarction: Rehabilitation strategies and their limitation. *Disability and Rehabilitation, 18*, 300–304.

Barbiturates

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Definition

Barbiturates belong to a class of medications known as sedative-hypnotics. Initially, they were prescribed for their anxiolytic and relaxing properties. Later, they were also used as anticonvulsants, and shorter-acting forms were developed for use as anesthetics in surgery (Feldman, Meyer, & Quenzer, 1997).

Barbiturates affect a subtype of the receptors of the neurotransmitter, gamma-aminobutyric acid (GABA), one of the most common inhibitory neurotransmitter systems in the brain. Their behavioral effects include relaxation, drowsiness, and feelings of euphoria. However, their widespread effects also result in the depression of reflexes and cardiovascular and respiratory functions, particularly at higher doses (Feldman et al., 1997).

The psychoactive effects of barbiturates increase their risk for drug dependence and abuse. Symptoms of tolerance and withdrawal develop with chronic use; however, greater tolerance develops to the psychoactive effects, but less so to the respiratory depressant effects of barbiturates, resulting in an increased risk of a toxic overdose (Feldman et al., 1997). Cross tolerance with other substances may also occur. For example, alcohol use may also increase tolerance to barbiturates, further increasing the risk of a toxic overdose.

Current Knowledge

The use of barbiturates has declined significantly with the development of other anxiolytic and anticonvulsant medications. Benzodiazepines, which are also anxiolytic compounds that interact with the $GABA_A$ receptor (although a different site than barbiturates), have a larger therapeutic window than barbiturates and have replaced their use as a safer alternative for the treatment of anxiety.

Cross References

- ▶ Benzodiazepines
- ▶ Gamma-Aminobutyric Acid (GABA)

References and Readings

Feldman, R. S., Meyer, J. S., & Quenzer, L. F. (1997). *Sedative-hypnotic and anxiolytic drugs. In Principles of neuropsychoparhmacology* (pp. 673–729). Sunderland, MA: Sinauer.

Barefoot v. Estelle (1983)

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Synonyms

Prediction of future dangerousness

Historical Background

Thomas A. Barefoot burned down a bar and shot and killed a police officer who was investigating the arson. Barefoot was convicted by the jury of capital murder of a police officer. During the death penalty phase of the case, the state used psychiatric testimony to demonstrate that Barefoot posed a threat to society in the future. Specifically, the state had Drs. John Holbrook and James Grigson review a hypothetical fact situation based on evidence from the case and asked each of the doctors if the convicted individual would commit violent acts in the future or would pose a threat to society. Both doctors testified that the criminal would be a continued threat to society. In fact, Dr. Grigson concluded that there was a "one hundred percent and absolute" probability that Barefoot would commit violent acts in the future and thus pose a continued threat to society. The judge sentenced Thomas A. Barefoot to death. Barefoot appealed the decision and in the Court of Criminal Appeals raised several concerns about the way his trial was handled, most notably with respect to the probability that he would commit future violent acts. Barefoot argued that the psychiatrists testifying against him had not even examined him and were making determinations based on a hypothetical fact-based situation. Moreover, Barefoot called into question the ability of psychiatrists to predict future dangerousness. The Court of Criminal Appeals rejected all of Barefoot's arguments and the U.S. Supreme Court rejected Barefoot's suggestion that psychiatrists are not competent to make determinations regarding dangerousness in future. The U.S. Supreme Court ruled that psychiatrists are no less reliable than laypersons and that laypersons' testimony of future dangerousness is indeed permissible. The Court upheld that the use of hypothetical questions to establish future dangerousness is just because such testimony is supported by the Federal Rules of Evidence (FRE) that death penalty cases do not present special evidentiary problems. Furthermore, there is evidence (e.g., Monahan, 1992; Monohan & Steadman, 1994; Mossman, 1994) to suggest that mental health professionals do indeed predict violence significantly better than chance when "relevant" factors are included in the determination.

References and Readings

Denney, R. L. (2005). Criminal responsibility and other criminal forensic issues. In G. Larrabee (Ed.), *Forensic neuropsychology: A scientific approach.* New York: Oxford University Press.

Melton, G. B., Petrila, J., Poythress, N. G., & Slobogin, C. (1997). *Psychological evaluations for the courts: A handbook for mental health professionals and lawyers.* New York: Guilford.

Monahan, J. (1992). Mental disorder and violent behavior: Perceptions and evidence. *American Psychologist, 47*, 511–521.

Monahan, J., & Steadman, H. J. (1994). Toward a rejuvenation of risk assessment research. In J. Monahan, & H. J. Steadman (Eds.), *Violence and mental disorder: Developments in risk assessment.* Chicago: University of Chicago Press.

Mossman, D. (1994). Assessing predictors of violence: Being accurate about accuracy. *Journal of Consulting and Clinical Psychology, 62*, 783–792.

Barona Index

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Synonyms

Premorbid intelligence regression model

Definition

Barona Index is a demographically based regression method to estimate premorbid intelligence in terms of index scores on the Wechsler Adult Intelligence Scale-Revised (WAIS-R).

Historical Background

Attempts have long been made to estimate premorbid intellectual functioning. A frequent method in clinical practice is to estimate the level of premorbid cognitive skill by subjectively considering aspects of the individual's history such as education and occupation. Another common approach to estimate premorbid IQ is to use tests of present ability, which are thought to be relatively resistant to change even during the phases of a psychiatric disorder or those following a neurologically based disorder. A variant is the best performance method in which the highest score obtained by an individual is assumed to be the most likely premorbid level (▶ Best Performance Method). Research has been inconsistent as to the effectiveness of this approach. In an attempt to reduce the error in estimating intelligence based on current

functioning and eliminate the subjectivity inherent in clinical judgment, demographically based regression equations were created to statistically predict intelligence test scores. A later method of combining demographic information and current performance on IQ has also been found to be relatively effective.

It is well established that demographic variables, such as education, social class and education, are correlated with measured IQ. Wilson et al. (1978) created a regression equation to predict WAIS IQ from demographic variables. They used regression modeling with WAIS Full Scale IQ, Verbal IQ, and Performance IQ as criteria and age, education, sex, race, and occupation as predictors. With the development of the WAIS-Revised (WAIS-R), further models were needed to estimate premorbid intelligence. Baron, Reynolds, and Chastain (1984) generated demographic equations for the estimation of premorbid WAIS-R IQ. Subsequently, research demonstrated successful discrimination of neurologically based patients from non-neurologically based patients utilizing the WAIS-R. As demonstrated in Fig. 1, the predictor variables incorporated into the model included those originally utilized by Wilson et al. (1978) as well as urban/rural residency, geographic location, and handedness. Although these equations resulted in less IQ variance and larger standard errors of estimate, cross validation studies were successful.

Current Knowledge

Currently, premorbid estimation of IQ functioning includes the WAIS-III (Wechsler, 1997). Algorithms derived from the WAIS-III with demographic variables have been developed by the Oklahoma Premorbid Intelligence Estimate (OPIE-3). Four sets of algorithms using between one and four WAIS-III subtests have been devised. Research consistently suggests that OPIE-3 methods are reasonably effective in estimating premorbid intelligence.

Future Directions

As we are on the brink of the release and utilization of the Wechsler Adult Intelligence Scale-IV, it is quite likely that future regression models to estimate premorbid functioning as indexed by scores on this test will be developed. It is necessary to continue to improve our methods of estimating premorbid abilities. Future models will most

WAIS-R VIQ = 54.23 + .49 (age) + 1.92 (sex) + 4.24 (race) + 5.25 (education) + 1.89 (occupation) + 1.24 (U- R residence.)

Standard Error of Estimate = 11.79; R^2 = .38

WAIS-R PIQ = 61.58 + .31 (age) + 1.09 (sex) + 4.95 (race) + 3.75 (education) + 1.54 (occupation) + .82 (region)

Standard Error of Estimate = 13.23; R^2 = .24

WAIS-R PIQ = 54.96 + .47 (age) + 1.76 (sex) + 4.71 (race) + 5.02 (education) + 1.89 (occupation) + .59 (region)

Standard Error of Estimate = 12.14; R^2 = .36

Sex:	Female = 1, Male = 2
Race:	Black = 1, Other ethnicity = 2, White = 3
Education	0–7 years = 1, 8 = 2, 9–11 = 3, 12 = 4, 13–15 = 5, 16+ = 6
Age:	16–17 years = 1, 18–19 = 2, 20–24 = 3, 25–34 = 4, 35–44 = 5, 45–54 = 6, 55– 64 = 7, 65–69 = 8, 70–74 = 9
Region:	Southern = 1, North Central = 2, Western = 3, Northeastern = 4,
Residence:	Rural = 1, Urban = 2
Occupation:	Farm Laborers, Farm Foremen & Laborers (unskilled) = 1 Operatives, Service Workers, Farmers, & farm Managers (semiskilled) = 2 Not in Labor Force = 3 Craftsmen & Foremen (skilled workers) = 4 Managers, Officials, Proprietors, Clerical & Sales Workers = 5 Professional & Technical = 6

Barona Index. Figure 1 Barona et al. (1984) regression formulas for pre-morbid IQ

likely consider other variables and/or include more specific criteria for the existing models. For example, the expansion of technology along with fewer labor-based jobs will very likely influence the occupations used for the equation. As age expectancy increases, the role of age on premorbid functioning will quite likely become a more important variable as well.

Cross References

- ▶ Best Performance Method
- ▶ Intelligence
- ▶ Premorbid Estimate
- ▶ Premorbid Functioning

References and Readings

Barona, A., & Chastin, R. L. (1986). An improved estimate of premorbid IQ for black and whites on the WAIS-R. *International Journal of Clinical Neuropsychology, 8*, 169–173.

Barona, A., Reynolds, C. R., & Chastain, R. (1984). A demographically based index of premorbid intelligence for the WAIS-R. *Journal of Consulting and Clinical Psychology, 52*, 885–887.

Schoenberg, M. R., Scott, J. G., Duff, K., & Adams, R. L. (2002). Estimation of WAIS-III intelligence from combined performance and demographic variables: Development of the OPIE-3. *The Clinical Neuropsychologist, 16*, 426–438.

Wilson, R. S., Rosenbuam, G., Broan, G., Rourke, D., Whitman, D., & Grisell, J. (1978). An index of premorbid intelligence. *Journal of Consulting and Clinical Psychology, 46*, 1554–1555.

Barthel Index

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Synonyms

BI

Description

The Barthel Index (BI) measures ten functions that are important for independent living – feeding, bathing, grooming, dressing, bowel and bladder continence, toileting, transfers, mobility, and stair use. Items are weighted and scored according to their perceived importance. Higher scores indicate better performance. In the most commonly used version, the maximum score of 100 indicates full independence. Several versions of the Barthel Index and their associated scoring methods exist. (Shah et al. 1989) expanded the scoring categories to improve the scale discriminability. Others have simplified the scoring system, while incorporating additional categories, to sum to a maximum of 20 points.

Historical Background

The BI evolved over a 10-year period from the mid-1950s until its publication in 1964. It was developed to permit nursing staff to assess the ability of patients with neuro-muscular and musculoskeletal disorders to care for themselves. It was one of the first measures of activities of daily living (ADL) to be developed. Since its initial publication, it has been modified to both expand and restrict the item scoring. The BI is widely used in rehabilitation centers, despite subsequent investigations identifying problems with the scaling and sum-scoring system. The BI remains popular as it includes the key physical and self-care items important for discharge planning and is simple to use.

Following the appearance of the BI, many other indices of function have been developed, underlining the importance of this type of tool in rehabilitation practice. The BI and the Functional Independence Measure (FIM) are the two most widely used measures of ADL in stroke research. The BI tends to be used more frequently in Europe while the FIM is more likely to be used in North America.

Psychometric Data

The original version of the BI was developed without the investigation of content validity for item inclusion or validity of the scoring system. Many authors have questioned and subsequently suggested modifications to the scoring system. Most recently, de Morton et al. (2008) used Rasch analysis to investigate the validity of item score summation for the BI's original and modified versions. They found that score summation was not valid and although rescoring may improve the validity of the data

collected at discharge, methods for rescoring outcome measures are not commonly used in rehabilitation.

Many studies have found the BI to have high inter-rater and retest reliability. The low number of scoring categories for some individual items means that the BI is less likely to be as discriminative or responsive to change as scales such as the Functional Independence Measure (FIM), which has seven scoring categories for each item.

Despite problems with some psychometric properties of the BI, it has good clinical utility in that it requires little staff training, is quick and easy to administer, and costs nothing.

Clinical Uses

The BI is widely used in inpatient rehabilitation settings. It encompasses most of the important physical aspects of daily function, but does not directly address impairment to communication, cognition, or hearing and vision. The BI is simple and easy to use with well-defined categories; so minimal training or familiarization is required.

Cross References

► Functional Independence Measure (FIM)
► Rivermead Mobility Index

References and Readings

de Morton, N., Keating, J., & Davidson, M. (2008). Rasch analysis of the Barthel index in the assessment of hospitalized older patients after admission for an acute medical condition. *Archives of Physical Medicine and Rehabilitation, 89*(4), 641–647.

Mahoney, F., & Barthel, D. (1965). Functional evaluation: The Barthel Index. *Maryland State Medical Journal, 14*, 61–65.

McDowell, I., & Newell, C. (1996). *Measuring health – a guide to rating scales and questionnaires* (2nd ed., pp. 56–63). New York: Oxford University Press.

Sangha, H., Lipson, D., Foley, N., Salter, K., Bhogal, S., Pohani, G., & Teasell, R. W. (2005). A comparison of the Barthel Index and the Functional Independence Measure as outcome measures in stroke rehabilitation: patterns of disability scale usage in clinical trials. *International Journal of Rehabilitation Research, 28*(2), 135–139.

Shah, S., Vanclay, F., & Cooper, B. (1989). Improving the sensitivity of the Barthel Index for stroke rehabilitation. *Journal of Clinical Epidemiology, 42*(8), 703–709.

Basal Forebrain

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Definition

The basal forebrain is a collection of nuclei and tracts that lie near the bottom and front of the brain. It includes the nucleus basalis, diagonal band of Broca, and medial septal nuclei. This area's neurons are major producers of acetylcholine which is then distributed throughout the brain and most importantly to the cerebral cortex and amygdala. The basal forebrain is most commonly damaged by an aneurysm of the anterior communicating artery. When this occurs, there is a reduction in the amount of acetylcholine in the brain, leading to impaired learning, amnesia, and confabulation. A decrease in cholinergic output by neurons of the basal forebrain is also known to occur in cases of Alzheimer's disease and senile dementia.

Cross References

► Anterior Communicating Artery

Basal Ganglia

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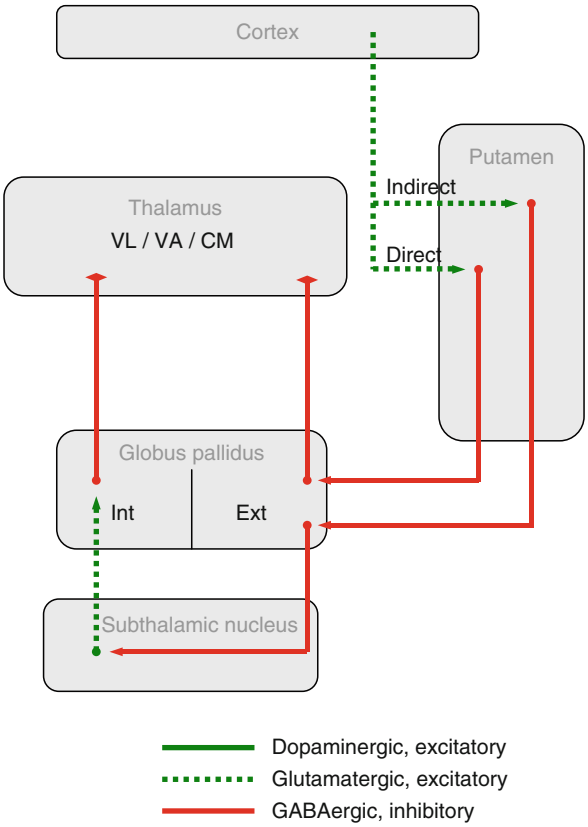
Synonyms

Basal nuclei

Definition

The basal ganglia refer specifically to a group of subcortical structures considered as extrapyramidal motor



Basal Ganglia. Figure 1 Basal ganglia circuitry. Diagram illustrates only major direct and indirect circuits. Significant cortical input is active at every level (not shown)

components. These components include caudate and putamen, substantia nigra, subthalamic nucleus, and globus pallidus (GP). Figure 1 depicts major circuitry within the basal ganglia.

Current Knowledge

Role in behavior and cognition. Rosvold demonstrated a topographical coupling between the prefrontal cortex and caudate nucleus (Rosvold, 1972; Johnson, Rosvold, & Mishkin, 1968). Considering the major outflow of the basal ganglia to the thalamus, it is not surprising that a substantial amount of research strongly supports the role of the basal ganglia in higher-order behavioral and cognitive tasks. Much of this research relies on striatal dopaminergic deficit and cortical lesion models in both human patients and animal models.

Studies in Parkinson's disease (PD) patients uncover the importance of the basal ganglia in attention – the behavior of target-focusing in the presence of distractors (Brown, Soliveri, & Jahanshahi, 1998). Levodopa therapy among PD patients improves motor behavior *and* attentional set-shifting; the absence of striatal dopamine has been shown to impair dual-task performance and self-monitoring (Brown & Marsden, 1991; Brown, Soliveri, & Jahanshahi, 1998; Taylor, Saint-Cyr, & Lang, 1986). For example, PD patients demonstrate significant impairment in Petrides' self-ordered pointing task (Petrides & Milner, 1982; West, Ergis, Winocur, & Saint-Cyr, 1998).

The basal ganglia have been shown to regulate temporal coupling and ordering of both motor and nonmotor sequences (Malapani et al., 1998; Kotz, Schwartze, & Schmidt-Kassow, 2009). Interestingly, a role in auditory rhythm detection and generation has been supported (Grahn & Brett, 2007, 2008); this is analogous to the well-established role of the basal ganglia in motor timing and pattern generation, making the circuitry a "rhythm-pattern generator" both in executive (motor) and perceptual (cognitive) realms. The implications of this and similar work for the perceptual and executive aspects of language are well-demonstrated (Kotz, Schwartze, & Schmidt-Kassow, 2009). For example, Smits-Bandstra et al. have described the basal ganglia in the setting of persons who stutter (Smits-Bandstra & De Nil, 2007).

The basal ganglia are involved in a number of other higher-order cognitive functions. For instance, problem-solving tasks that activate the prefrontal cortex also activate the basal ganglia. Recent research has shown that the basal ganglia are significantly involved in learning, including motor skill learning, sequence learning, habit learning, and category learning. In addition, the basal ganglia are involved in a number of other cognitive functions including working memory, attentional systems, and executive decision making and control. In addition, the basal ganglia are important for performing tasks automatically. While many of the behaviors engaged in seem simple and are taken for granted, these daily behaviors are really patterns of highly organized behaviors with very specific goals and purposes. As tasks are learned and practiced, they become automated, and require little to no conscious control. The basal ganglia play a critical role in the smooth and efficient operation of such highly automated behavior, and as such are part of the complex "executive" system of the brain. Thus, the basal ganglia play a critical in performing everyday practical tasks in an effortless and efficient manner (Koziol & Budding, 2009).

Organization. The *striatal complex* is composed of the neostriatum (caudate and putamen) and ventral striatum (nucleus accumbens and olfactory tubercle) (Haines, 2004). Embryologically the same, the caudate and putamen are separated by the internal capsule. Striosomes and matrix constitute a chemical and functional separation of the striatal complex: striosomes are areas of low acetylcholinesterase and high neuropeptide content, whereas matrix regions are rich in acetylcholinesterase (Bernacer, Prensa, & Gimenez-Amaya, 2007). This difference in acetylcholinesterase content provides a convenient histochemical differentiation between neostriatal regions (DiFiglia, Pasik, & Pasik, 1976).

Striatal function. The GABAergic cells of the striatum project to the internal segment of GP and substantia nigra (striosomes project mainly to pars compacta; matrix projects mainly to pars reticulata). These nuclei also receive substance-P and enkephalinergic input from the striatum (Menguala, de las Herasb, Erroa, Lanciegoa, & Gimenez-Amaya, 1999). The striatum tonically inhibits its pallidal and nigral targets.

The striatum itself receives inhibitory GABAergic projections from substantia nigra pars reticulata (Boyes & Bolam, 2007). Major excitatory input is found in glutamatergic projections from thalamus (centromedian and parafascicular nuclei) and cortex (several motor areas), as well as dopaminergic input from substantia nigra pars compacta (Kubota et al., 1987). The latter dopaminergic input terminates in both D1 and D2 dopamine receptor subtypes, an important determinant in *excitation or inhibition* of striatal neurons (Surmeier, Ding, Day, Wang, & Shen, 2007).

The *substantia nigra* (*SN*) generally refers to two nuclei, pars compacta and pars reticulata (SNpc, SNpr, respectively). The SN lies within the midbrain, caudal to the crus cerebri and rostral to the red nucleus (Haines, 2002). The SNpc contains dopaminergic neurons, while the SNpr contains mostly GABAergic neurons. Intranigral connections serve as modulatory loops: GABAergic input to SNpc decreases dopaminergic activity within the pars compacta; dopaminergic input to SNpr decreases GABAergic activity (Boyes & Bolam, 2007; DeLong & Wichmann, 2007).

Nigral function. The pars reticulata provides tonic inhibition of the thalamus, while the major function of the pars compacta is dopaminergic input to the striatum (Haines, 2002).

The *subthalamic nucleus* (STN) is inferior to the thalamus and medial to the GP; a biconvex-shaped structure, the STN is surrounded by dense bundles of myelinated fibers. The internal capsule separates the STN from the GP (Haines, 2004). Three major fiber tracts are associated with the STN: the subthalamic fasciculus (STF), the ansa lenticularis (AL), and the lenticular fasciculus (LF). The STF connects the STN and GP, crossing the internal capsule; the AL connects the GPi and the thalamus and differs from the STF in that it does not directly cross the internal capsule. Lastly, the LF crosses the internal capsule and ultimately joins the AL to form the thalamic fasciculus (or the H1 Field of Forel).

Subthalamic function. The STN is thought to modulate the entire circuitry of the basal ganglia (Hamani, Saint-Cyr, Fraser, Kaplitt, & Lozano, 2004).

The *GP* consists of two segments: internal (medial, GPi) and external (lateral, GPe). The nucleus is bounded medially by the internal capsule, and laterally by the putamen (Haines, 2004). Frequently, the term "lentiform nucleus" is used to refer to the GP and putamen together.

Pallidal function. The internal segment tonically inhibits the ventroanterior and ventrolateral nuclei of the thalamus. The external segment tonically inhibits the STN and provides transient inhibition to the internal segment (DeLong & Wichmann, 2007). It is convenient to consider the GP as the "gateway" between the basal ganglia and the thalamus. The thalamus, in turn, relays to the motor areas of the cortex.

The basal ganglia have been described in terms of functionally opposing direct and indirect pathways. Broadly, the direct pathway promotes VA/VL thalamic relay to cortex, while the indirect pathway inhibits such traffic. The following description of direct and indirect pathways is a summary and integration of previous sources.

Direct pathway. The VA/VL thalamic complex is under tonic inhibition from both GPi and SNpr; transient inhibition of these nuclei is provided by the striatum. In this way, excitation of the striatum inhibits GPi output to the thalamus, and the net effect is *disinhibition* of the VA/VL thalamic complex. The activation of striatal GABAergic projections to SNpr and GPi has two sources: cortical glutamatergic stimulation and nigral dopaminergic stimulation acting upon D1 striatal receptors. In this way, the direct pathway is a case of thalamic disinhibition by suppression of GPi activity.

Indirect pathway. If the direct pathway is considered as a suppression of GPi activity leading to disinhibition of the thalamus, the indirect pathway is described as suppression of the GPe leading to disinhibition of STN. Tonic inhibition of STN comes from GPe (whereas tonic inhibition of thalamus comes from GPi). The striatum serves to transiently inhibit GPe (as well as inhibit GPi as

previously described). The striatum contains both D1 and D2 dopamine receptors. While the direct pathway uses D1 receptors, the D2 subtype is the main striatal receptor of the indirect pathway. SNpc inhibits striatal output to GPe through these D2 receptors.

In general terms, activity through the direct pathway promotes thalamocortical activity by disinhibition of the thalamus; the indirect pathway suppresses thalamocortical activity. This opposing circuitry is thought to modulate the net effect of the basal ganglia on thalamic output.

Illness

Huntington disease, hyperkinetic, choreiform disease, autosomal dominant inheritance, pathological CAG trinucleotide repeats (Shao & Diamond, 2007). Mechanism of disease may include enhanced corticostriate activity and enhanced thalamic disinhibition (Centonze, Bernardi, & Koch, 2007). The Unified Huntington's Disease Rating Scale is widely accepted to represent an array of disease signs and symptoms (Huntington Study Group, 1996). Dopamine and glutamate antagonists, as well as GABAergic therapy have been described (Bonelli, Wenning, & Kapfhammer, 2004). Speech and gait therapy are often employed. Depression is common among Huntington's disease patients, and antidepressant treatment has been described (Korenyi & Whittier, 1967).

PD, late idiopathic onset, early onset, signs include resting tremor, oculomotor disturbance, loss of postural reflexes, among other dyskinesias. Pathology includes loss of nigral dopaminergic neurons, although the cause is multivariable (Nagatsu & Sawada, 2007; Bergman et al., 1998). Treatment can involve levodopa therapy and decarboxylase antagonists, among a variety of other pharmacological agents (Pahwa, 2006). Surgical intervention is a relatively recent development, often targeting STN and GPi (Kern & Kumar, 2007).

Other basal ganglia disorders: Wilson disease, Sydenham chorea, and ballismus

Summary of Major Components and Circuitry (See Fig. 1)

Striatum: Caudate, Putamen

Afferent

Thalamostriatal: glutamatergic, mainly from caudal intralaminar nuclei (centromedian and parafascicular nuclei); glutamatergic

Corticostriatal: glutamatergic, from primary-, pre-, supplementary-, and cingulate-motor areas

Nigrostriatal: dopaminergic from pars compacta; fibers terminate on two separate dopamine receptor types; also GABAergic from pars reticulata

Efferent

Striatopallidal: GABAergic and substance-P projections to internal segment; GABAergic and enkephalin projections to external segment of globus pallidus

Striatonigral: striosomal GABAergic projections to pars compacta; matrix GABAergic and enkephalinergic projections to pars reticulata

Globus Pallidus: Internal, External Segments

Afferent

Striatopallidal (see above)

Subthalamopallidal: glutamatergic mainly to internal segment

Nigropallidal: dopaminergic to external segment

Efferent

Pallidothalamic: GABAergic from internal segment mainly to ventral anterior nucleus of thalamus

Pallidonigral: GABAergic from external segment to pars reticulata

Pallidosubthalamic: GABAergic from external segment to subthalamic nucleus

Substantia Nigra: Pars Compacta, Pars Reticulata

Afferent

Striatonigral and pallidonigral (see above)

Subthalamonigral: glutamatergic to pars reticulata

Efferent

Nigrostriatal and nigropallidal (see above)

Nigrosubthalamic: dopaminergic from pars compacta to subthalamus

Nigrothalamic: GABAergic to ventromedian and ventrolateral nuclei of thalamus

Subthalamic Nucleus

Afferent

Pallidosubthalamic and nigrosubthalamic (see above)

Efferent
Subthalamopallidal and subthalamonigral (see above)

Cross References

- ▶ Action Tremor
- ▶ Afferent
- ▶ Assisted Living
- ▶ Ataxia
- ▶ Bradykinesia
- ▶ Caudate Nucleus
- ▶ Cerebral Cortex
- ▶ Cholinesterase Inhibitors
- ▶ Chorea
- ▶ Cortical Motor Pathways
- ▶ Cortical–Subcortical Loop
- ▶ Corticobasal Ganglionic Degeneration
- ▶ Corticobasilar Degeneration
- ▶ Deep Brain Stimulator (Parkinsons)
- ▶ Diencephalon
- ▶ Dopamine Related Dyskinesia
- ▶ Dystonia
- ▶ Efferent
- ▶ Essential Tremor
- ▶ Executive Functioning
- ▶ Gait Disorders
- ▶ Globus Pallidus
- ▶ Huntington's Disease
- ▶ Internal Capsule
- ▶ Masked Facies
- ▶ Mesolimbic Dopaminergic Projections
- ▶ Midbrain
- ▶ Movement Disorders
- ▶ Pallidotomy
- ▶ Pallidum
- ▶ Parkinson Plus Syndromes
- ▶ Parkinson's Disease
- ▶ Parkinsonian Movements
- ▶ Parkinson's Dementia
- ▶ Physiologic Tremor
- ▶ Postural Tremor
- ▶ Putamen
- ▶ Pyramidal System
- ▶ Resting Tremor
- ▶ Rigidity
- ▶ Striatum
- ▶ Substantia Nigra
- ▶ Supplementary Motor Area
- ▶ Tardive Dyskinesia
- ▶ Thalamus
- ▶ Tremor

References and Readings

Bergman, H., Feingold, A., Nini, A., Raz, A., Slovin, H., Abeles, M., et al. (1998). Physiological aspects of information processing in the basal ganglia of normal and parkinsonian primates. *Trends in Neuroscience, 21*(1), 32–38.

Bernacer, J., Prensa, L., & Gimenez-Amaya, J. M. (2007). Cholinergic interneurons are differentially distributed in the human striatum. *PLoS ONE, 2*(11), e1174.

Bonelli, R. M., Wenning, G. K., & Kapfhammer, H. P. (2004). Huntington's disease: Present treatments and future therapeutic modalities. *International Clinical Psychopharmacology, 19*(2), 51–62.

Boyes, J., & Bolam, J. P. (2007). Localization of GABA receptors in the basal ganglia. *Progress in Brain Research, 160*, 229–243.

Brown, R., & Marsden, C. (1991). Dual task performance and processing resources in normal subjects and patients with Parkinson's disease. *Brain, 114*, 215–231.

Brown, R., Soliveri, P., & Jahanshahi, M. (1998). Executive process in Parkinson's disease – random number generation and response suppression. *Neuropyschologia, 36*, 1355–1362.

Centonze, D., Bernardi, G., & Koch, G. (2007). Mechanisms of disease: Basic-research-driven investigations in humans–the case of hyperkinetic disorders. *Nature Clinical Practical Neurology, 3*(10), 572–580.

Chang, H. T. (1988). Dopamine-acetylcholine interaction in the rat striatum: A dual-labeling immunocytochemical study. *Brain Research Bulletin, 21*, 295–304.

DeLong, M. R., & Wichmann, T. (2007). Circuits and circuit disorders of the basal ganglia. *Archives of Neurology, 64*, 20–24.

DiFiglia, M., Pasik, P., & Pasik, T. (1976). A Golgi study of neuronal types in the neostriatum of monkeys. *Brain Research, 114*, 245–256.

Grahn, J., & Brett, M. (2007). Rhythm and beat perception in motor areas of the brain. *Journal of Cognitive Neuroscience, 19*, 893–906.

Grahn, J., & Brett, M. (2008). Impairment of beat-based rhythm discrimination in Parkinson's disease. *Cortex, 45*, 54–61.

Haines, D. (2002). *Fundamental neuroscience.* New York: Churchill Livingstone.

Haines, D. (2004). *Neuroanatomy: An atlas of structures, sections, and systems.* Philadelphia: Lippincott Williams & Wilkins.

Hamani, C., Saint-Cyr, J., Fraser, J., Kaplitt, M., & Lozano, A. (2004). The subthalamic nucleus in the context of movement disorders. *Brain, 127*(Pt 1), 4–20.

Huntington Study Group (1996). Unified Huntington's disease rating scale: Reliability and consistency. *Movement Disorders, 11*, 136–142.

Johnson, T., Rosvold, H., & Mishkin, M. (1968). Projections from behaviorally-defined sectors of the prefrontal cortex to the basal ganglia, septum, and diencephalons of the monkey. *Experimental Neurology, 21*, 20–34.

Kern, D., & Kumar, R. (2007). Deep brain stimulation. *Neurologist, 13*(5), 237–252.

Korenyi, C., & Whittier, J. R. (1967). Drug treatment in 117 cases of Huntington's disease with special reference to fluphenazine (Prolixin). *Psychiatric Quarterly, 41*, 203–210.

Kotz, S., Schwartze, M., & Schmidt-Kassow, M. (2009). Non-motor basal ganglia functions: A review and proposal for a model of sensory predictability in auditory language perception. *Cortex, 45*, 982–990.

Koziol, L. F., & Budding, D. E. (2009). *Subcortical structures and cognition.* New York: Springer.
Kubota, Y., Inagaki, S., Shimada, S., Kito, S., Eckenstein, F., et al. (1987). Neostriatal cholinergic neurons receive direct synaptic inputs from dopaminergic axons. *Brain Research, 413*, 179–184.
Malapani, C., Rakitin, B., Levy, R., Meck, W., Deweer, B., Dubois, B., et al. (1998). Coupled temporal memories in Parkinson's disease: A dopamine-related dysfunction. *Journal of Cognitive Neuroscience, 10*, 316–331.
Menguala, E., de las Herasb, S., Erroa, E., Lanciegoa, J. L., & Gimenez-Amaya, J. M. (1999). Thalamic interaction between the input and the output systems of the basal ganglia. *Journal of Chemical Neuroanatomy, 16*(3), 185–197.
Nagatsu, T., & Sawada, M. (2007). Biochemistry of postmortem brains in Parkinson's disease: Historical overview and future prospects. *Journal of Neural Transmission Supplement, 72*, 113–120.
Pahwa, R. (2006). Understanding Parkinson's disease: An update on current diagnostic and treatment strategies. *Journal of the American Medical Directors Association, 7*(7 Suppl. 2), 4–10.
Petrides, M., & Milner, B. (1982). Deficits on subject-ordered tasks after frontal and temporal lobe lesions in man. *Neuropsychologia, 20*, 601–604.
Rosvold, H. (1972). The frontal lobe system: Cortical-subcortical interrelationships. *Acta Neurobiologica Experimentalis (Warsaw), 32*, 439–460.
Shao, J., & Diamond, M. I. (2007). Polyglutamine diseases: Emerging concepts in pathogenesis and therapy. *Human Molecular Genetics, 15* (16) Spec No. 2, R115–R123.
Smits-Bandstra, S., & De Nil, L. (2007). Sequence skill learning in persons who stutter: Implications for cortico- striato-thalamo-cortical dysfunction. *Journal of Fluency Disorders, 32*(4), 251–278.
Surmeier, D. J., Ding, J., Day, M., Wang, Z., & Shen, W. (2007). D1 and D2 dopamine-receptor modulation of striatal glutamatergic signaling in striatal medium spiny neurons. *Trends in Neurosciences, 30*(5), 228–235.
Taylor, A., Saint-Cyr, J., & Lang, A. (1986). Frontal lobe dysfunction in Parkinson's disease: The cortical focus of neostriatal outflow. *Brain, 109*, 845–883.
West, R., Ergis, A., Winocur, G., & Saint-Cyr, J. (1998). The contribution of impaired working memory monitoring to performance of the self-ordered pointing task in normal aging and Parkinson's disease. *Neuropsychology, 12*, 546–554.

Basal Ganglia-Thalamocortical Circuit

► Cortical–Subcortical Loop

Basal Nuclei

► Basal Ganglia

Basal Nucleus of Meynert

► Nucleus Basalis of Meynert

Base Rate (Population)

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Definition

The population prevalence of a variable of interest is known as the base rate.

Current Knowledge

Base rates can be calculated using the following formula (Gouvier, 1999):

$$\text{Base Rate} = \frac{\#\text{cases with condition of interest}}{\#\text{cases in a population}}$$

In neuropsychological settings, base rates are often used to characterize diagnostic accuracy and interpret the sensitivity and specificity of a clinical assessment. The sensitivity of a test is the probability of correctly identifying an individual with impaired functioning as actually being impaired, while the specificity of a test is the probability of correctly identifying an individual with normal functioning as actually being normal (Lezak, Howieson & Loring, 2004). When the base rates of a condition are low, the sensitivity of a test may be misleading. When the base rates of a condition are high, the specificity of a test may be misleading (Podell, DeFina, Barrett, McCullen & Goldberg, 2003). The neuropsychologist should consider base rates of a disorder when selecting tests for use in a specific population. Knowledge of base rates may also indicate that impairment cutoff scores should be adjusted to interpret diagnostic accuracy. Assessments of malingering or suboptimal effort should also be conducted with consideration of base rates for a particular condition of interest (Gouvier, 1999).

Cross References

► Sensitivity

References and Readings

Gouvier, W. D. (1999). Base rates and clinical decision making in neuropsychology. In J. J. Sweet (Ed.), *Forensic neuropsychology* (pp. 27–38). New York: Taylor & Francis.

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment.* Oxford: Oxford University Press.

Podell, K., Defina, P. A., Barrett, P., McCullen, A., & Goldberg, E. (2003). Assessment of neuropsychological functioning. In I. B. Weiner, D. K. Freedheim, J. A. Schinka, & W. F. Velicer (Eds.), *Handbook of psychology* (pp. 443–466). New York: Wiley.

BASI

► Basic Achievement Skills Inventory

Basic Achievement Skills Inventory

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Synonyms

BASI

Description

The Basic Achievement Skills Inventory (BASI) is a commercially published, norm-referenced achievement test that assesses math, reading, and language skills for children and adults. Information on the test is easily accessible through the publishers' webpage (http://www.pearsonassessments.com/basi.aspx), which includes relevant excerpts from the manual, a flash demo, sample reports and others.

Forms

There are two forms: a comprehensive form and a survey form. The comprehensive form comprises six timed subtests: vocabulary, spelling, language mechanics, reading comprehension, math computation, and math application. The survey form is a screening tool comprising two subtests: verbal skills and math skills. The subtests can be administered independently to measure specific skills or in any combination. There are four grade levels (I–IV), 3rd to 4th grade, 5th to 6th grade, 7th to 8th, and 9th to 12th. Student progress can be assessed through Form A with Fall norms (August to December) and Form B with Spring norms (January to July). A growth scale value (GSV) is also made available to measure the progress of students.

Administration

The tests can be administered individually or in groups, timed or untimed; the comprehensive form takes about 2 h and the survey form takes about 50 min to complete.

Scoring and Report

Scoring for the comprehensive form is available through Q local software, mail-in scoring, or hand-scoring. There are two reports (student summary report with parent summary report, and adult summary report). The student summary report includes standard scores, percentile scores, age equivalents, and grade equivalents as well as classification (low average, average to above average) by learning objective. The adult summary report includes percent correct, grade equivalent, and classification by learning objective.

Scoring for the survey form can be obtained through Q local software and hand-scoring but not mail-in scoring. In addition to the summary report, an employment report is available for the survey form, providing standard scores compared to those of adults with different education levels.

Historical Background

Achilles N. Bardos, PhD, is the author of the test. (http://www.unco.edu/cebs/SchoolPsych/faculty/BASI/index.html)

The BASI is a newly developed test published in 2004. It was developed with the assistance of teachers who wrote the test items, which were then reviewed by curriculum experts. Content was based on curriculum standards from The Model Curriculum and Assessment Database (MCAD), a database used by educators to align with district, state, and national curriculum requirements and standards.

Psychometric Data

Standardization is reportedly based on stratified random sampling to match closely with the US Census 2000. For the comprehensive form, a grade-appropriate sample was stratified according to gender, race, parental education, and region. Standardization of Form A was based on 2,439 students tested during Fall 2002 and standardization of Form B was based on 2,130 students tested in Spring 2003. The survey form included a school-age standardization sample of 2,518 students (aged 8–18) tested in school settings and an adult sample of 2,452 adults (aged 19–80) recruited in a variety of settings.

The test has good reliability. Buros Institute test reviewers (Rhoades, 2007; Trevisan, 2007) reported the test–retest stability, internal consistency, and alternate-forms reliability to be fairly strong, with estimates ranging from 0.54 to 0.96 for individual subtest scores and 0.67–0.98 for composite scores.

Test validity is established through the Iowa Tests of Basic Skills (ITBS), the Iowa Tests of Education Development (ITED), the Tests of Adult Basic Education (TABE), the Wechsler Individual Achievement Test – Second Edition (WIAT-II) and the Woodcock Johnson Psychoeducational Battery III (WJ-III). A review of the data (http://www.pearsonassessments.com/basi-correlation.htm) indicates that intercorrelations between subtests are supported; correlation coefficients range from low to high. Rhoades (2007) noted that correlations between subtests with similar constructs are not particularly strong. Since some of the correlation studies were based on small sample size of students (around 40), unstable correlations are not unusual (Trevison, 2007).

Clinical Uses

The author proposes that the comprehensive form provides a complete evaluation of academic skills to (1) determine academic strengths and weaknesses; (2) screen for and assist in diagnosing learning disabilities; (3) place for college students; (4) make placement decisions for ESL, GED, and program placement; (5) track academic progress, etc. Specific applications in four settings are proposed in the BASI Flash Demo (http://www.pearsonassessments.com/basidemo/basi.swf): (1) K-12 school/educational setting, (2) corrections setting for intake and evaluation of offenders for placement in programs, (3) public safety for employment screening, and (4) adult and child clinical setting. Since BASI is a relatively new test, there are limited research data to support its use across the wide variety of proposed settings. Literature search using PSYCINFO and ERIC databases identifies one study (Griffith, 2006) using the BASI with military students.

Rhoades (2007) and Trevisan (2007) cautioned against individual administration of the BASI due to the lack of standardization procedures. In adult and child clinical settings, the BASI Comprehensive Form is recommended to be used as a time- and cost-effective screening, providing an overview of achievement or alternative for individually administered achievement test when detailed information is not needed. The test is not recommended for diagnostic purposes but serves only as a screener to assist in the diagnostic process. There is no current support of its use in neuropsychological testing. BASI appears best for cost-effective, group-based assessment and has been selected to be used in educational and forensic settings such as pre- and posttesting under the Florida Juvenile Justice Common Assessment Program. When more research data are available, the potential use of the BASI can be reexamined.

Cross References

► Academic Skills

References and Readings

Griffith, R. (2006). An examination of factors influencing differences in academic performance among active duty military students and naval reserve officer training corps scholarship students in a university setting. *Dissertation Abstracts International Section A*, 67, 2071. Retrieved from PsycINFO database.

Rhoades, E. K. (2007). Test review of the basic achievement skills inventory. In K. F. Geisinger, R. A. Spies, J. F. Carlson, & B. S. Plake (Eds.), *The seventeenth mental measurements yearbook [Electronic Version]*. Retrieved February 17, 2009, from the Buros Institute's Test Reviews Online website: http://unl.edu/buros

Trevisan, M. S. (2007). Test review of the basic achievement skills inventory. In K. F. Geisinger, R. A. Spies, J. F. Carlson, & B. S. Plake (Eds.), *The seventeenth mental measurements yearbook [Electronic Version]*. Retrieved February 17, 2009, from the Buros Institute's Test Reviews Online website: http://unl.edu/buros

Basilar Artery

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Definition

The *basilar artery* provides blood to the brain. This artery and the two vertebral arteries comprise the *vertebrobasilar system*, which supplies blood to the posterior part of circle of Willis and connects ("anastomoses") with blood supplied to the anterior part of the circle of Willis from the carotid arteries. It arises from the confluence of the two vertebral arteries, next to the lower brain stem, ascends parallel to the brain stem, and gives rise to the anterior inferior cerebellar artery, which supplies part of the cerebellum, some smaller branches that supply the brain stem, and the superior cerebellar artery. It finally divides into the two posterior cerebral arteries (PCA). These supply the upper brain stem, the occipital lobe, and the posterior portion of the temporal lobes.

Current Knowledge

The clinical manifestations of basilar artery occlusion depend on the location of the occlusion, the extent of thrombus, and the collateral flow. Normally, the blood flows in an anterograde fashion from the vertebral arteries to the basilar artery up to its terminal branches. This pattern of flow may vary. If the proximal segment of the basilar artery is occluded and the occlusion resulted from a slowly progressive stenosis, collateralization occurs within the cerebellum into the circumferential branches of the basilar artery. In addition, flow can be reversed from the PCAs into the distal basilar artery. Thrombosis of the basilar artery causes various clinical syndromes that result from brainstem ischemia, including cranial nerve dysfunction, difficulty in swallowing and breathing, and at its most severe, locked-in syndrome. Basilar artery thrombosis is the most common cause of locked-in syndrome. Mortality rate of basilar artery occlusion is 70%, but this can be reduced substantially through the use of antithrombotic agents.

Cross References

► Circle of Willis
► Posterior Cerebral Artery
► Vertebrobasilar System

Battery Approach

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Definition

A battery approach to neuropsychological assessment is the administration of multiple measures that cover a wide range of cognitive abilities to fully characterize an individual's neuropsychological strengths and weaknesses.

Current Knowledge

The battery approach is predicated on the existence of a variety of instruments that have been empirically developed to measure myriad aspects of neuropsychological function. Neuropsychological batteries generally contain a measure of general intellectual functioning or premorbid functioning as well as assessments of basic neuropsychological functions that may include attention, executive function, language, memory, visuospatial perception and construction, and psychomotor function. Performance on a test of general intellectual function serves as a context in which performance across neuropsychological domains can be considered. Selection of individual tests that comprise a neuropsychological battery is very likely to depend on the assessment setting, nature of the presenting problem and differential diagnosis, and the theoretical orientation of the clinician.

One of the first battery approaches was what is commonly referred to today as the "fixed battery." In the fixed battery approach, test selection is predetermined irrespective of the patient's presenting problem. A comprehensive battery of tests is administered to all patients in the same standardized manner. Collection of collateral medical and social history is obtained following administration and scoring of the neuropsychological data to avoid response bias on the part of the test administrator. Examples of this psychometrically oriented, data-driven approach include the Luria–Nebraska Battery and the Halstead–Reitan Battery. An advantage of the fixed battery approach is that it facilitates comparison of test scores across patient groups and assessment settings. Another advantage of the battery approach, when using standardized tests, is that this approach facilitates the use of technicians in the administration of the tests. This approach can also facilitate the

development of data banks for research purposes. A disadvantage, however, is that it is often time-consuming, cost-prohibitive, and may produce excessive testing sessions that are poorly tolerated by patients (Mitrushina, Boone, Razani, & D'Elia, 2005).

An alternative neuropsychological assessment method is the "flexible battery" approach. In this hypothesis-driven approach, initial test selection is guided by the patient referral question, presenting problem, and the clinical interview. A modest range of measures that survey a broad range of cognitive functions is specifically chosen to probe and characterize the patient's presumed strengths and weaknesses. Following this initial assessment, which is sometimes referred to as a "core" or "screening" battery, the clinician will then select additional tests based on the patient's performance on the core battery and reported cognitive concerns (Strauss, Sherman, & Spreen, 2006). The flexible battery approach is more focused on each individual patient's presenting problem and differential diagnosis than the fixed battery approach. As a result, the total assessment period is restricted and may be more cost-effective. However, inherent in the flexible battery approach is the inconsistent administration of tests across patient groups. That is, not all of a neuropsychologist's patients will receive the same tests, thereby limiting comparisons of findings across patient groups or settings (Mitrushina et al., 2005).

A variant of the flexible battery approach is the "process" approach, also known as the Boston process approach (Lezak, Howieson, & Loring, 2004). This method entails emphasis on the more qualitative aspects of neuropsychological performance. When completing a task, patients are closely observed for strategy formation and execution. Atypical performances will be further probed by the clinician with direct questioning or modified re-administration of the task to more fully examine the nature of the behavioral dysfunction. This approach affords a more in-depth characterization of the patient's neuropsychological abilities. However, it has been criticized for its lack of normative data and standards for the reliability and validity of its methods (Strauss et al., 2006).

Future Directions

Although all battery approaches to neuropsychological assessment have advantages and disadvantages, results from a recent survey from Sweet, Moberg and Sucy (2000) suggest that the flexible battery approach is the method that is most preferred by clinicians. According to this report, the percentage of clinicians who endorsed the flexible battery approach increased from 54% in 1989 to 70% in 1999. These data reflect the relative popularity of this approach, and suggest that it is likely to remain in favor in the coming years.

Cross References

- ▶ Boston Process Approach
- ▶ Fixed Battery
- ▶ Flexible Battery

References and Readings

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment.* Oxford: Oxford University Press.

Mitrushina, M., Boone, K. B., Razani, J., & D'Elia, L. F. (2005). *Handbook of normative data for neuropsychological assessment.* Oxford: Oxford University Press.

Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary.* Oxford: Oxford University Press.

Sweet, J. J., Moberg, P. J., & Sucy, Y. (2000). Ten-year follow-up survey of clinical neuropsychologists: Part 1. Practices and beliefs. *The Clinical Neuropsychologist, 14*, 18–37.

Battle Fatigue

- ▶ Posttraumatic Stress Disorder

Battle Sign

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Synonyms

Periauricular or mastoid ecchymosis

Definition

Crescent-shaped bruise wrapping behind the base of the ear lobe and extending posteriorly towards the point of the neck where the base of the skull meets the neck. This

clinical symptom indicates the presence of a skull fracture. A patient with this symptom may present with bloody discharge of the ear. The bruise results from the force of impact, which forces the flow of blood out of the vascular endothelium of a vessel into adjacent skin tissue. The battle sign may occur a few days following the onset of the skull fracture.

Cross References

► Depressed Skull Fracture

References and Readings

Victor, M., & Ropper, A. H. (2001). *Principles of neurology* (7th ed., pp. 925–953). New York: McGraw-Hill.

Baxter v. Temple (2005)

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Synonyms

Admissibility of psychological/neuropsychological evidence

Historical Background

One of the first decisions to address the admissibility of expert testimony by a psychologist or neuropsychologist as to the existence of a brain injury or mental defect was *Jenkins v. United States* (1962). This was a criminal trial in which the jury was instructed to disregard the testimony of the psychologists on the grounds that they could not give a medical opinion as to mental disease or defect because they did not have medical training. The appellate court reversed the decision holding that the expert did not need to be a medical practitioner. A later opinion, in *United States v. Riggleman* (1969) supported the position that psychologists were not excluded from testifying about criminal sanity solely because they lacked medical training. *Simmons v. Mullins* (1975) was an early appellate court decision that essentially reversed a trial court opinion that neuropsychologists were not competent to offer expert testimony on brain malfunctions from motor vehicle accidents. The appellate court held that to exclude such testimony on physical matters by psychologists would be to ignore present medical and psychological practice. Most states allow neuropsychological testimony about brain damage (Richardson & Adams, 1992) while there is a greater diversity of opinion as to testimony about causation.

Current Knowledge

In *Baxter v. Temple* (2005), defense filed a motion *in limine* to exclude the testimony of a neuropsychologist in a case of lead exposure as insufficiently unreliable because opinions were based on results from a flexible neuropsychological test battery. The defense argued successfully that the neuropsychologist's testimony should be excluded because the Boston Approach had not been subject to peer review and publication, has no known or potential error rate, and is not generally accepted in the appropriate scientific literature. In other words, Daubert factors were used by the trial judge to exclude expert neuropsychological evidence. Furthermore, the court made an important distinction between the roles of a clinical provider and forensic examiner, emphasizing that neuropsychologists in forensic practice must employ objective methods that allow them to be unbiased truth seekers. The defendant motion *in limine* was granted. Some (Reed, 1996) have argued that Daubert challenges of idiosyncratic (flexible) test combinations will eliminate the use of flexible neuropsychological batteries in forensic consulting. However, recent surveys of neuropsychologists show that the majority of neuropsychologist practitioners use a carefully constructed battery approach specifically tailored to the patient/examinee's specific issues. In 2008, the New Hampshire Supreme Court reviewed the neuropsychological literature, practices of neuropsychologists, considered relevant Daubert standards and various amicus briefs, and concluded that the exclusion of the neuropsychological testimony in *Baxter v. Temple* (2005) was in error.

Cross References

► Admissibility
► *Daubert v. Merrell Dow*
► *Expert v. Treater Role*
► Federal Rules of Evidence
► *Kumho Tire v. Carmichael*

References and Readings

Chapple v. Ganger, 851 F. Suppl. 1481 (E. D. Wash, 1994).

Greiffenstein, M. F. (2009). Basics of forensic neuropsychology. In J. Morgan & J. Ricker (Eds.), *Textbook of clinical neuropsychology.* New York: Taylor & Francis.

Greiffenstein, M. F., & Cohen, L. (2005). Neuropsychology and the law: Principles of productive attorney–neuropsychologists relations. In G. Larrabee (Ed.), *Forensic neuropsychology: A scientific approach.* New York: Oxford University Press.

Jenkins v. United States (1962). 307 F. 2d 637.

Kaufmann, P. M. (2008). Admissibility of neuropsychological evidence in criminal cases: Competency, insanity, culpability, and mitigation. In R. Denney & J. Sullivan (Eds.), *Clinical neuropsychology in the criminal forensic setting.* New York: Guilford Press.

Richardson, R. E. L., & Adams, R. L. (1992). Neuropsychologists as expert witnesses: Issues of admissibility. *The Clinical Neuropsychologist, 6,* 295–308.

Simmons v. Mullins (Pa. Super Ct. 1975). 331 A2D 892, 897.

United States v. Riggleman (4th Cir. 1969). 411 F.2d 1190.

Bayley

► Bayley Scales of Infant and Toddler Development

Bayley Scales of Infant and Toddler Development

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Synonyms

Bayley; BSID-III

Description

The Bayley Scales of Infant and Toddler Development-Third Edition (BSID-III; 2006) is often considered to be the reference standard for developmental assessment. It is an individually administered test, applicable from 1 to 42 months of age. The stated primary purpose of the BSID-III is to identify children with developmental delay and to provide information for interventions.

The BSID-III was normed on 1,700 children (divided into 17 age groups) and development was assessed across five domains: Cognitive (91 items), Language (49 receptive and 48 expressive), Motor (66 fine motor and 72 gross motor), Social-Emotional, and Adaptive. Like its predecessors, the BSID-III is a modified power test. Assessment of the first three domains is accomplished by item administration, while the latter two are completed using caregiver response to a questionnaire. A Behavior Observation Inventory is completed by both the examiner and the caregiver, and allows assessment of the child's behavior during testing and at home. The Language scale includes Receptive Communication and Expressive Communication subtests; the Motor scale includes a Fine Motor and a Gross Motor subtest. The BSID-III Social-Emotional scale is an adaptation of the Greenspan Social-Emotional Growth Chart: A Screening Questionnaire for Infants and Young Children (Greenspan, 2004). The Adaptive Behavior scale is composed of items from the Parent/Primary Caregiver Form of the Adaptive Behavior Assessment System-Second Edition (Harrison & Oakland, 2003). This scale measures areas such as communication, community use, health and safety, leisure, self-care, self-direction, functional pre-academics, home living, social and motor, and yields a General Adaptive Composite (GAC). Discrepancies between scaled scores can be reviewed to determine whether the differences between subtests are statistically significant.

Historical Background

The original BSID (Bayley, 1969) evolved from versions of developmental tests that were administered to infants enrolled in the landmark National Collaborative Perinatal Project. It was considered the reference standard for the assessment of infant development and administered to infants over the first 2½ years. The BSID was theoretically eclectic and borrowed from different research and test instruments. The test contained three components: the Mental Developmental Index (MDI), the Psychomotor Developmental Index (PDI) (M = 100, SD = 16) and the Infant Behavior Record, and was applicable from 2 to 30 months.

The BSID subsequently was revised into the BSID-II (Bayley, 1993), this due in part to the upward drift of approximately 11 points on the MDI and 10 points on the PDI, reflecting the Flynn effect. Although the mean remained the same, the SD was now 15. When compared to the original BSID, the BSID-II scores were 12 points lower on the MDI and 10 points lower on the PDI. The Behavior Rating Scale was developed to enable the assessment of state, reactions to

the environment, motivation, and interaction with people. The age range of the BSID-II was expanded to span 1–42 months. The instrument contained 22 item sets and basal and ceiling rules that differed from the original BSID. These rules were controversial because if correction for prematurity was used to determine the item set to begin administration, or if an earlier item set was employed because of developmental problems, scores tended to be somewhat lower, because the child was not automatically given credit for passing the lower item set. The BSID-II was also criticized because it did not provide area scores compatible with IDEA requirements for cognitive, motor, communication, social, and adaptive function.

Psychometric Data

On the BSID-III, norm-referenced scaled scores (M = 10, SD = 3), composite scores (M = 100, SD = 15), percentile ranks, and growth scores are provided in addition to confidence intervals for the scales and developmental age equivalents. Composite scores range from 55 to 155, depending on the scale. Internal consistency of the subtests range from .86 to .93; intercorrelations between Cognitive and Language composites was .52, for Cognitive and Motor composites, .50, and the intercorrelation between Language and Motor composites was .49. Growth scores are new and are used to longitudinally plot the child's growth over time for each subscale. This metric is calculated based on the subtest total raw score and ranges from 200 to 800 (M = 500, SD = 100). Similar to the original BSID, there are basal rules (passing the first three items at the appropriate age start-point) and ceiling or discontinue rules (a score of 0 for five consecutive items).

The correlation between the BSID-III Language Composite and the previous BSID-II MDI is .71, the Motor Composite and the BSID-II PDI = .60, and the Cognitive Composite and the BSID-II MDI = .60. However, in contrast to the expected Flynn effect, the Bayley-III Mental and Motor composite scores are approximately 7 points *higher* than the corresponding BSID-II MDI and PDI. This phenomenon has also been reported with other developmental tests such as the Battelle Developmental Inventory-Second Edition (Newborg, 2005).

Clinical Uses

Administration of the BSID-III provides quantitative and qualitative data that give insight into the child's current levels of development. Repeated administration can document the effects of an intervention program. However, changes in test content and alteration of scales in conjunction with the Flynn effect and the more recent increase in mean scores (in comparison to the previous version) make longitudinal comparisons of scores difficult in individual children or cohorts. Extracting language items from the cognitive scale also affects comparability with the MDI found in previous versions. Conversely, the five domains now allow the BSID-III to be more compatible with early intervention requirements (IDEA; PL 108–446, Part C). A criticism of the test is that it can take an exceptionally long time to administer and this can be very problematic when testing young children. The BSID-III can be used in multidisciplinary clinics, NICU follow-up programs, or as a follow-up evaluation after a child has been identified by the use of a screening test.

References and Readings

Bayley, N. (1969). *The Bayley scales of infant development.* San Antonio, TX: The Psychological Corporation.

Bayley, N. (1993). *The Bayley scales of infant development* (2nd ed.). San Antonio, TX: The Psychological Corporation.

Bayley, N. (2006). *Bayley scales of infant and toddler development* (3rd ed.). San Antonio, TX: The Psychological Corporation.

Greenspan, S. I. (2004). *Greenspan social-emotional growth chart. A screening questionnaire for infants and young children.* San Antonio, TX: Harcourt Assessment, Inc.

Harrison, P. L., & Oakland, T. (2003). *Adaptive behavior assessment system* (2nd ed.). San Antonio, TX: The Psychological Corporation.

Newborg, J. (2005). *The battelle developmental inventory* (2nd ed.). Itasca, IL: Riverside Publishing.

BBA

► Brunel Balance Assessment

BBS

► Berg Balance Scale

BCRS

► Brief Cognitive Rating Scale

BCT

► Category Test

BDAE

► Boston Diagnostic Aphasia Examination

BDI

► Beck Depression Inventory

BDI-II

► Beck Depression Inventory

Bearing

► Orientation

Beck Anxiety Inventory

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Synonyms

BAI

Description

The Beck Anxiety Inventory (BAI) (Beck, Epstein, Brown, & Steer, 1988; Beck & Steer, 1993) is a 21-item scale developed to assess the severity of anxiety symptoms. Respondents are asked to rate each item on a 4-point scale ranging from 0 (not at all) to 3 (severely, can barely stand it). Ratings are for the past week. Items are summed to obtain total scores ranging from 0 to 63 (Beck & Steer, 1993).

Beck Anxiety Inventory. Table 1 Anxiety Level by Score (Beck & Steer, 1993)

Anxiety Level	Minimal	Mild	Moderate	Severe
Score	0–7	8–15	16–25	26–63

Time to complete the measure is 10 min. An additional 5 min is required for scoring. Although the age range stated in the manual is between 17 and 80, studies have been conducted with younger populations (Osman, Hoffman, Barrios, Kopper, Breitenstein, & Hahn, 2002), providing support for its use among adolescents. The Inventory can be administered and scored by hand. Administration via interview or computer is possible, and computerized scoring is available.

Historical Background

The measure was developed to assess cognitive and psychological symptoms of anxiety, with the goal of reliably discriminating between anxiety- and depression-related symptoms. An initial item pool (86 items) was compiled from three existing scales (the Anxiety Checklist [Beck, Steer, & Brown, 1985], Physician's Desk Reference Checklist [Beck, 1978], and Situational Anxiety Checklist [Beck, 1982]). These measures contained items regarding the wide range of symptoms reported by those with anxiety disorders (Beck et al., 1988). A series of analyses were conducted to reduce the item pool to 21 (Beck et al., 1988). Initially, redundant items were eliminated. Successive iterated principal factor analyses were used to achieve further item reduction (Beck et al., 1988). Remaining items were evaluated (validity and reliability analyses) and results yielded a 21-item scale (Beck et al., 1988).

Psychometric Data

Work by Beck et al. (1988) suggests high internal consistency (0.92) and test-retest reliability over 1 week, $r(81) = 0.75$. The Inventory was shown to discriminate between those with and without anxiety-related disorders. The BAI was also moderately correlated with the revised Hamilton Anxiety Rating Scale, $r(150) = 0.51$. In addition, the validity of the BAI has been supported by findings

which suggest that the BAI and the Beck Depression Inventory (BDI) (Beck, Rush, Shaw, & Emery, 1979) measure different symptoms (Hewitt & Norton, 1993).

A number of factor-analytic studies have been conducted regarding the BAI and results have provided two- (cognitive and somatic components) (Hewitt and Norton, 1993), four- (subjective, neurophysiological, autonomic, and panic) (Beck and Steer, 1991), five- (subjective fear, somatic nervousness, neurophysiological, muscular/motoric, and respiration) (Borden, Peterson, & Jackson, 1991), six- (somatic distress, fear, autonomic hyperactivity, panic, nervousness, and motor tension) (Morin, Landreville, Colecchi, McDonald, Stone, & Ling, 1999) factor solutions. Hewitt and Norton (1993) suggest that this range in number of identified factors may, in part, be related to the inconsistency of specific items composing the factors (somatic and cognitive) and differences in factor-analytic approaches used. In psychiatric patients, Hewitt and Norton (1993) provided evidence to support that their two-factor structure was similar for men and women, despite the fact that women seemed to report more anxiety than men. Exploring the psychometric properties of the BAI with older adults, Morin et al.'s six-factor solution included "multiple subjective, motoric, and physiological dimensions of anxiety". A trend for older subjects to obtain slightly higher scores was also noted. Given the lack of consensus regarding underlying factors for the BAI, use of the total score, as recommended in its initial development (Beck & Steer, 1993), remains the predominant approach for measuring anxiety with this instrument.

Clinical Uses

The BAI appears to adequately measure anxiety-related symptoms, but may be more sensitive to physiological aspects of anxiety disorders (Ferguson, 2000). This is likely in part related to the fact that items were selected to adequately discriminate between those with anxiety versus depression. As such, Ferguson (2000) notes that the "BAI may not be sensitive to detecting anxiety disorders where psychophysiological arousal was not a prominent feature," (p. 520). He also notes that the instrument has a high level of face validity and, as such, respondent motivation may impact reporting. In general clinical settings, the Inventory may be best used in conjunction with additional findings to diagnose anxiety-related disorders and assess treatment needs and responses.

Although it has been suggested the BAI is a valuable screen tool for older adults, Morin and colleagues (1999) note that scores should be interpreted based on normative data taking into account gender, age, and living situation. In addition to its use with general clinical populations, the BAI has demonstrated utility in neuropsychological populations. The BAI has been used in clinical trials of psychotropic interventions for depression following traumatic brain injury (TBI) (Ashman et al., 2009), and as a measure of anxiety following TBI (Cantor et al., 2005). Trahan, Ross, and Trahan (2001) used the BAI to study the relationship between self-reported post-concussive and psychiatric symptoms in neurologically normal young adults and those recovering from mild TBI. They found correlations between a composite of measure of post-concussive symptoms and the BAI. The BAI has also been used to measure anxiety following stroke (Moon, Kim, Kim, Won, & Kim 2004), though some research suggests that caution is warranted in its use with this population due to the potential overlap between somatosensory anxiety symptoms and neurological consequences of stroke (Schramke, Stowe, Ratcliff, Goldstein, & Condray, 1998).

Cross References

- ▶ Anxiety
- ▶ Beck Depression Inventory

References and Readings

Ashman, T. A., Cantor, J. B., Gordon, W. A., Spielman, L., Flanagan, S., Ginsberg, A., et al. (2009). A randomized controlled trial of sertraline for the treatment of depression in persons with traumatic brain injury. *Archives of Physical Medicine & Rehabilitation, 90*(5), 733–740.

Beck, A. T. (1978). *PDR checklist.* Philadelphia: University of Pennsylvania, Center for Cognitive Therapy.

Beck, A. T. (1982). *Situational anxiety checklist (SAC).* Philadelphia: University of Pennsylvania, Center for Cognitive Therapy.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology, 56*, 893–897.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression: A treatment manual.* New York: Guilford Press.

Beck, A. T., & Steer, R. A. (1991). Relationship between the Beck Anxiety Inventory and the Hamilton Anxiety Rating Scale with anxious outpatients. *Journal of Anxiety Disorders, 5*, 213–223.

Beck, A. T., & Steer, R. A. (1993). *Beck Anxiety Inventory manual.* San Antonio, TX: Psychological Corporation.

Beck, A. T., Steer, R. A., & Brown, G. (1985). *Beck anxiety checklist.* Unpublished manuscript, University of Pennsylvania.

Borden, J. W., Peterson, D. R., & Jackson, E. A. (1991). The Beck Anxiety Inventory in nonclinical samples: Initial psychometric properties. *Journal of Psychopathology and Behavioral Assessment, 13*, 345–356.

Cantor, J. B., Ashman, T. A., Schwartz, M. E., Gordon, W. A., Hibbard, M. R., Brown, M., et al. (2005). The role of self-discrepancy theory in understanding post-traumatic brain injury affective disorders: A pilot study. *Journal of Head Trauma Rehabilitation, 20*(6), 527–543.

Ferguson, R. (2000). Using the Beck Anxiety Inventory in primary care. In M. E. Maruish (Ed.), *Handbook of psychological assessment in primary care settings* (pp. 509–536). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Hewitt, P. L., & Norton, G. R. (1993). The Beck Anxiety Inventory: A psychometric analysis. *Psychological Assessment, 5*, 408–412.

Morin, C. M., Landreville, P., Colecchi, C., McDonald, K., Stone, J., & Ling, W. (1999). The Beck Anxiety Inventory: Psychometric properties with older adults. *Journal of Clinical Geropsychology, 5*, 19–29.

Moon, Y. S., Kim, S. J., Kim, H. C., Won, M. H., & Kim, D. H. (2004). Correlates of quality of life after stroke. *Journal of the Neurological Sciences, 224*(1–2), 37–41.

Osman, A., Hoffman, J., Barrios, F. X., Kopper, B. A., Breitenstein, J. L., & Hahn, S. K. (2002). Factor structure, reliability, and validity of the Beck Anxiety Inventory in adolescent psychiatric inpatients. *Journal of Clinical Psychology, 58*(4), 443–456.

Schramke, C. J., Stowe, R. M., Ratcliff, G., Goldstein, G., & Condray R. (1998). Poststroke depression and anxiety: Different assessment methods result in variations in incidence and severity estimates. *Journal of Clinical & Experimental Neuropsychology, 20*(5), 723–737.

Trahan, D. E., Ross, C. E., & Trahan, S. L. (2001). Relationship among postconcussional-type symptoms, depression, and anxiety in neurologically normal young adults and victims of mild brain injury. *Archives of Clinical Neuropsychology, 16*, 435–445.

Beck Depression Inventory

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Synonyms

BDI; BDI-II

Description

The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is one of the most widely used self-report inventories to assess depressive symptom severity in adolescent and adult populations. It has been employed as a screening instrument in over 2,000 studies (Richter, Werner, Heerlein, Kraus, & Sauer, 1998). An amended version of the original 1961 instrument (BDI-IA; Beck, Rush, Shaw, & Emery, 1979) was developed in 1979, with publication of its manual in 1987 (Beck & Steer, 1987). A more substantial revision led to the development of the Beck Depression Inventory-II in 1996 (BDI-II; Beck, Steer, & Brown, 1996).

The BDI-II is comprised of 21 individual items reflecting specific cognitive, affective, and physical symptoms of depression. Each item includes four statements that vary in the description of symptom severity. Scores range from 0 to 3, with a score of "3" indicating severe symptoms and a score of "0" indicating an absence of concern with that particular aspect of depressive symptomatology (Lezak, Howieson, Loring, Hannay, & Fischer, 2004). The total score is the sum of all endorsed statements. If more than one statement from a given item is chosen by the patient, only the statement of greatest severity is scored. The maximum total score is 63.

As per the BDI-II Manual, the instrument requires approximately 5–10 min to complete. This, of course, may vary depending on the patient. The BDI-II is typically self-administered, but the examiner may read the items aloud to the patient if difficulties with reading or vision are evident. The administration instructions differ depending on the mode of administration (self or examiner). The BDI-II instructs individuals to respond to the items based on how they felt during "the past 2 weeks, including today." The time frame was increased from 1 week in the original instrument to 2 weeks in the BDI-II to remain consistent with the American Psychiatric Association's Diagnostic and Statistical Manual on Mental Disorders-Fourth Edition (American Psychiatric Association, 1994) diagnostic criteria for clinical depression. The BDI-II is applicable to patients aged 13–80 years.

Historical Background

The original BDI was developed with the use of descriptors provided by psychiatric patients (predominantly with depression). These descriptors were then consolidated and adopted into 21 items, several of which differ from those in the BDI-II. In an attempt to remove alternative wordings and double negatives from the original BDI instrument, Beck and colleagues (1979) developed the BDI-IA. Fifteen of the 21 original items were modified. Moran and Lambert (1983) discovered that the BDI met only six of the nine criteria for depression listed in the American Psychiatric Association's Diagnostic and Statistical Manual on Mental Disorders-Third Edition (American Psychiatric Association, 1980). Further, they reported that the BDI lacked items addressing psychomotor activity and agitation and permitted only the endorsement of decreases (not increases) in appetite and sleep (which was inconsistent with DSM-III criteria for clinical depression).

Further changes to the diagnostic criteria for clinical depression following the publications of the DSM-III-R (1987) and-DSM-IV (1994) led to further revision of the instrument. Changes implemented for what became the BDI-II included rewording of select statements, as well as (1) introduction of the items of "Agitation," "Worthlessness," "Concentration Difficulty," and "Loss of Energy;" (2) modifications to the "Insomnia" and "Loss of Appetite" items to reflect both increases and decreases in sleep and appetite; and (3) the removal of the "Body Image Change," "Work Difficulty," "Weight Loss," and "Somatic Preoccupation" items.

Psychometric Data

Standardization data for the BDI-II was obtained from 500 psychiatric outpatients and 120 undergraduates. Internal consistency was high for each sample ($\alpha = 0.92$ and 0.93, respectively). These coefficient alphas are consistent with those reported in independent study samples including: 140 psychiatric outpatients (mean age = 37.6 years) ($\alpha = 0.91$; Beck, Steer, Ball, & Ranieri, 1996), 408 psychiatric adolescents (age range = 13–17 years) ($\alpha = 0.93$; Osman, Kopper, Barrios, Gutierrez, & Bagge, 2004), 414 nonclinical undergraduates (age range = 17–39 years) ($\alpha = 0.90$; Storch, Roberti, & Roth, 2004), 414 nonclinical high-school-aged students (age range = 14–18 years) ($\alpha = 0.92$; Osman, Barrios, Gutierrez, Williams, & Bailey, 2008), and 147 nonclinical community-dwelling older adults (age range = 59–90 years) ($\alpha = 0.86$; Segal, Coolidge, Cahill, & O'Riley, 2008). Test-retest reliability of the BDI-II was assessed in a sample of 26 psychiatric outpatients (Beck et al., 1996), yielding a statistically significant correlation coefficient ($r = 0.93$).

Beck et al. (1996) demonstrated the convergent and discriminant validity of the BDI-II in 87 psychiatric outpatients by correlating BDI-II scores with scores from the Hamilton Psychiatric Rating Scale for Depression (Hamilton, 1960) and the Hamilton Rating Scale for Anxiety (Hamilton, 1959). Here, the BDI-II was more positively correlated with the Hamilton depression scale ($r = 0.71$) than the Hamilton anxiety scale ($r = 0.47$). While assessing the factor validity of the BDI-II, Beck and colleagues (1996) reported a two-factor solution using their outpatient standardization sample that yielded a "Somatic-Affective" factor (12 items) and a "Cognitive-Affective" factor (9 items). Items that did not significantly load onto either factor included "Pessimism" and "Loss of Interest in Sex." Similar findings were replicated using their standardization sample of undergraduates. Storch et al. (2004) replicated Beck and colleagues' (1996) two-factor solution in a sample of 414 undergraduates. However, in their model, the "Pessimism" and "Loss of Interest in Sex" items significantly loaded onto the "Cognitive-Affective" factor. The internal consistency for both cognitive and somatic factors were reported to be good ($\alpha = 0.87$ and 0.74, respectively).

Clinical Uses

The BDI-II Manual designates the following raw score classifications of depression severity: ≤ 13 = *minimal*; 14–19 = *mild*; 20–28 = *moderate*; ≥ 29 = *severe*. The instrument's developers suggested that different cut-off scores may be required depending on the characteristics of the sample and the purpose for using the instrument. Hence, on an individual basis, clinical judgment must be used, as the cut-off score ranges should be viewed as guidelines (Lezak et al., 2004). As per the BDI-II Manual, clinicians may give particular consideration to items 2 (Pessimism) and 9 (Suicidal Thoughts or Wishes) to gauge suicide risk in a given psychiatric patient.

Given the burgeoning ethnic and cultural diversity of psychiatric and neurological patient populations, there is an ever-increasing need for linguistically and culturally sensitive psychiatric inventories. Wiebe and Penley (2005) report on the psychometric properties of a Spanish translation of the BDI-II using a sample of 895 undergraduates. They administered the English and/or Spanish version of the BDI-II twice to each participant, with a 1-week interval between administrations. The two versions of the instrument were administered to English-only speaking or bilingual participants. In their design, the monolingual participants ($n = 539$) received the English version of the BDI-II at each time-point. One subset of the bilingual participants ($n = 355$) were administered the Spanish version of the BDI-II at each time-point, whereas the remaining bilingual subset ($n = 254$) were randomized in the administration of the Spanish and English versions of the BDI-II at each time-point. Internal consistencies for the English only and Spanish only administration were good ($\alpha = 0.89$ and 0.91, respectively). Test-retest reliabilities were statistically significant for each version across condition (English only [$r = 0.73$], Spanish only [$r = 0.86$], and randomized administration [$r = 0.76$]). Confirmatory factor analyses revealed that their data from both English and Spanish versions of the BDI-II demonstrated good fit with the factor solution reported in the BDI-II Manual.

Cognitive complexity of a given instrument of depressive symptom severity is important to consider, as such

measures may be administered to patient populations with known cognitive impairment (e.g., ► Parkinson's disease [Zgaljardic, Borod, Foldi, & Mattis, 2003] and Traumatic Brain Injury [Draper & Ponsford, 2008]). Shumway, Sentell, Unick, and Bamberg (2004) demonstrated that three versions of the BDI including the original instrument, the BDI-II, and the BDI-Primary Care (BDI-PC; Beck, Guth, Steer, & Ball, 1997) had high overall complexity based on the characteristics of (1) number of instructions, questions, and response options; (2) item length; (3) readability (grade level); and (4) linguistic problems (likely to result in "memory overload"). Nevertheless, good reliability and validity of the BDI-II have been demonstrated in patients with significant cognitive impairment (Powell, 2003).

There has been concern that the somatic complaint items included in the BDI-II may be confounded in select patients, such as the elderly or those with movement disorders (e.g., ► Parkinson's disease [PD]) (Lezak et al., 2004). In these patient populations, physical sequelae might mimic somatic symptoms of depression, leading to greater endorsement of "somatic" items on the BDI-II and resulting in false classifications of depressive symptomatology. Using the original BDI instrument, Levin, Llabre, and Weiner (1988) reported that among patients with PD, somatic complaints were associated with the depression and not the PD symptomatology (e.g., bradykinesia), suggesting that the BDI is a valid measure of depression in this particular patient population. Internal consistency for the total score of the BDI was acceptable ($\alpha = 0.88$). When the scores for the "somatic" items were removed from the total score, the internal consistency remained relatively unchanged ($\alpha = 0.89$). Green, Felmingham, Baguley, Slewa-Youan, and Simpson (2001) administered the BDI to 117 patients with Traumatic Brain Injury (TBI) 2 years after discharge from a residential brain injury rehabilitation program. Internal consistency was high ($\alpha = 0.92$). A principal component factor analysis using the BDI total score yielded a three-factor solution. The "affective" factor accounted for the largest percentage of variability, followed by "negative attitudes toward the self" and "somatic disturbance" factors. Their findings were relatively consistent with the findings from studies using non-TBI samples and suggest that BDI scores, at least for moderate levels of depression, did not appear to be influenced by high incidences of somatic complaints. Siegert, Walkey, and Turner-Stokes (2009) reported on the existence of three-factor solution, yielding a depression factor (all 21 BDI-II items) as well as a "somatic" (11 items) and a "cognitive/affective" (8 items) factor in a sample of patients with TBI. Internal consistency for item scores from each factor was strong. Siegert et al. (2009) stressed that, while the BDI-II can provide adequate means in assessing the severity of depression in patients with TBI, the separate "dimensions of depression" need to be addressed by considering all three score subtypes in this patient population in order to avoid misattributing somatic symptoms of TBI to symptoms of depression.

The BDI-II was developed to correspond with depressive disorder criteria set forth by the DSM-IV. The reliability and validity of the instrument have been established across several studies including psychiatric and neurological patients as well as nonclinical community-dwelling individuals. This appears to extend to ethnic and culturally diverse populations as well. The BDI-II is not intended to be used for the sole purpose of "specifying a clinical diagnosis," but rather as an indicator of the existence and severity of depressive symptoms (Beck et al., 1996). Further, that depression may occur concurrently with other psychiatric diagnoses (e.g., apathy) across patient populations also mandates caution when interpreting findings from the BDI-II.

Cross References

- ► Center for Epidemiologic Studies-Depression
- ► Geriatric Depression Scale
- ► Hamilton Rating Scale for Depression
- ► Self-Report Measures
- ► Zung Self-Rating Depression Scale

References and Readings

American Psychiatric Association (1980). *Diagnostic and statistical manual of mental disorders.* Washington, DC: American Psychiatric Association.

American Psychiatric Association (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., Rev.). Washington, DC: American Psychiatric Association.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Beck, A. T., Guth, D., Steer, R. A., & Ball, R. (1997). Screening for major depression disorders in medical inpatients with the Beck Depression Inventory for primary care. *Behavior Research and Therapy, 35*, 785–791.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Guilford Press.

Beck, A. T., & Steer, R. A. (1987). *Manual for the Beck Depression Inventory.* San Antonio, TX: The Psychological Corporation.

Beck, A. T., Steer, R. A., Ball, R., & Ranieri, W. (1996). Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *Journal of Personality Assessment, 67*(3), 588–597.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory Manual* (2nd ed.). San Antonio, TX: The Psychological Corporation.

Beck, A., Ward, C. H., Mendelson, l., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4*, 561–571.

Draper, K., & Ponsford, J. (2008). Cognitive functioning ten years following traumatic brain injury and rehabilitation. *Neuropsychology, 22* (5), 618–625.

Green, A., Felmingham, K., Baguley, I. J., Slewa-Younan, S., & Simpson, S. (2001). The clinical utility of the Beck Depression Inventory after traumatic brain injury. *Brain Injury, 15*(12), 1021–1028.

Hamilton, M. (1959). The assessment of anxiety states by rating. *British Journal of Medical Psychology, 32*, 50–55.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology and Neurosurgical Psychiatry, 23*, 56–62.

Levin, B. E., Llabre, M. M., & Weiner, W. J. (1988). Parkinson's disease and depression: Psychometric properties of the Beck Depression Inventory. *Journal of Neurology, Neurosurgery & Psychiatry, 51*(11), 1401–1404.

Lezak, M. D., Howieson, D. B., Loring, D. W., with Hannay, H. J., & Fischer, J. S. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.

Moran, P. W., & Lambert, M. J. (1983). A review of current assessment tools for monitoring changes in depression. In J. S. Lambert, E. R. Christensen, & S. DeJulio (Eds.), *The assessment of psychotherapy outcome* (pp. 263–303). New York: Wiley.

Osman, A., Barrios, F. X., Gutierrez, P. M., Williams, J. E., & Bailey, J. (2008). Psychometric properties of the Beck Depression Inventory-II in nonclinical adolescent samples. *Journal of Clinical Psychology, 64* (1), 83–102.

Osman, A., Kopper, B. A., Barrios, F., Gutierrez, P. M., & Bagge, C. L. (2004). Reliability and validity of the Beck Depression Inventory-II with adolescent psychiatric inpatients. *Psychological Assessment, 16* (2), 120–132.

Powell, R. (2003). Psychometric properties of the Beck Depression Inventory and the Zung Self Rating Depression Scale in adults with mental retardation. *Mental Retardation, 41*(2), 88–95.

Richter, P., Werner, J., Heerlein, A., Kraus, A., & Sauer, H. (1998). On the validity of the Beck Depression Inventory. A review. *Psychopathology, 31*(3), 160–168.

Segal, D. L., Coolidge, F. L., Cahill, B. S., & O'Riley, A. A. (2008). Psychometric properties of the Beck Depression Inventory II (BDI-II) among community-dwelling older adults. *Behavior Modification, 32*(1), 3–20.

Shumway, M., Sentell, T., Unick, G., & Bamberg, W. (2004). Cognitive complexity of self-administered depression measures. *Journal of Affective Disorders, 83*, 191–198.

Siegert, R. J., Walkey, F. H., & Turner-Stokes, L. (2009). An examination of the factor structure of the Beck Depression Inventory-II in a neurorehabilitation inpatient sample. *Journal of the International Neuropsychological Society, 15*(1), 142–147.

Storch, E. A., Roberti, J. W., & Roth, D. A. (2004). Factor structure, concurrent validity, and internal consistency of the Beck Depression Inventory-Second Edition in a sample of college students. *Depression & Anxiety, 19*(3), 187–189.

Wiebe, J. S., & Penley, J. A. (2005). A psychometric comparison of the Beck Depression Inventory-II in English and Spanish. *Psychological Assessment, 17*(4), 481–485.

Zgaljardic, D. J., Borod, J. C., Foldi, N. S., & Mattis, P. (2003). A review of the cognitive and behavioral sequelae of Parkinson's disease: Relationship to frontostriatal circuitry. *Cognitive & Behavioral Neurology, 16*(4), 193–210.

Beery Developmental Test of Visual-Motor Integration (VMI)

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Synonyms

Beery VMI; Developmental test of visual motor integration

Description

The Beery–Buktenica Developmental Test of Visual-Motor Integration (VMI; Beery, Buktenica, & Beery, 2004) is designed to identify deficits in visual-motor integration, visual perception, and motor coordination. This brief paper and pencil test requires the examinee to copy geometric designs of increasing complexity arranged in a developmental sequence. The 5th edition (2004) provides standardized normative data for individuals aged 2–18 years, and more recent norms have been established using 1,021 adults aged 19–100 years. The VMI has a Full Form and a Short Form, both of which can be administered to a group or individuals in approximately 10–15 min. The Short Form, for children aged 2–7 years, has 21 items and the Full Form extends to 30 items. Each reproduction of a geometric form is scored as 1 point if correct and 0 if incorrect, with a discontinuation rule of three consecutive failures. Raw scores are converted to age-based standard scores, percentiles, and scales scores. Two optional subtests use the same stimulus forms as the VMI, the visual perception test and the motor coordination test. These subtests are useful to compare visual-motor integration efficiency to pure visual or pure motor performance.

Historical Background

The Beery VMI, originally known as the *Developmental Form Sequence*, began to be developed in 1961. While other design-copying measures were available, they did not comprise a sequence of designs that increased in complexity or that reflected normal development. Keith Beery believed that visual-motor integration correlated with academic achievement and wanted to construct a

measure of such skill development. He began with 72 designs administered to 600 children in Illinois. Thirty designs were selected and administered to another 600 children, leading to the selection of the final 24 forms administered today.

Beery VMI was published in 1967 by Keith Beery, Norman Buktenica, and Natasha Beery, with normative data obtained in 1964 on 1,030 children. The Beery VMI has since been re-normed on more than 10,000 children. Updated editions of the Beery VMI were published in 1982 (2nd Edition), 1989 (3rd Edition), 1997 (4th Edition), and 2004 (5th Edition). Each edition offered updated norms. The most recent 5th Edition includes standardized norms for children of 2 years of age and provides visual-motor teaching methods from birth through early elementary school.

Psychometric Data

The manual reports overall average reliabilities at .92 for visual-motor integration, .91 for visual perception, and .90 for motor coordination. Beery VMI correlates .52 with the Wide Range Assessment of Visual Motor Abilities (WRAVMA) drawing subtest and .75 with the Developmental Test of Visual Perception (DTVP-2) copying subtest. Strong correlations between the Beery VMI and other visual motor assessments highlight the test's validity. Normative scores are valid for both individual and group administration, but it is recommended that those who perform below average be individually tested. It is also suggested that preschoolers be screened individually to maintain validity.

Clinical Uses

The Beery VMI is designed to be an early screening tool to identify children with visual-motor deficits that may lead to learning, neuropsychological, and behavioral problems. Beery VMI is primarily used for children because of its ability to assess age appropriate development. Beery VMI is also beginning to be used as an early screening tool for dementia in adults. Research has found that visuoconstructional deficits may be an early indicator of dementia (Malloy, Belanger, Hall, Aloia, & Salloway, 2003). Other uses of the Beery VMI are to test the effectiveness of educational and early intervention programs.

While the manual states that the visual perception and motor coordination subtests are optional, research has shown that all areas should be assessed for optimal results and clinical interpretation. Even children who perform well on the visual-motor integration portion may have deficits in the subareas of visual perception and motor coordination (Kulp & Sortor, 2003). In fact, visual perception specifically has been associated with math and reading ability (Sortor & Kulp, 2003). Research has also found a significant relation between visual-motor integration and academic achievement (Sortor & Kulp, 2003); however, these two areas are not exclusively correlated. Beery VMI is more strongly correlated with chronological age than academic achievement. Poor performance on the Beery VMI may or may not indicate academic achievement impairments.

Cross References

► Beery Developmental Test of Visual-Motor Integration (VMI)
► Bender-Gestalt, Second Edition
► Rey-Osterrieth Complex Figure Test (ROCF)

References and Readings

Beery, K. E., Buktenica, N. A., & Beery, N. A. (2004). *The Beery-Buktenica developmental test of visual-motor integration: Administration, scoring and teaching manual.* Minneapolis, MN: NCS Pearson.

Kulp, M. T., & Sortor, J. M. (2003). Clinical value of the Beery visual-motor integration supplemental test of visual perception and motor coordination. *Optometry and Vision Science, 80*(4), 312–315.

Malloy, P., Belanger, H., Hall, S., Aloia, M., & Salloway, S. (2003). Assessing visuocontructional performance in AD, MCI and normal elderly using Beery visual-motor integration test. *The Clinical Neuropsychologist, 17*(4), 544–550.

Sortor, J. M., & Kulp, M. T. (2003). Are the results of the Beery-Buktenica developmental test of visual motor integration and its subtests related to achievement test scores? *Optometry and Vision Science, 80*(11), 758–763.

Beery VMI

► Beery Developmental Test of Visual-Motor Integration (VMI)

Behavior

► Behavior Assessment System for Children (BASC)

Behavior Assessment System for Children (BASC)

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Synonyms

Behavior; Behavioral assessment; Diagnosis; Psychopathology

Description

The Behavior Assessment System for Children, Second Edition (BASC-2; Reynolds & Kamphaus, 2004) is a multimethod, multidimensional system of related instruments used to evaluate the behavior and self-perceptions of children, adolescents, and young adults aged 2 through 25 years. The BASC-2 is multimethod in that it has the following components, which may be used individually or in any combination:

1. Two rating scales, one for teachers (Teacher Rating Scales, or TRS) and one for parents (Parent Rating Scales, or PRS), which gather descriptions of the child's observable behavior, each divided into age-appropriate forms
2. A self-report scale (Self-Report of Personality, or SRP), on which the child or young adult can describe his or her emotions and self-perceptions
3. A Structured Developmental History (SDH) form
4. A form for recording and classifying directly observed classroom behavior (Student Observation System, or SOS), which is also available for PDA applications as an electronic version known as the BASC-2 POP or Portable Observation Program
5. A self-report for parents of children ages 2–18 years, designed to capture a parent's perspective on the parent–child relationship in such domains as communication, disciplinary styles, attachment, involvement, and others.

The BASC-2 is multidimensional in that it measures numerous aspects of behavior and personality, including positive (adaptive) as well as negative (clinical) dimensions. The BASC-2 is a revision of the BASC (Reynolds & Kamphaus, 1994). Users of the original BASC can shift easily to using the BASC-2. The BASC-2 retains all of the key features of the BASC and makes numerous improvements such as: improved reliabilities and additional scales (functional communication, TRS and PRS; activities of daily living, PRS; adaptability, TRS–A and PRS–A; and attention problems and hyperactivity on SRP) without lengthening the forms; a standardization sample matched to recent US population figures (Current Population Survey, 2001); greater similarity of item content across levels and between the TRS and PRS; a reduction in the length of the TRS; newly devised content scales that can be used as an aid in interpreting the primary scales and to broaden the coverage of the behavior areas assessed by BASC-2; a mixed item-response format on the SRP, which improves both scale reliability and the ability to measure at the extremes of the score ranges; expanded age range for assessing students through age 21 years who are still attending secondary school, and, on the SRP, students aged 18 through 25 attending postsecondary institutions; and more detailed clinical norms.

Scoring and interpretive software programs are available for the BASC-2 that make actuarial as well as content matches to the educational classification of emotional disturbance, and also to various DSM-IV-TR clinical diagnoses. The most popular and useful of the programs is the BASC-2 Plus program, which also scores and interprets the Content Scales, which other programs do not.

The BASC-2 was designed to facilitate the differential diagnosis and educational classification of a variety of emotional and behavioral disorders of children and to aid in the design of treatment plans. When used individually, the BASC-2 components are reliable and psychometrically sophisticated instruments that provide an array of beneficial data. When used as a total system, the BASC-2 provides information about a child from a variety of sources, providing the clinician with a coordinated set of tools for evaluation, diagnosis, and treatment planning.

As a system, the BASC-2 components afford a triangulated view of the child's behavioral problems by (1) examining behavior in multiple settings (at home and school); (2) evaluating the child's emotions, personality, and perceptions of self; (3) providing important background information, useful when making educational classifications or clinical diagnoses, an area commonly shortchanged in educational settings; and (4) a software that provides a sophisticated means for evaluating change or progress of individuals over time in response to interventions.

Key Features of the BASC-2

The BASC-2 has numerous features that make it a sophisticated and reliable system of behavior assessment. The BASC-2 assesses a wide range of distinctive dimensions. In addition to evaluating personality and behavioral problems and emotional disturbance, the BASC-2 identifies positive attributes that can be capitalized on in the treatment process, attributes that are valuable but too often ignored in clinical assessment.

The range of dimensions assessed helps in making differential diagnosis of specific categories of disorder, such as those denoted in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000), as well as general categories of problems, such as those addressed by The Individuals with Disabilities Education Act. The BASC-2 is highly relevant to federal regulations concerning the diagnosis of severe emotional disturbance among children in the schools and also is sensitive enough to detect even mild behavior problems among children in other disability categories, including learning disabilities and mental retardation.

The BASC-2 allows information from multiple sources to be compared using instruments with overlapping norms to help achieve reliable and accurate diagnoses. The software programs available for the BASC-2 will also compare data from each of the BASC-2 components and assess them for similarities and differences statistically and graphically.

Each BASC-2 component is designed for a specific setting or type of respondent because some constructs or behaviors are more important or measurable in some settings than in others. This also gives clinician's a clear view of the generalizability of the individual's behavior as well as the setting-specific nature of any behavior problems that occur principally in one type of surrounding.

The BASC-2, PRS, SRP, and SDH are available in Spanish as well as English and are also available on audio for nonreaders. The BASC-2 can be either hand scored or computer scored (manual or scannable item entry). The BASC-2 Spanish version sold in the United States was developed simultaneously with the English version and thus is far more than a translation, being a fully developed scale with yoked items (see Reynolds & Kamphaus, 2004, for a more detailed explanation).

The BASC-2 has been widely used and is available throughout the English-speaking world from Pearson Assessments of Bloomington, MN and throughout the Spanish-speaking world from TEA Ediciones in Madrid. Versions in several other languages are also available and updated information on these versions can be obtained by contacting Pearson Assessments (www.pearsonassessments.com). Estimates from sales figures indicate the BASC-2 is one of the frequently, if not the most, administered individual psychological tests in the public schools of the United States.

Historical Background

The original BASC (Reynolds & Kamphaus, 1994) was published by American Guidance Service following 7 years of development work. The original BASC was standardized for use with ages 2.5 through 18 years, and was rapidly adopted as the most frequently administered behavior scales in the schools in the United States. Versions were subsequently developed for international applications by TEA Ediciones in Spain. Clinical settings also began to adopt and use the BASC throughout North America as research demonstrated strong sensitivity and specificity in diagnostic applications. The original BASC has been used in more than 125 such research studies, many of which were large-scale, longitudinal analyses of both developmental psychopathology and treatment efficacy. Once the BASC-2 was released in 2004, use grew geometrically in all areas of practice.

Psychometric Data

The BASC-2 scales were designed to be highly interpretable and are built around clearly specified constructs with matching item content, developed through a balance of theory and empirical data. Experienced clinicians wrote the initial items and they were assigned initially to scales using expert methods but refined iteratively using an empirical approach based on the responses of nearly 50,000 individuals. Scales are consistent not only across sex and age levels but also between the teacher and parent forms. This provides a basis for consistent interpretation of scales and for meaningful across-source and across-time score comparisons. BASC-2 norms are based on large, US Census Bureau representative samples and are differentiated according to the age, sex, and clinical status of the child. Clinicians have a choice of sex-based norms or combined-sex norms when deriving standard scores for the various subscales and composites. The BASC-2 covers the full age range, 2 through 21 years, of students in preschool through high school settings, while maintaining developmental sensitivity and continuity of constructs.

The BASC-2 also offers a version of the SRP for ages 18 through 25 for use in technical schools, colleges, and universities.

The BASC-2 scales and composites have high internal consistency and test–retest reliability. Most alpha coefficients for the BASC-2 subscales and composites exceed 0.80 and are sufficiently reliable for application to diagnostic and treatment issues. Additionally, the BASC-2 offers various types of validity checks to help the clinician detect careless or untruthful responding, misunderstanding, or other threats to validity. The BASC-2 Manual demonstrates validity evidence for the proposed applications of the BASC-2 scales that is extensive and covers both theoretical and actuarial bases. Correlations with numerous other rating scales and self-reports are given as well as studies of a large number of clinical groups.

Clinical Uses

Clinical Diagnosis

The BASC-2 aids in the clinical diagnosis of disorders that are usually first apparent in childhood or adolescence. It assesses a variety of symptoms that are noted in the DSM-IV-TR.

Because the components of the BASC-2 can be used separately or in combination, the BASC-2 may be used easily in residential settings, in clinics, or by private practitioners. The PRS and SDH can be completed by a parent while the child is being evaluated by the practitioner, thus reducing the practitioner's time in the data collection process. The rating scales, the SRP, and the SOS can be repeated on a regular basis to monitor a child's progress and response to treatment.

It is highly desirable that diagnosis be linked clearly to intervention. In this respect, treatment planning can also be facilitated by the BASC-2. Problem behaviors can be delineated and targeted in a program leading to their reduction. A similar strategy can be used with deficits in adaptive skills.

Differential diagnosis is becoming an increasingly important issue in school settings. This is partly because the complexity of many children's problems requires an array of interventions that must be tailored to the individual child's needs. Consequently, the BASC-2 is designed to be sensitive to numerous presenting problems in the classroom, including deficiencies in social skills, study skills, or other adaptive skills.

Academic difficulties are frequently linked to behavior problems. Syndromes such as attention-deficit hyperactivity disorder (ADHD) and depression have known academic consequences; learning disabilities and mental retardation are often associated with adjustment problems such as low self-concept or anxiety. It is strongly suggested that every child experiencing academic difficulties receive a behavioral assessment. Additionally, research demonstrates that good behavioral assessment of constructs such as attitude to school, attitude to teachers, study skills, attention problems, and adaptability, in tandem with cognitive assessment, improves the prediction of both school performance and response to intervention.

The BASC-2 is also useful for assessing severe emotional disturbance. The rating scales can help distinguish between children with conduct disorders or social maladjustment on the one hand and those with severe emotional disturbance on the other, as called for by Federal laws. With its various components, the BASC-2 can help assess all aspects of the federal definition of severe emotional disturbance.

The BASC-2 may be particularly useful in designing individual educational plans (IEPs) for emotionally disturbed children. It allows the selection of target behaviors as well as clusters of behaviors to delineate syndromes that are an important focus of the IEP.

The BASC-2 rating scales and the SOS were also designed for use at the preschool level to help develop family service plans (FSPs) for 4- and 5-year-olds with disabilities. The SDH is well suited to identifying the service needs of families.

The BASC-2 is also useful in manifestation determination.

Manifestation determination refers to a process for determining the origin of behavior. The procedure is commonly encountered in special education and in 504 proceedings related to disciplinary actions or conduct problems. Prior to the application of any adverse action against a student with a disability, a multidisciplinary team must determine that the behavior in question was not a direct result of the student's disability. The method is based on the premise that students with a disability should not be punished for behavior that is considered to be a manifestation of the disability. For example, suppose a child with schizophrenia is experiencing auditory hallucinations at school and tells a teacher that another child is threatening to commit a violent act. Because these auditory experiences were a manifestation of the child's disability, he or she should not be punished for reporting them as actual threats.

In most cases, the BASC-2 will prove helpful in the manifestation determination process. The BASC has a long history of effectiveness in differentiating social maladjustment and conduct disorder from behavior associated with an emotional disturbance (Reynolds & Kamphaus, 2002). The BASC-2 content scales such as anger control, bullying, and executive functioning enhance its utility for such purposes.

Assessment of Individuals with Limitations of Vision and Hearing

The BASC-2 scales can be used to evaluate the behavioral and emotional status of children and adolescents with sensory impairments. The interpretation of BASC-2 test scores for these individuals requires specialized training, expertise, and supervised experience in working with groups with sensory impairment.

Program Evaluation

Repeated use of the BASC-2 TRS, PRS, SRP, and SOS can aid in identifying a child's progress in specific programs. Improvement in designated areas of behavior and in affective states may be noted, and the strengths and weaknesses of programs thus identified. The original BASC was shown in a number of evaluation studies to be sensitive to the effects of various intervention programs for young children (including the evaluation component of Head Start's Project Mastery) and adolescents (e.g., the evaluation by the Civilian Health and Medical Program of the Uniformed Services, or CHAMPUS, of the effectiveness of residential treatment for adolescents). These and other applications of the BASC in program evaluation are reviewed in Reynolds and Kamphaus (2002). With the improved score reliabilities and broadened content coverage in both clinical and adaptive domains, the BASC-2 is expected to surpass the BASC in the evaluation of programs and of interventions at both the individual and program levels.

Forensic Evaluation

The BASC-2 is appropriate for use in legal or forensic settings. According to several US Supreme Court rulings of the 1990s, evidence of the psychometric properties of tests used in a forensic setting is crucial for determining the admissibility of expert testimony based on test results. Reynolds and Kamphaus (2002) provides examples of uses of the original BASC in forensic situations such as child custody evaluations, personal injury litigation, and juvenile certification. This manual contains considerable information on the reliability of BASC-2 scores and associated standard errors of measurement, on the normative samples, and on validation studies, all of which are considered by judges in determining admissibility of testimony based partially or wholly on test data. Also presented here are additional crucial data on the ability of the BASC to measure child and adolescent psychopathology and to discriminate among various diagnostic groups, capabilities that also are included in the consideration of admissible evidence. The BASC is well established in clinical environments such as schools, child guidance centers, university clinics, and private practice settings in the United States and abroad. The use of tests in a wide variety of settings is important in establishing credibility and admissibility in various legal proceedings.

When choosing instruments for forensic evaluations, it is also important for clinicians to evaluate the instruments' ability to detect dissimulation (Reynolds & Kamphaus, 2002). In court proceedings, individuals may have much to gain by appearing to have more or fewer problems than actually exist. Because nearly any behavioral or emotional problem or disorder can be minimized or exaggerated, objective methods are needed to determine whether dissimulation has occurred. The BASC-2 has scales designed and tested for the detection of dissimulation in responding by parents, teachers, and children. In particular, the BASC-2 validity scales can identify exaggerated responding, minimization of problem reporting, inconsistencies, random answering patterns, and other response methods that lead to inaccurate depictions of the child's or adolescent's behavior. Because of the strength of its validity scales, its documented psychometric properties, and the validity of its application in behavior evaluation and differential diagnosis, the BASC-2 provides support to educate professionals or those challenged in legal proceedings regarding their diagnostic or placement decisions.

The Behavioral and Emotional Screening System (BESS)

The BESS (Kamphaus & Reynolds, 2007, a member of the BASC-2 system or family of assessment devices), is a

variant of the BASC-2 designed to identify risk for behavioral or emotional problems and predict mental health and educational outcomes. The BESS includes two parent rating forms for ages 3 through 5 and 6 through 18, two teacher rating forms for the same age groups, and one self-report form for ages 8 through 18. Each form of the BESS is brief and, therefore, practical at less than 30 items each and taking about 5–10 min of informant time and no prior training and coaching of the informant.

Using the same item response formats as the BASC-2, each BESS form produces a single score indicating "normal" (T = 20–60), "elevated" (61–70), or "extremely elevated" risk (T = 71 or higher). Validity indexes are provided for each form, and Spanish adaptations and translations for parent and teacher forms. Test scoring software is designed for large-scale screening of entire classes, grade levels, or school systems by incorporating scanned data entry of forms and sophisticated individual and group reporting options, and a tracking report option for community monitoring of behavioral and emotional adjustment status.

The BESS manual includes a detailed discussion of development procedures and a separate chapter devoted to validity and reliability evidence collected to date. Articles by DiStefano and Kamphaus (2007) and Kamphaus et al. (2007) provide additional validity evidence associated with pilot studies of the BESS. Virtually all reliability coefficients exceed 0.90 and predictive validity studies of behavioral, emotional, and educational outcomes from 2 to 4 years in length are reported, as well as correlations with numerous behavior rating scale measures of adjustment.

The BESS manual also provides a detailed discussion of promising screening practices, including the use of multiple gates and informants, desired age or grade groups for screening, and linking screening to early intervention aimed at preventing the onset of mental health disorders or unsuccessful educational outcomes.

BASC-2 Intervention Guide for Emotional and Behavioral Problems

The BASC-2 Intervention Guide, another component of the BASC-2, (Vannest, Reynolds, & Kamphaus, 2008) provides an articulation of evidence-based practices for each of 11 common types of emotional and behavioral problems: aggression, conduct problems, hyperactivity, attention problems, academic problems, anxiety, depression, somatization, functional communication, adaptability, and social skills. Each of these areas is assessed in detail (along with numerous others) on the assessment components of the BASC-2.

Each chapter provides a theoretical framework, annotated bibliography citing selections of the research literature, and the essential elements of each intervention followed by procedural steps and considerations for application. The BASC-2 Intervention Guide is a comprehensive source book (472 pp.) of interventions. The essential elements and the procedural steps are designed for use by psychologists, counselors, or other school professionals with mental health training (unlike the assessment components of the BASC-2 which require more extensive training and supervised experience in clinical assessment and are considered to be Level C products).

Corresponding Classroom guides include evidence based interventions appropriate for classroom settings organized by both externalizing and internalizing groups of behaviors and contain some sample lesson plans and forms for common interventions and procedures. Parent Tip Sheets present information about each type of problem for communication with parents or caregivers on how to help at home.

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Current Population Survey (2001). United Stated Bureau of the Census, Current Population Survey, Washington, DC: US Bureau of the Census.

DiStefano, C., & Kamphaus, R. W. (2007). Development and validation of a behavioral screener for preschool-age children. *Journal of Emotional and Behavioral Disorders, 15*, 93–102.

Kamphaus, R. W., & Reynolds, C. R. (2007). *Behavior assessment system for children – second edition (BASC-2): Behavioral and emotional screening system (BESS)*. Bloomington, MN: Pearson.

Kamphaus, R. W., Thorpe, J. S., Winsor, A. P., Kroncke, A. P., Dowdy, E. T., & VanDeventer, M. (2007). Development and predictive validity of a teacher screener for child behavioral and emotional problems at school. *Educational and Psychological Measurement, 6*, 1–15.

Ramsay, M., Reynolds, C. R., & Kamphaus, R. W. (2002). *Essentials of behavioral assessment.* New York: Wiley.

Reynolds, C. R., & Kamphaus, R. W. (1994). *Behavior assessment system for children.* Bloomington, MN: Pearson Assessments.

Reynolds, C. R., & Kamphaus, R. W. (2002). *The clinician's guide to the behavior assessment system for children.* New York: Guilford.

Reynolds, C. R., & Kamphaus, R. W. (2004). *Behavior assessment system for children* (2nd ed.). Bloomington, MN: Pearson Assessments.

Vannest, K. J., Reynolds, C. R., & Kamphaus, R. W. (2008). *Behavior assessment system for children – second edition (BASC-2) intervention guide for emotional and behavioral problems.* Bloomington, MN: Pearson.

Behavior Management

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Definition

Techniques used to control or modify an action or performance of a subject. This is a less-intensive version of behavior modification in which the goal is to develop, strengthen, maintain, decrease, or eliminate behaviors in a planned or systematic way. Behavior management skills are particularly important to enhance the probability that individuals, or groups, choose behaviors that are pro-social. Prosocial behaviors are typically seen as personally fulfilling, productive, and socially acceptable. Persons surviving a traumatic brain injury (TBI) often have behavioral disturbances such as disinhibition and/or agitation. Due to learning impairments as a result of their TBI, the traditional behavior management approaches, which are based on learning theory principles, are modified. For example, behavior management approaches with TBI survivors may focus more on stimulus control (e.g., controlling environmental cues) than operant conditioning (e.g., recalling the contingency between behaviors and the resulting consequences).

Cross References

- ▶ Behavior Analysis
- ▶ Behavior Modification
- ▶ Behavior Therapy
- ▶ Behavioral Therapy

References and Readings

Jacobs, H. E. (1993). *Behavior analysis guidelines and brain injury rehabilitation: People, principles and programs.* Gaithersburg, MD: Aspen.

Karol, R. L. (2003). *Neuropsychological intervention: The practical treatment of severe behavioural dyscontrol after acquired brain injury.* Boca Raton, FL: CRC Press.

Behavior Modification

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Synonyms

Applied behavioral analysis; Behavior therapy; Cognitive-behavioral modification

Definition

Behavior modification is the use of basic learning techniques, such as conditioning, biofeedback, assertiveness training, positive or negative reinforcement, hypnosis, or aversion therapy, to change unwanted individual or group behavior. A technique, typically based on functional assessment, used to reinforce adaptive behaviors while diminishing or extinguishing maladaptive behaviors. Seven characteristics of behavior modification, identified by Martin and Pear (2007), include:

- A strong emphasis on defining problems in terms of measurable behavior
- Making environmental adjustments to improve functioning
- Precise methods and rationales
- Dynamic real-life application of techniques
- Techniques grounded in learning and behavior theory
- Scientific demonstration linking the imposed technique with behavior change
- Strong emphasis on accountability

Cross References

- ▶ Applied Behavior Analysis
- ▶ Behavior Management
- ▶ Behavioral Analysis
- ▶ Behavioral Assessment
- ▶ Behavioral Therapy

References and Readings

Crum, C. (2004). Using a cognitive-behavioral modification strategy to increase on-task behavior of a student with a behavior disorder. *Intervention in School & Clinic, 39*(5), 305.

Hicinbothem, J., Gonsalves, S., & Lester, D. (2006). Body modification and suicidal behavior. *Death Studies, 30*(4), 351.

Lindsey, N., Reif, J., Bachand, A., & Seys, S. (2005). Behavior modification following a diagnosis of hepatitis C infection. *American Journal of Health Behavior, 29*(6), 512–519.

Martin, G. L., & Pear, J. (2007). *Behavior modification: What it is and how to do it* (8th ed.). Upper Saddle River: Pearson Prentice Hall.

Ntinas, K. (2007). Behavior modification and the principle of normalization: clash or synthesis? *Behavioral Interventions, 22*(2), 165–177.

Behavior Rating Inventory for Executive Functions

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Synonyms

BRIEF; BRIEF-A; BRIEF-P; BRIEF-SR

Description

The Behavior Rating Inventory of Executive Function (BRIEF) family of measures are rating scales completed by parents and teachers of school children aged 5–18 years (BRIEF), by parents and caregivers of preschool children aged 2–5 years (BRIEF-Preschool Version; BRIEF-P), by adolescents aged 11–18 years (BRIEF-Self-Report Version; BRIEF-SR), or by adults aged 18–90 years and their informants (BRIEF-Adult Version; BRIEF-A). Each version facilitates assessment of the everyday behavioral manifestations of executive dysfunction in the home, school, and/or work environments.

The BRIEF consists of two forms, a parent questionnaire and a teacher questionnaire, designed to assess executive function behaviors of children and adolescents aged 5–18 years in the home and school environments. It includes 86 items within eight non-overlapping clinical scales measuring Inhibit, Shift, Emotional Control, Initiate, Working Memory, Plan/Organize, Organization of Materials, and Monitor. The BRIEF also includes two validity scales – Inconsistency and Negativity. The eight scales form two broader indexes based on the factor structure, Behavioral Regulation and Metacognition, as well as an overall score, the Global Executive Composite (GEC).

The BRIEF-P measures the behavioral manifestations of executive function in preschool children aged 2–5. The BRIEF-P consists of a single form completed by parents and/or teachers/caregivers to rate the child's executive functions within the home and preschool settings. The questionnaire consists of 63 items comprising five theoretically and empirically derived clinical scales measuring: Inhibit, Shift, Emotional Control, Working Memory, and Plan/Organize. These scales form three factor-derived indexes: Inhibitory Self-Control, Flexibility, and Emergent Metacognition and one composite score – the GEC. The BRIEF-P also includes two validity scales – Inconsistency and Negativity.

The BRIEF-SR is an 80-item adolescent self-report behavior rating scale designed for older children and adolescents, ages 11–18 years with a fifth-grade or higher reading ability, to complement the BRIEF Parent and Teacher Forms. The 80 items yield information for eight non-overlapping clinical scales measuring: Inhibit, Shift, Emotional Control, Monitor, Working Memory, Plan/Organize, Organization of Materials, and Task Completion. The clinical scales form two broader indexes – the Behavioral Regulation Index (BRI) and the Metacognition Index (MI) – and yield an overall summary score, the GEC. The BRIEF-SR also includes the Negativity and Inconsistency validity indexes.

The BRIEF-A measures the adult's executive functions in his or her everyday home and work environments. Two formats are used – a self-report and an informant report. The Self-Report Form is designed to be completed by adults 18–90 years of age while the Informant Report Form is administered to an adult who is familiar with the rated individual's everyday functioning. The latter form can be used alone when the rated individual is unable to complete the Self-Report Form or has limited awareness of his or her own difficulties, or with the Self-Report Form to gain multiple perspectives on the individual's functioning. The BRIEF-A is composed of 75 items within nine nonoverlapping clinical scales measuring: Inhibit, Self-Monitor, Plan/Organize, Shift, Initiate, Task Monitor, Emotional Control, Working Memory, Organization of Materials. The clinical scales form two broader factor-based indexes: BRI and MI, and these indexes form the overall summary score, the GEC. The BRIEF-A also includes three validity scales – Negativity, Inconsistency, and Infrequency.

Historical Background

Executive functions have been historically evaluated using laboratory-based performance tests. While these types of measures offer the advantages of control over extraneous

variables and potential to fractionate and examine components of executive function separately such as planning versus working memory, they are limited in ecological validity, or the ability to predict functioning in the everyday environment. Fundamentally, executive functions are necessary for organization of goal-directed behavior in the everyday, "real world," environment. Thus, in addition to assessing these functions with clinical performance measures, it is important to also capture behavioral manifestations of executive function or dysfunction. The BRIEF was developed to measure executive function through the assessment of individuals' behavior in their everyday environments. Given the challenges of executive function assessment in the laboratory and inherent limitations to applicability in the everyday environment and to treatment, attention has increasingly turned to alternative methods of evaluation that offer enhanced ecological validity. Assessment methods that reliably tap the individual's everyday executive problem-solving in natural settings offer a complementary approach to clinical performance-based assessment.

Executive function is generally viewed as a broad umbrella term that encompasses a set of interrelated subdomains. Although authors vary in which functions are viewed as executive function domains, they typically include: initiation of goal-directed behavior, inhibition of competing actions or stimuli, planning and selection of relevant task goals, organization of behavior to solve complex problems, flexible shifting of problem-solving strategies when necessary, and monitoring and evaluation of problem-solving behavior. In support of these behaviors, working memory capacity plays a fundamental role in holding information actively "on-line" in the service of problem-solving, including planning and organization. Importantly, the executive functions are not exclusive to cognition; emotional control is also relevant to effective problem-solving activity and should be considered in any definition. Historically, executive functions have been closely associated with the integrity of the frontal lobes of the brain. Much of the evidence supporting a role for the frontal lobes in executive functions has come from studies of individuals with acquired focal damage to this region. More recently, studies using advanced brain imaging techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) have shown that the frontal lobes play an essential role in executive functions. However, these same studies have also clearly shown that executive functions are not subserved by the frontal lobes alone, but rather by distributed neural circuitry that includes other cortical regions and subcortical structures as well as the cerebellum. Furthermore, studies of individuals with acquired focal lesions in non-frontal brain regions such as the basal ganglia have provided further support for a distributed circuitry model of executive functions. Damage to any given component of this circuitry may result in executive dysfunction.

The BRIEF was originally developed beginning in 1994 by the authors following a commonly accepted developmental model of executive function. The impetus arose from frequent observations in clinical practice that parent and teacher reports of a child's functioning in the everyday environment did not always, or even often, fit with the same child's test performance on putative executive function performance tests. Although originally intended for the authors' own clinical and research use, the measure found acceptance initially in the pediatric neuropsychology domain, and was published first in 2000, with subsequent versions published in the ensuing years. Since then, the several versions of the BRIEF have become widely used across the age spectrum and across clinical, school, and research settings. Since publication, a substantial body of literature has developed examining BRIEF profiles with a wide range of clinical groups (Strauss, Sherman, & Spreen, 2006).

Psychometric Data

BRIEF (Age 5–18 Years; Parent, Teacher Forms)

Standardization: Normative data are based on 1,419 parents and 720 teachers from rural, suburban, and urban areas, reflecting 1999 US Census estimates for distributions of SES, ethnicity, and gender. Separate normative tables, including T scores, percentiles, and confidence intervals, are provided for four age groups for boys and girls separately, with norms for both the parent and teacher forms. Clinical data are based on 212 parents and 120 teachers rating children with developmental disorders or acquired neurological disorders (e.g., learning disabilities, attention-deficit/hyperactivity disorder (ADHD), traumatic brain injury (TBI), Tourette's syndrome, mental retardation, epilepsy, and language disorders).

Reliability: High internal consistency (Cronbach's alpha = 0.80–0.98 for both parent and teacher forms). Test retest reliability (Mean $r = 0.82$ for normative sample parent ratings, Mean $r = 0.88$ for clinical sample teacher ratings). Inter-Observer (teacher–parent) reliability reflected in moderate correlations (Mean $r = 0.32$ for normative samples, Mean $r = 0.34$ for clinical samples).

Validity: Evidence of validity is demonstrated by several lines of evidence including high inter-rater agreement for item-scale assignments by expert panel, factor analytic studies, and structural equation modeling. Convergent and divergent validity evidence is demonstrated by convergence with scales of inattention and impulsivity and divergence of behavioral/emotional functioning from executive functioning using the ADHD-IV, Behavior Assessment System for Children (BASC), CBCL, and CRS. Principal factor analysis of the BRIEF Parent and Teacher Forms yielded a consistent 2-factor solution (i.e., Behavioral Regulation, Metacognition) for normative and clinical samples. Two of the Scales, Working Memory and Inhibit, are clinically useful in detecting and predicting the diagnosis of ADHD.

BRIEF-Preschool (BRIEF-P; Age 2–5 Years; Parent, Caretaker Forms):

Standardization: Normative data based on child ratings from 460 parents and 302 teachers from urban, suburban, and rural areas, reflecting 1999 US Census estimates for race/ethnicity, gender, socioeconomic status, and age. Clinical samples included children in the following diagnostic/clinical groups: ADHD, low birth weight/prematurity, language disorders, autism spectrum disorders, and a mixed clinical group.

Reliability: High internal consistency (α = 0.80–0.95 for parent sample and α = 0.90–0.97 for teacher sample); test–retest reliability (r = 0.78–0.90 for parents and 0.64–0.94 for teachers); and modest correlations between parent and teacher ratings (r = 0.14–0.28).

Validity: Convergent and discriminant validity evidence established with other measures of inattention, hyperactivity-impulsivity, depression, atypicality, anxiety, and somatic complaints (ADHD-IV-P, CBCL/1½–5, BASC–PRS). Factor analytic studies provide support for a three-factor model of executive functioning embodied by the three indexes in the parent and teacher normative groups, respectively. The Working Memory and the Plan/Organize scales define the first component; the Shift and Emotional Control scales comprise the second component; and the Inhibit and Emotional Control scales define the third component.

BRIEF-Self-Report (BRIEF-SR):

Standardization: The BRIEF-SR was standardized and validated for use with children and adolescents aged 11–18 years. The normative sample includes 1,118 participants from a wide range of racial/ethnic backgrounds, educational backgrounds, as well as geographic regions that are matched to US Census data.

Reliability: The BRIEF-SR scales demonstrate appropriate reliability. Internal consistency is high for the GEC (α = 0.96) and moderate to high for the clinical scales (α = 0.72–0.96). Temporal stability is strong (r = 0.89 for the GEC over a period of approximately 5 weeks), and there is strong inter-rater agreement for the GEC with parent ratings on the BRIEF (r = 0.56). Teacher ratings on the BRIEF correlated less strongly with adolescent ratings on the BRIEF-SR (GEC r = 0.25) but were well within expectations.

Validity: Principal factor analysis of the BRIEF-SR yielded a 2-factor solution (i.e., Behavioral Regulation, Metacognition) for normative and clinical samples. Correlational analyses with other self-report behavior rating scales (i.e., Child Behavior Checklist/Youth Self-Report (CBSL/YSR), Behavior Assessment System for Children Self-Report of Personality (BASC-SRP), Child Health Questionnaire (CHQ), Profile of Mood States-Short Form (POMS-SF)) provide evidence of convergent and divergent validity for the BRIEF-SR. Examination of BRIEF-SR profiles in a variety of clinical groups provides further evidence of validity based on clinical utility. BRIEF-SR ratings for groups of adolescents with ADHD-I, ADHD-C, Insulin-Dependent Diabetes Mellitus, Autism Spectrum Disorders, and Anxiety and Depressive Disorders showed different patterns of scale elevations for each group compared to matched control groups. Correlations between adolescent and parent ratings for the clinical groups were strong, suggesting good agreement much of the time.

BRIEF-Adult (BRIEF-A; Self-Report, Informant Report)

Standardization: The BRIEF-A was standardized and validated for use with men and women from ages 18 to 90 years. The normative sample includes adults from a wide range of racial/ethnic backgrounds, educational backgrounds, as well as geographic regions that are matched to US Census data.

Reliability: The BRIEF-A has demonstrated multiple lines of evidence for reliability. Internal consistency was moderate to high for the Self-Report normative sample (α = 0.73–0.90 for clinical scales; 0.93–0.96 for indexes and GEC) and high for the Informant Report normative sample (α = 0.80–0.93 for clinical scales; 0.95–0.98 for indexes and GEC). Using a mixed sample of clinical or

healthy adults who were seen for clinical evaluation or research study participation, internal consistency was high for the Self-Report Form (α = 0.80–0.94 for clinical scales; 0.96–0.98 for indexes and GEC) and the Informant Report Form (α = 0.85–0.95 for clinical scales; 0.96–0.98 for indexes and GEC). Test-retest correlations over a 4-week period across the clinical scales ranged from r = 0.82–0.93 for the Self-Report Form (n =0.50) and from r = 0.91 to 0.94 for the Informant Report Form (n = 0.44). Correlations between Self-Report ratings and Informant Report ratings were moderate, ranging from r = 0.44 to 0.68 for the clinical scales and from 0.61 to 0.63 for the indexes and the GEC.

Validity: The BRIEF-A exhibits multiple lines of validity evidence as an ecologically sensitive measure of executive functioning in individuals with a range of conditions across a wide age range. In terms of convergent validity evidence, the Self- and Informant Report Form of the BRIEF-A scales, indexes, and GEC demonstrated significant correlations in the expected direction with self- and informant reports on the Frontal Systems Behavior Scale, Dysexecutive Questionnaire, and Cognitive Failures Questionnaire. Validity was further demonstrated via concurrent profiles of BRIEF-A scores in clinical populations such as ADHD, multiple sclerosis, epilepsy, mild cognitive impairment, and TBI. Validity studies also are reported in the Professional Manual comparing the BRIEF-A with the Clinical Assessment of Depression, the Geriatric Depression Scale, the Beck Depression Inventory-II, and the State Trait Anxiety Inventory. Factor analysis of Self-Report Form data yielded a 2-factor solution (i.e., Behavioral Regulation, Metacognition) for normative and mixed clinical/healthy adult samples, accounting for 73 and 76% of the variance, respectively. Factor analysis of Informant Report Form data also yielded a similar 2-factor solution for the normative and mixed clinical/healthy adult samples, accounting for 81 and 78% of the variance, respectively.

Clinical Uses

Given the central importance of the executive functions to the direction and control of dynamic "real world" behavior, the BRIEF was designed for a broad range of individuals with developmental, neurological, psychiatric, and medical conditions. Deficits in various subdomains of the executive functions are central characteristics of many developmental and acquired neurological disorders across the lifespan. Executive function deficits measured via the BRIEF have been demonstrated in a variety of populations such as ADHD, TBI, lesions of the frontal lobes, Type 1 Diabetes Mellitus, autism spectrum disorders, learning disabilities, myelomeningocele and hydrocephalus, Tourette's syndrome, phenylketonuria, bipolar disorder, obstructive sleep apnea, 22q11 deletion syndrome, galactosemia, sickle cell disease, and early focal frontal lesions. The BRIEF-Adult Version has been examined in individuals with Mild Cognitive Impairment, ADHD, Multiple Sclerosis, Alzheimer's disease, epilepsy, and TBI. The measures also show promise for veridicality, i.e., predicting behavior in the natural environment. The measures are increasingly being examined in relationship to other indications of everyday functioning. Correlational analyses suggest strong, logical relationships between executive function and everyday behaviors such as impulsivity with aggression and working memory with attention problems. Correlations with other real-world functioning such as adaptive functioning are reported in individuals with developmental disabilities and scholastic achievement. Teacher ratings of executive function on the BRIEF predict student performance on high-stakes testing. While there are modest correlations between the BRIEF and performance tests that tap aspects of executive functions, there are more intriguing relationships between the BRIEF and biological markers such as genetic conditions (e.g., 22q11 deletion syndrome), brain injuries (e.g., frontal lobe lesions), metabolic conditions (e.g., phenylketonuria), and structural and functional imaging (e.g., frontal volume and activation). Finally, certain profiles of executive function in the everyday environment predict diagnosis of ADHD.

An understanding of the individual's profile of executive function strengths and weaknesses can lead to targeted pharmacological, behavioral, cognitive, or other therapeutic interventions. Such strategies may be specifically targeted toward one area of executive functions such as antecedent management for children with inhibitory control deficits, or may be more programmatic, such as the comprehensive cognitive rehabilitation programs. The BRIEF has potential uses for clinical treatment design, monitoring, and outcome measurement. Data from the BRIEF can help the clinician focus on potentially problematic areas requiring further assessment. The same data may inform decisions about targets for treatment and types of interventions based on the potential for ameliorating real-world problems. Given that the BRIEF captures the patient's everyday functioning, the scales can suggest specific problems for which treatment goals and strategies can be targeted. For example, an individual who is described as disinhibited in the everyday world might have treatments and supports targeted specifically toward boosting

inhibitory control or limiting opportunity for impulsive behavior. A child with difficulties shifting set might benefit from teaching and intervention strategies that incorporate use of routines and schedules to reduce agitation and anxiety when change is needed.

Finally, behavioral assessment of executive function can also contribute to treatment monitoring and eventual outcome evaluation. Given the inherent difficulty in administering performance measures of executive function in a repeated fashion, behaviorally anchored measures may be more suited to such within-subjects methods. For example, a patient concerned about attentional difficulties might reveal problems with inhibitory control and working memory on the BRIEF. After appropriate interview and clinical diagnosis, treatment methods might include medication and cognitive-behavior therapy. To evaluate effectiveness of treatment, the measure may be administered again after starting medication, and again after a longer period to determine whether the effects of treatment are maintained. Ratings can be provided by the individual themselves or an informant in their environment who has the opportunity to regularly observe their behavior (e.g., parent, teacher, spouse). More frequent monitoring might also be appropriate in some cases, such as for individuals who sustain a mild TBI, where full neuropsychological evaluation may not be feasible or appropriate at the time, but rapid, timely assessment of functioning is important for determining when the individual may return to normal activities.

Cross References

- ▶ Attention Deficit/Hyperactivity Disorder
- ▶ Attention/Executive Functions
- ▶ Concussion
- ▶ Frontal Systems Behavior Scale (FrSBe)
- ▶ Traumatic Brain Injury

References and Readings

Baron, I. S. (2000). Behavior rating inventory of executive function [Test Review]. *Child Neuropsychology, 6*, 235–238.

Gioia, G. A., & Isquith, P. K. (2004). Ecological assessment of executive function in traumatic brain injury. *Developmental Neuropsychology, 25*, 135–158.

Gioia, G. A., Isquith, P. K., & Guy, S. C. (2001). Assessment of executive functions in children with neurologic impairment. In R. J. Simeonsson & S. Rosenthal (Eds.), *Psychological and developmental assessment: Children with disabilities and chronic conditions*. New York: Guilford Press.

Gioia, G. A., Isquith, P. K., & Kenealy, L. (2008). Assessment of behavioral aspects of executive function. In V. Anderson, R. Jacobs, & P. Anderson, (Eds.), *Executive functions and the frontal lobes: A life span approach*. Sussex, England: Psychology Press.

Gioia, G. A., Isquith, P. K., Kenworthy, L., & Barton, R. M. (2002). Profiles of everyday executive function in acquired and developmental disorders. *Child Neuropsychology, 8*, 121–137.

Isquith, P. K., Crawford, J. S., Espy, K. A., & Gioia, G. A. (2005). Assessment of executive function in preschool children. *Mental Retardation and Developmental Disability Research Review, 11*, 209–215.

Malloy, P., & Grace, J. (2005). A review of rating scales for measuring behavior change due to frontal systems damage. *Cognitive and Behavioral Neurology, 18*, 18–27.

Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). Behavior rating inventory of executive function (BRIEF). *A compendium of neuropsychological tests, administration, norms, and commentary* (3rd ed., pp. 1090–1099). New York: Oxford University Press.

Behavior Therapy

▶ Behavior Modification

Behavior/Behavioral Analysis

▶ Behavioral Assessment

Behavior/Behavioral Observation

▶ Behavioral Assessment

Behavioral Assessment

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Synonyms

Behavior/behavioral analysis; Behavior/behavioral observation

Definition

Behavioral assessment is a systematic collection of data, obtained through direct observation, often in natural

settings, rather than sole administration of standardized tests. Behavior assessment can be informal or formal and standardized. Based on learning theory, behavioral assessment considers the context of a person's actions, including antecedents that precede and might trigger the action, as well as consequences that follow the behavior which might reinforce the behavior. Behavior assessment can be used to describe a person's functioning (i.e., arousal, initiation, or agitation) and evaluate effectiveness of therapy interventions or medications. In persons who have behavioral disorders due to neurological causes, behavior assessment is the first step for evaluating the situation so that remediation recommendations can be made. Patients are often directly observed in physical or occupational therapy sessions and in the home or classroom.

Cross References

- ▶ Behavior Assessment System for Children (BASC)
- ▶ Behavior Management
- ▶ Behavior Modification
- ▶ Behavior Rating Inventory for Executive Functions
- ▶ Behavioral Analysis
- ▶ Behavioral Assessment of the Dysexecutive Syndrome
- ▶ Behavioral Therapy
- ▶ Conners Comprehensive Behavior Rating Scale
- ▶ Functional Assessment
- ▶ Functional Assessment Measure

References and Readings

Jacobs, H. E. (1993). *Behavior analysis guidelines and brain injury rehabilitation*. Gaithersburg, MD: Aspen.

Behavioral Assessment of the Dysexecutive Syndrome

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Synonyms

BADS

Description

The BADS (Wilson, Alderman, Burgess, Emslie, & Evans, 1996) is a test battery designed to predict everyday difficulties that arise as a result of Dysexecutive Syndrome (DES). It consists of six subtests and a 20-item questionnaire that assesses executive functioning in an ecologically valid way. The subtests are the Rule Shift Cards Test, Action Program Test, Key Search Test, Temporal Judgment Test, Zoo Map Test, and Modified Six Elements Test.

The Rule Shift Cards Test is a measure of cognitive flexibility that consists of 21 spiral-bound cards that are used to assess the ability to respond correctly to a rule and to shift from one rule to another. It is scored based on how long it takes to complete the task and number of errors. In the first part, examinees are asked to respond "Yes" to a red card and "No" to a black card. This component establishes a pattern of behavior that is designed to increase the probability of perseverative errors in the second part, when the rules are changed. In the second part, examinees are asked to respond "Yes" if the card just turned over is of the same color as a previously turned card and "No" if it is different.

The Action Program Test involves five steps that require simple skills that are typically part of most people's repertoires. It requires examinees to determine what needs to be done prior to concentrating on how that end is to be achieved. The test consists of a rectangular stand with a thin transparent tube with a removable lid and a cork on the bottom, while at the other end, there is a beaker that is two thirds full of water. An L-shaped rod that is not long enough to reach the cork is to the left of the stand. Examinees are asked to get the cork out of the tube by using any of the available objects without lifting the stand, the tube, or the beaker, and without touching the lid with their fingers.

In the Key Search Test, examinees are presented with an A4-sized piece of paper with a 100 mm square in the middle and a small black dot 50 mm below it. Examinees are told to pretend that the square is a field in which they lost their keys and are asked to draw a line, starting at the black dot, demonstrating how they would go about searching the field. The test is scored based on the efficiency of the search process.

The Temporal Judgment Test comprises four questions concerning everyday events, which range from requiring a few seconds to several years. Examinees are asked to make a sensible guess as to how long an event will take (e.g., How long do most dogs live?).

In the Zoo Map Test, examinees are asked to show how they would visit a series of designated locations on a map of a zoo, while following certain rules. In the high demand component, examinees incur a high number of errors by simply visiting the locations in the order given in the instructions. In the low demand component, examinees are simply required to follow the instructions to produce an error-free performance. The goal of the test is to assess spontaneous planning abilities.

The Modified Six Elements Test requires the completion of three tasks (i.e., dictation, arithmetic, and picture naming), each of which is divided into parts A and B. Examinees are required to attempt at least a part from each of the six subtests within a 10-min period and are instructed not to complete both parts of the same task consecutively. The goal of this component is to determine the examinee's ability to plan, organize, and monitor behavior.

The Dysexecutive Questionnaire (DEQ) consists of 21 items that sample the range of problems commonly associated with the Dysexecutive Syndrome. Four broad areas are sampled: emotional or personality changes, motivational changes, behavioral changes, and cognitive changes. Items are scored on a 5-point (0–4) Likert scale, ranging from "Never" to "Very Often." Two versions are available, one completed by the examinee and another by an informant.

Historical Background

The BADS is designed to evaluate patterns of deficits that are typically subsumed under the functions of the frontal lobes. Rylander (1939) enumerated the deficits as involving disturbances in attention, increased distractibility, impaired ability to learn new tasks, and deficits contending with complex information. Shallice (1982) described this pattern of deficits as comprising impairment in attentional control, which he termed the supervisory system. Baddeley (1986) analogized the supervisory system to the central executive component of working memory and suggested the term Dysexecutive Syndrome as a way of characterizing patients with this pattern of impairment. Such patients are most likely to present as impulsive, distractible, unable-to-use feedback to modify responses, and to behave inappropriately in social situations.

The BADS was developed because patients who exhibited obvious impairments with executive functioning in their day-to-day functioning often performed adequately on traditional tests of executive functioning such as the Wisconsin Card Sorting Test or the Stroop Color Word Test. To address this discrepancy, Shallice and Burgess (1991) developed the Six Elements Test, which required examinees to carry out six tasks in a limited time frame without violating certain rules. It was tailored to a difficulty level that was consistent with the high level of functioning of Shallice's and Burgess's patients. Wilson et al. (1996) modified the Six Elements Test, simplifying it for the more severely impaired and less intellectually able patients who are often seen by neuropsychologists. These modifications evolved into the BADS.

Psychometric Data

Multiple studies established the psychometric properties of the BADS. Wilson et al. (1996) found that inter-rater reliability was high, ranging from 0.88 to 1.00. Test–retest reliability was also examined, with examinees generally performing slightly, but not significantly, higher after the second administration. Correlations between the first and second test administrations were moderate, with the exception of the Zoo Map Test, where virtually no correlation was found. This lack of correlation was attributed to the presence of outliers and small sample size ($n = 25$). The test–retest reliability of the BADS was similar in pattern to other tests of "frontal lobe" functioning administered at the same time (e.g., Modified Card Sorting Test; Nelson, 1976).

The validity of the BADS was assessed across varied populations. Bennett, Ong, and Ponsford (2005) investigated the sensitivity of the BADS to executive dysfunction in a sample of 64 Australian patients who were involved in motor vehicle or workplace accidents. All the patients experienced a loss of consciousness and varying degrees of post-traumatic amnesia (PTA). The authors concluded that scores derived from the BADS and other measures used in their study were only moderately useful in assessing executive dysfunction. In contrast, several studies found the BADS to discriminate between patients and control subjects. Krabbendam, de Vugt, Derix, and Jolles (1999) were able to discriminate between schizophrenic patients and controls. Additionally, Katz, Tadmor, Felzen, and Hartman-Maeir (2007) were able to discriminate between acute and chronic schizophrenics, with the latter evidencing greater executive dysfunction. Verdejo-Garcia and Perez-Garcia (2007) examined the usefulness of the BADS in determining executive dysfunction in a Spanish sample of substance-dependent examinees (SDI). They concluded that the BADS yielded greater effect sizes for differences between SDI and controls than did traditional measures of

executive function. SDI performance on the BADS was also useful as a predictor of problems in daily activities. Additionally, deficits in BADS scores persisted following protracted abstinence, even when other neuropsychological indices showed recovery.

Clinical Uses

Wilson et al. (1996) developed the BADS to aid those involved in the assessment of examinees with brain injury to determine the extent of executive dysfunction and the likelihood that the dysfunction interferes with everyday life. The BADS can also be used to determine the presence of executive dysfunction in other patient groups, such as persons with schizophrenia and problems with substance abuse. The BADS can be a useful part of the rehabilitation process as a tool that can detect subtle difficulties with planning and organization, which then become targets of intervention.

Cross References

► Frontal Lobe

References and Readings

Baddeley, A. D. (1986). *Working memory.* Oxford: Clarendon Press.

Bennett, P. C., Ong, B., & Ponsford, J. (2005). Assessment of executive dysfunction following traumatic brain injury: Comparison of the BADS with other clinical neuropsychological measures. *Journal of the International Neuropsychological Society, 11*, 606–613.

Katz, N., Tadmor, I., Felzen, B., & Hartman-Maeir, A. (2007). The Behavioural Assessment of the Dysexecutive Syndrome (BADS) in schizophrenia and its relation to functional outcomes. *Neuropsychological Rehabilitation, 17*, 192–205.

Krabbendam, L., de Vugt, M. E., Derix, M. M. A., & Jolles, J. (1999). The Behavioural Assessment of the Dysexecutive Syndrome as a tool to assess executive functions in schizophrenia. *The Clinical Neuropsychologist, 13*, 370–375.

Nelson, H. E. (1976). A modified card sorting test sensitive to frontal lobe defects. *Cortex, 12*, 313–324.

Rylander, G. (1939). Personality changes after operations on the frontal lobes. *Acta Psychiatrica et Neurologica, Suppl. 20*, 5–81.

Shallice, T., & Burgess, P. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain, 114*, 727–741.

Verdejo-Garcia, A., & Perez-Garcia, M. (2007). Ecological assessment of executive functions in substance dependent examinees. *Drug and Alcohol Dependence, 90*, 48–55.

Wilson, B. A., Alderman, N., Burgess, P. W., Emslie, H., & Evans, J. J. (1996). *Behavioural assessment of the dysexecutive syndrome.* Bury St Edmunds, UK: Harcourt Assessment.

Behavioral Control

► Self-Regulation

Behavioral Inattention Test

► Rivermead Behavioral Inattention Test

Behavioral Marital Therapy

► Cognitive Behavioral Couples Therapy

Behavioral Memory Aids

► Cognitive Correctors

Behavioral Prothestics

► Cognitive Correctors

Behavioral Psychology

► Behaviorism

Behavioral Redirection, Cognitive Shifting, Cognitive Redirection, Refocusing

► Redirection

Behavioral Regulation

► Self-Regulation

Behavioral Therapy

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Synonyms

Behavior management; Behavior modification

Definition

Behavioral therapy is a type of psychotherapy that focuses on changing and gaining control over unwanted behaviors based upon the principles of classical and operant conditioning. It is useful in the treatment of depression, anxiety disorders, phobias, smoking cessation, weight loss, stuttering, enuresis, tics, and other medical conditions.

Historical Background

Attempts to help people solve behavioral problems through attempts that closely mirror today's "behavioral therapy" has a very long history. Nineteenth-century British penal colonies used "token economies" to reinforce inmates for obeying prison rules. The early Romans used "aversive conditioning" (e.g., placement of "putrid" spiders in the glasses of alcohol abusers) in order to decrease problem drinking. Seventeenth-century French physicians were using "thought stopping" to treat cases of obsessional thinking.

Behavioral therapy's philosophical roots are from the School of Behaviorism, which posits that psychological matters can be studied scientifically by observing overt behaviors and without reference to internal mental states. Some of the early behavior therapists included Joseph Wolpe (South Africa) and Hans Eysenck (United Kingdom). Perhaps the most well-known contributors to the early development of behavioral therapy are Ivan Pavlov and B.F. Skinner.

Ivan Pavlov (1849–1936) was a Russian physician and physiologist who published extensively in the early part of the twentieth century on his conditioned learning experiments, later to be termed "classical conditioning." In classical conditioning, also called respondent conditioning, Pavlov found that dogs would naturally salivate ("unconditioned response") when presented with food ("unconditioned stimulus"). If he paired the presentation of the unconditioned stimulus with a previously neutral stimulus, like a bell ("conditioned stimulus"), the previously neutral stimulus produced the same unconditioned response as the unconditioned stimulus, even if the unconditioned stimulus was absent. The unconditioned response thus became the "conditioned response" to the newly acquired conditioned stimulus. In other words, Pavlov found that if he rang a bell before feeding the dogs (who naturally salivated when the food was presented), eventually the bell ringing alone would make the dogs salivate whether or not the food appeared. An important behavioral therapy principle derived from this work is that if the conditioned stimulus (bell) is repeatedly presented without the unconditioned stimulus (food), the conditioned response (salivation) decreases in intensity. This process is termed "extinction" and can be found in human behavioral therapy in the treatment of phobias. For example, Wolpe treated phobic patients with a technique he named "systematic desensitization," which involves gradually exposing a patient to an anxiety-provoking stimulus until the anxiety reaction is extinguished.

Burrhus Frederick Skinner (1904–1990) expanded on the work of Pavlov with his concept of "operant conditioning," which postulates that behavior can be affected by rewards and punishments. In a famous operant conditioning experiment, a rat is in a box equipped with an automatic food dispenser. When the rat hears the dispenser release food pellets, it moves to the food tray and eats. Next, a lever is placed in the box that dispenses a few pellets of food when pressed. When the rat touches the lever, food is dispensed. Soon the rat is pressing the lever repeatedly to obtain the food. Through "operant conditioning," the rat's behavior of pressing the lever is reinforced as the rat learns to pair the pressing of the lever with the reward. In terms of behavioral therapy, human behavior can be affected by reinforcement in that desired behaviors can be rewarded (reinforced) and thereby increase in frequency while undesired behaviors can be cut off from their reinforcement and extinguished. Skinner found that the frequency and timing of the rewards given also affected how fast the new behaviors were acquired and how hard it was to extinguish them. These became known as "schedules of reinforcement." The work of Skinner also led to what is called "shaping," in which the desired behavior (e.g., training a chicken to peck a piano)

could be gradually acquired by rewarding approximations to the behavior.

Current Knowledge

Behavioral therapy has been successfully used for a variety of problem behaviors including, but not limited to, chronic pain, substance abuse, depression, phobias, autism, obesity, managing stress, smoking cessation, anorexia, obsessive–compulsive disorder, and attention-deficit/hyperactivity disorder. It has been extensively used in patients with developmental disabilities, severely disturbed psychotic patients, survivors of brain injury, and others where insight-oriented or cognitive therapies may not be effective. There are a myriad of methods involved including:

- Systematic desensitization (SD)
- Exposure and response prevention
- Contingency management
- Flooding
- Modeling
- Applied behavior analysis
- Operant conditioning
- Respondent conditioning

Some of these techniques are used in everyday life. For example, parents and teachers place stars on a refrigerator chart or bulletin board to reward desirable behavior by children. Some techniques involve accumulating points for performing a desired behavior, points that can later be exchanged for some desirable reinforcer. These "token economies" are a variation of operant conditioning and are used in a variety of settings. In addition, extinction of undesired behavior has penetrated the mainstream as seen by the use of "time out," a technique involving the removal of a child from reinforcement, seen by the child as somewhat aversive, or punishing, with the hope of decreasing the unwanted behavior.

Behavioral therapy is based on the concepts that (1) targeted behaviors can be modified by a variety of behavioral techniques and (2) that the newly acquired behaviors will be more adaptive than the undesired ones. These techniques tend to be empirical (data-driven) and observable. They do not rely for their effectiveness on any mental (cognitive) constructs like unconscious motivations. They simply identify a behavior to change and change it rather than trying to understand why the individual was performing that behavior. An example of one of these techniques is the use of SD.

This technique is often used with people who have a specific phobia (e.g., fear of snakes, fear of closed spaces, fear of heights, etc.). The phobic behavior can be defined as avoidance of, or escape from, the phobic stimulus (e.g., escaping/running away from a spider or avoiding situations involving public speaking). By escaping from the phobic stimulus patients can reduce their anxiety. The behavior of escape/avoidance is reinforced since the reduction of the anxiety is reinforcing for the individual (negative reinforcement is a concept derived from operant conditioning). In SD, patients are gradually exposed to the phobic stimuli, allowing them to acclimate themselves to it, until they are able to tolerate it. Patients create a hierarchy of fear steps that they must overcome to reach the last step, the phobic stimulus. These hierarchies can be imaginable, pictures or actual exposure. Patients deal with each successive step until the hierarchy is completed. Typically, patients are taught relaxation skills to control fear responses during exposure to the hierarchy.

Behavioral therapy treatment tends to be of shorter duration than more traditional modes of psychotherapy (e.g., psychodynamic). Initial sessions are dedicated to the explanation of the basic tenets of behavioral change (e.g., reinforcement, extinction, punishment, etc.). Once established, a variety of techniques may be utilized including:

- *Role-playing* – therapist models desired behaviors or reactions.
- *Skills training* – patient is taught new desired behaviors to replace undesired ones for parenting, social situations, public speaking, etc.
- *Flooding* – form of systematic desensitization where the patient is exposed directly to the feared stimulus to extinguish the fear response.
- *Homework* – patients are to try out new behaviors learned in therapy in real-life situations.
- *Conditioning* – application of reinforcement to increase a desired behavior or the removal of reinforcement to decrease an unwanted behavior (e.g., token economies).
- *Relaxation training* – used to help patients relieve anxiety/tension. An important component of systematic desensitization.

The use of behavioral therapy in the treatment of survivors of severe traumatic brain injury (TBI) can be problematic. Those who demonstrate severe behavioral dyscontrol as a result of their TBI also likely possess cognitive sequelae that hinder the successful therapeutic use of these techniques. Persons with severe memory deficits may not be able to recall the behavior they performed to earn a reinforcer in a contingency management system. Memory problems may also interfere

with a survivor's ability to recall that a particular behavior led to a particular consequence. Without this ability to recall contingencies, survivors are likely to not be able to make different choices (i.e., make behavior changes) for which behavior they exhibit in given situations. Therefore, behavior management strategies place special emphasis on controlling environmental stimuli in order to reduce problem behaviors (e.g., disinhibition and agitation).

The goal of behavior therapy with moderate or mild TBI survivors is to provide the patient with a behavioral repertoire that they can use to solve daily life problems as a result of cognitive deficits (i.e., compensatory approaches). Critical behavioral therapy techniques utilized include self-monitoring, scheduling of activities, role-playing, modeling, and contingency contracting.

Future Directions

Chronic diseases have replaced acute illness as the leading cause of premature death. These chronic conditions often have unhealthy behaviors at their root cause. Examples include cigarette smoking, obesity, and lack of exercise, poor nutritional habits, substance abuse, and medical noncompliance. For this reason, behavioral therapy has demonstrated great clinical value in the treatment and prevention of chronic health problems. An example of behavioral therapy's potential can be seen in the work of Carl Simonton in the treatment of cancer patients. His results confirm that patients who have received behavioral treatment plus conventional oncology treatment live twice as long as patients who had received conventional cancer treatment alone.

Cross References

- ▶ Behavioral Analysis
- ▶ Behavioral Assessment
- ▶ Behaviorism
- ▶ Cognitive Behavioral Therapy
- ▶ Homework
- ▶ Psychotherapy
- ▶ Relaxation Training
- ▶ Social Skills Training

References and Readings

Gelder, M. (1997). The future of behavior therapy. *Journal of Psychotherapy Practice, 6*(4), 285–293.

Kazdin, A., & Hersen, M. (1980). The current status of behavior therapy. *Behavior Modification, 4*(3), 283–302.

Masters, J., Burish, T., Holton, S., & Rimm, D. (1987). *Behavior therapy: Techniques and empirical findings.* San Diego: Harcourt Press Jovanovich.

Wilson, K. (1997). Science and treatment development: Lessons from the history of behavior therapy. *Behavior Therapy, 28*, 547–558.

Behavioral Variant Frontotemporal Dementia

- ▶ Frontal Temporal Dementia
- ▶ Pick's Disease

Behaviorism

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Synonyms

Behavioral psychology; Cognitive behaviorism

Definition

Behaviorism is a psychological theory (and branch of psychology), focusing on observable behavior rather than mental phenomena, that attempts to explain behavior by learning principles such as classical and operant conditioning. In classical conditioning, an unconditioned stimulus already eliciting a response is paired with a neutral stimulus. With repeated pairing, the neutral (conditioned) stimulus begins to elicit the same response as the unconditioned stimulus. Operant conditioning focuses on environmental consequences that increase (positive reinforcement) or decrease (negative reinforcement) the frequency of behavior. Early behaviorists focused exclusively on observable behavior while more recent cognitive behaviorists have applied learning principles to patterns of thought. As behaviorism historically attempted to account for behavior solely in terms of environmental factors, neuropsychology has had limited impact on the development of this approach to psychology. In contrast, neuropsychologists have attempted to

understand the neural mechanisms of learning, a notable example being Donald Hebb's seminal postulate that concordant firing in synaptically coupled neurons increases the strength of the connection between the two neurons. This early postulate proved highly influential in subsequent research on the neural mechanisms of learning.

Cross References

- ▶ Behavior Modification
- ▶ Cognitive Behavior Therapy
- ▶ Learning

References and Readings

Beggs, J. M., Brown, T. Y., Byrne, J. H., Crow, T., LeDoux, J. E., LeBar, K. (1999). Learning and memory: Basic mechanisms. In M. J. Zigmond, F. E. Bloom, S. C. Landis, J. L. Roberts, & L. R. Squire (Eds.), Fundamental neuroscience (pp. 1411–1454). San Diego, CA: Academic Press.

Mills, J. A. (1998). *Control: A history of behavioural psychology.* New York: New York University Press.

O'Donohue, W. T. (2001). *The psychology of B.F. Skinner.* Thousand Oaks, CA: Sage.

Staddon, J. E. R. (2000). *The new behaviorism: Mind, mechanism, and society.* Philadelphia, PA: Psychology Press.

Behavioral Inattention Test

- ▶ Rivermead Behavioral Inattention Test

Bell Curve

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Synonyms

Gaussian distribution; Normal curve; Normal distribution

Definition

A normal distribution of observations/scores is shaped like a "bell," with the majority of observations/scores occurring around the mean and increasingly fewer observations/scores occurring farther (above/below) from the mean (68.26% of observations/scores fall within one standard deviation of the mean; 95.44% fall within two standard deviations of the mean). A normal distribution of observations is typical in large samples acting additively and independently, and is assumed in parametric statists (e.g., t-tests, ANOVA). Standardized scores derived from neuropsychological measures are based upon (assume) normal distribution of the standardization sample, and provide for direct comparison of performance between different measures.

Cross References

- ▶ Base Rate (Population)
- ▶ Cut Off Scores, Cutting Scores
- ▶ Intelligence Quotient
- ▶ Mental Age
- ▶ Percentiles
- ▶ Standard Scores

References and Readings

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (pp. 154–156). New York: Oxford University Press.

Sattler, J. M. (2001). *Assessment of children: Cognitive applications* (pp. 91–92). La Mesa, CA: Jerome M. Sattler.

Bell's Palsy

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Synonyms

Idiopathic facial paralysis

Definition

Bell's palsy is the acute onset of paralysis of the muscles innervated by the facial nerve, not due to obvious causes such as trauma, stroke, or local infection.

Current Knowledge

Anatomy

The facial nerve innervates the muscles that control the forehead and eyebrow, close the eyelids, and move the cheeks and lips. It also supplies taste to the anterior two thirds of the tongue, and innervates the stapedius muscle (a small muscle in the middle ear, connecting the tympanic membrane to the stapes, that dampens excessive vibration in the tympanic membrane due to loud noises).

Clinical Presentation

The onset of paralysis may be preceded by pain behind the ear for 1 or 2 days. The paralysis is complete in 2 days in half the patients, and by 5 days in almost all the patients. If the stapedius muscle is involved, there may be sensitivity to noise. Taste is impaired in almost all patients. Clinically, the forehead is unfurrowed, the eye cannot close fully, the lower eyelid droops, and tears may run down the cheek. Due to weakness of the oral muscles, saliva may drip from the corner of the mouth on the effected side.

This is distinguished from a central facial palsy (e.g., due to stroke), by forehead weakness and weakness of eye closure. In a central facial weakness, there is little or no forehead involvement. In the Bell's palsy, the forehead is unfurrowed, the eyebrow is lower than on the uninvolved side, and cannot be voluntarily raised. In a central facial weakness, the eyelid closes fully, though closure may be weaker than on the uninvolved side. In Bell's palsy, eye closure is incomplete, and the lower sclera and cornea may be reddened due to exposure to air without lubrication from tears.

Epidemiology

- Incidence: 23/100,000 annually.
- Cases in women and men are equal.
- Season: no seasonal preference.
- Age: occurs equally in all age groups.

Etiology

The etiology of Bell's palsy is thought to be viral. The genome of herpes simplex virus type 1 has been identified in the fluid surrounding the facial nerve in several cases, but there is no convincing evidence that this is the case in the majority of cases.

Lyme disease can also cause Bell's palsy. In endemic areas, Lyme disease antibody tests should be done.

Ramsay-Hunt syndrome refers to Bell's palsy caused by Varicella-Zoster (the virus that causes chicken-pox and shingles). The distinguishing characteristic of Ramsay-Hunt is the presence of vesicles (small fluid filled blisters) in the eternal auditory canal or on the eternal ear.

Treatment

Antiviral agents are not effective in idiopathic cases. Steroids (prednisone is most common) decrease the probability of permanent paralysis, or aberrant re-innervation. Because of the paralysis of the muscles that close the eye, the cornea must be protected, especially at night. Artificial tears, liquid or ointment, and taping the eye shut are common treatments.

Antiviral treatment is indicated in Ramsay-Hunt, as is antimicrobial treatment in Lyme positive patients.

Prognosis

Eighty percent recover within a few weeks to 2 months. Recovery of some motor function in the first week is a good prognostic sign.

Cross References

► Lyme Disease

References and Readings

Gilden, D. (2004). Clinical practice. Bell's palsy. *New England Journal of Medicine, 351*, 1323–1331.

Hazin, R., Azizzadeh, B., & Bhatti, M. (2009). Medical and surgical management of facial nerve palsy. *Current Opinion in Opthalmology, 20*(6), 440–450.

Belle Indifference

► La Belle Indifference

Bender Visual–Motor Gestalt Test II

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Synonyms

Bender–Gestalt, second edition; BG-II

Description

As with any measure, research identifies various measurement, scoring, and standardization issues. Recent research culminated in the revision of the test, the Bender–Gestalt second edition, which was revised by Brannigan and Decker in 2003. The Bender–Gestalt second edition maintains many of the historical properties of the test that appealed to clinicians while improving the psychometric adequacy of the test. Similar to its predecessor, the Bender–Gestalt second edition requires the copying of various geometric line drawing images on a blank piece of paper. The drawing productions are rated on a 5-point scale (Global Scoring System) based on the similarity with the original line drawings, which are shown on individual cards and displayed in a sequential order. Additionally, a memory recall period ensues after the copy period.

Like the original, the Bender–Gestalt second edition has high reliability and validity and discriminates performance in individuals with a variety of learning and psychological problems. It is this latter finding – that individuals from a wide variety of clinical conditions show poor performance on line copy tasks – that contributes to the test's clinical utility. Unfortunately, theoretical explanations for poor performance as well as explanations for qualitative errors, such as figure rotations and perseverations, are still lacking. Although previous research incorporated the use of psychodynamic and personality paradigms, the most evidence-based supported inference of performance on the Bender–Gestalt second edition is as a measure of visual/perceptual–motor integration. Although many subcomponents are required in performance such as visual acuity and graphomotor skills, the integration of a visual percept with a motor programming controlling seems to be the largest source of variance on test performance (Decker, Allen, & Choca, 2006).

Historical Background

The Bender–Gestalt has historically been one of the most used measures in psychology. The Bender–Gestalt originated from Lauretta Bender's research in perception and psychopathology. She adapted designs used by Wertheimer (1923) to be used as a measure of development and psychopathology. Initially, performance was qualitatively interpreted but a need for standardized scoring systems emerged. Numerous scoring systems have been developed, with the most notable being the Pascal and Suttell (1951) method, the Koppitz (1963) developmental scoring system, and Lacks (1999) scoring system for screening for brain dysfunction. The various scoring systems and the multifaceted use of the Bender–Gestalt test, whether used as a "warm-up" prior to more intellectually challenging tasks or to screen for brain injury, have contributed to the long-standing and continued use of the measure.

Clinical Uses

The Bender–Gestalt was initially utilized by Lauretta Bender as a measure of perception and psychopathology.

The Bender–Gestalt, second edition has been used extensively for educational, medical, and other purposes. In particular, it has been utilized in education as a determinant of fine motor or visual–spatial difficulties.

References and Readings

Bender, L. (1938). *A visual motor Gestalt test and its clinical use.* New York: American Orthopsychiatric Association.

Decker, S. L., Allen, R., & Choca, J. P. (2006). Construct validity of the Bender-Gestalt II: Comparison with Wechsler intelligence scale for children III. *Perceptual and Motor Skills, 102*, 113–141.

Koppitz, E. M. (1963). *The Bender Gestalt test for young children.* New York: Grune and Stratton.

Lacks, P. (1999). *Bender Gestalt screening for brain dysfunction* (2nd ed.). New York: Wiley.

Pascal, G. R., & Suttell, B. J. (1951). *The Bender Gestalt test.* New York: Grune & Stratton.

Wertheimer, M. (1923). Studies in the theory of Gestalt psychology. *Psychologische Forschung, 4*, 301–350.

Bender–Gestalt, Second Edition

► Bender Visual–Motor Gestalt Test II

Benign Epilepsy of Childhood with Centrotemporal Spikes (BECTS)

► Benign Rolandic Epilepsy

Benign Rolandic Epilepsy

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Synonyms

Benign epilepsy of childhood with centrotemporal spikes (BECTS); Rolandic epilepsy; Sylvian seizures

Definition

Benign rolandic epilepsy (BRE) is one of the most common epilepsy syndromes and belongs to a class of epilepsy syndromes called idiopathic partial epilepsies of childhood. These syndromes are characterized by seizure onset between the ages of 18 months and 13 years and seizure remittance during childhood or adolescence. Exclusionary criteria for these syndromes include the following: absence of brain damage as evidenced on clinical examination or imaging techniques; absence of generalized tonic–clonic seizures; and absence of long-term neuropsychological impairment. A family history of idiopathic epilepsy is typical, but it is not required for diagnosis.

BRE is the most common idiopathic partial epilepsy in childhood and is distinguished by focal epileptiform discharges that are typically localized over the midtemporal or lower rolandic region. Clinical seizures are consistent with simple partial seizures, with behavioral semiology suggestive of onset in the lower portions of the pre- and postcentral gyri.

Epidemiology

It is estimated that 15–25% of school-aged children with epilepsy meet diagnostic criteria for BRE. Males are believed to be more affected (3:2). Perinatal complications, head trauma, central nervous system infections, or other minor neurological insults have been associated with about 10% of cases.

BRE is presumed to be genetically determined, with a family history of seizures documented in 21–32% of cases. The electrographic abnormality seen in BRE is believed to be the inherited trait and follows a transmission method of autosomal dominance. The gene associated with the electrographic trait is located on chromosome 15q14, though the expression of the gene appears to be complex and likely affected by a multitude of genetic, environmental, and pathological factors. As such, only about 10% of children with the electrographic trait exhibit clinical seizures.

Natural History, Prognostic Factors, Outcomes

Typical age of seizure onset in BRE is between the ages of 2 and 13 years, but seizures have been known to begin as early as 1 year of age and as late as 15 years. About 75% of cases experience seizure onset between the ages of 7 and 10 years.

Children with BRE typically have an excellent outcome. The majority of patients will experience fewer than ten seizures, and it is not uncommon for a patient to have only one seizure. Most patients experience seizure remission within 1 or 2 years of onset. Even in cases that involve difficult to control seizures, full recovery is expected and seizures beyond the age of 16 years are rare. Only about 2% of patients with BRE will experience additional seizures as an adult.

BRE is associated with good neurocognitive and social outcomes. While transient neurodevelopmental concerns have been elucidated in more recent research, it is typically believed that there is no long-term impact of BRE on adult functioning. There also appears to be no relation between seizure frequency and the duration of BRE on functional outcome.

Neuropsychology and Psychology of BRE

BRE has been traditionally thought to be a benign syndrome, resulting in few if any neurodevelopmental problems. However, more recent research has suggested that BRE can be associated with some neurocognitive problems, particularly during the active phase of the syndrome.

Among the available research, children with BRE, as a group, have been consistently demonstrated to have average intelligence. Nevertheless, more extensive neuropsychological assessment has revealed evidence of at least mild neurocognitive difficulties in up to 78% of children with BRE. Almost 56% are estimated to have neurocognitive difficulties in more than one area. The types of reported difficulties experienced by this population are varied, including problems with motor coordination, visual–motor integration, spatial processing, language processing, memory, attention, and executive functioning. However, absence of control groups, questionable quality of some cognitive measures, and variability in the definition of a deficit in cognitive functioning among the available studies has made it difficult to make generalizations about the presence of neurocognitive problems in BRE.

A 2005 study out of Australia identified the presence of cognitive deficits in a relatively large sample of children with BRE. These included problems with verbal memory and visual memory, with about 10% of children falling below two standard deviations on the verbal memory index of the wide range assessment of memory and learning (WRAML). In contrast, only 5% were below two standard deviations on the visual memory index. However, 33% were below two standard deviations on the Rey Figure Delay. Deficits in phonological processing were also noted and were believed to correspond with reading and spelling problems in the sample (Northcutt et al., 2005).

A more recent study in 2008 also confirmed the presence of academic problems in children with BRE. Specifically, children with BRE were found to have normal intelligence levels but higher rates of reading, writing, and mathematics disorders than children in a control group. Attention problems were not identified. Of note, the study reported that children with BRE who had a younger age of seizure onset were more likely to have a learning disability, and children with BRE who had prolonged interictal EEG abnormalities in sleep were significantly more likely to have a learning disability. It was concluded that children with BRE who have seizure onset prior to 8 years of age and have EEG abnormalities that persist for more than 1 year are at greater risk for developing learning disabilities (Piccinelli et al., 2008).

While neurodevelopmental problems have been verified in the active phase of BRE, it is not yet clear whether these problems remit after resolution of the EEG abnormalities. The evidence thus far would suggest no long-term impairment, but this needs to be verified through longitudinal studies.

Evaluation

The diagnosis of BRE can only be made when characteristic electrographic abnormalities and seizures are present concurrently. Individual seizures are brief (up to 1–2 min), simple partial seizures that are typically sensorimotor in expression. Oropharyngolaryngeal symptoms are common and are associated with unilateral numbness and motor features that result in gurgling and grunting noises. Hemifacial motor movements often occur and consist of sudden clonic contractions of the lower lip. Ipsilateral tonic deviation of the mouth is also a common accompanying feature. Ictal speech arrest can occur in about 40% of cases, regardless of the side of seizure focus. The neurological examination is typically normal.

Eighty percent of children with BRE experience seizures during sleep or the waking period. About 75% of seizures occur in non-REM sleep, shortly after initiating sleep or shortly before waking. Secondary generalizing seizures occur in about 20% of cases and typically occur later in the sleep cycle. Partial seizures have a tendency to occur at the beginning or end of the sleep cycle.

The characteristic EEG pattern of BRE involves frequent to occasional unilateral or bilateral centrotemporal (rolandic) sharp and slow waves that are superimposed on a normal EEG background. Interictal activity can be present while the patient is awake and is markedly activated during NREM sleep. The rolandic sharp and slow waves have a voltage dipole that is negative in the rolandic and sylvian fissures and positive in the frontal head regions.

Treatment

BRE is associated with spontaneous remission of seizures, regardless of whether the patient is treated with an antiepileptic medication. The decision to treat a child with BRE is often based upon the impact of the seizures on quality of life. Because the seizures typically occur infrequently and most commonly during sleep, many clinicians chose not to initiate treatment. If the events are frequent and potentially frightening to the child or parent, treatment with an antiepileptic medication may be

considered. However, there is currently no formal consensus on the best treatment for BRE. Carbamazepine is likely the medication most commonly used, particularly due to the side effects associated with phenobarbital and phenytoin. Valproic acid or gabapentin may also be helpful. In most cases, polytherapy should be avoided. There is currently no information about whether treatment with an antiepileptic medication can help avoid or minimize the neurocognitive difficulties associated with BRE.

Cross References

- ▶ Carbamazepine
- ▶ Gabapentin
- ▶ Partial Seizure (Simple)
- ▶ Valproate

References and Readings

Aicardi, J. (1998). *Diseases of the nervous system in childhood.* London: Mac Keith.

Ferrie, C. D., Nordli, D. R., & Panayiotopoulos, C. P. (2008). Benign focal epilepsies of childhood. In J. M. Pellock, B. F. D. Bourgeois, & W. E. Dodson (Eds.), *Pediatric epilepsy: Diagnosis and therapy* (3rd ed., pp. 335–350). New York: Demos.

Loiseau, P. (2001). Idiopathic and benign partial epilepsies. In E. Wyllie (Ed.), *The treatment of epilepsy: Principles and practice* (3rd ed., pp. 475–484). Philadelphia: Lippincott Williams & Wilkins.

Northcutt, E., Connolly, A. M., Berroya, A., Sabaz, M., McIntyre, J., Christie, J., et al. (2005). The neuropsychological and language profile of children with benign rolandic epilepsy. *Epilepsia, 46*, 924–930.

Piccinelli, P., Borgatti, R., Aldini, A., Bindelli, D., Ferri, M., Perna, S., et al. (2008). Academic performance in children with rolandic epilepsy. *Developmental Medicine and Child Neurology, 50*, 353–356.

Benign Senescent Forgetfulness

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Synonyms

Age-associated memory impairment (AAMI); Late-life forgetfulness

Definition

The term "benign senescent forgetting" was coined by V.A. Kral (see Kral, 1962) to describe an age-related memory decline that is distinct from memory impairment due to known neurological damage or disease.

Current Knowledge

Changes in cognitive functioning are prevalent in aging populations. It has become clear that there is most likely a continuum between normal and abnormal mental function in those individuals who will ultimately develop dementia. Recent studies focusing on the characterization of the earliest stages of cognitive impairment have identified an intermediate period between the cognitive changes of normal aging and dementia (see Petersen, Stevens, Ganguli, Tangalos, Cummings, & DeKosky, 2001). This transitional zone has been described using a variety of terms, including benign senescent forgetfulness (BSF), age-associated memory impairment (AAMI), age-associated cognitive decline (ACCD), cognitive impairment-no dementia (CIND), and, most recently, mild cognitive impairment (MCI). AAMI differs from BSF in that it includes specific memory test performance criteria of 1 SD below young-adult levels (see Larrabee & Crook, 1994). AACD expands the definition to decrements in performance in other cognitive domains. MCI further refined the definition to include the presence of memory complaints, normal activities of daily living, normal global cognitive functioning, but abnormal memory performance compared to age- and education-matched controls (see Smith & Rush, 2006). The clinical concept of MCI is important because it is a significant risk factor for dementia. While conversion rates vary widely, most researchers estimate that individuals with MCI develop dementia at a rate of 10–15% per year, in contrast to the rate of 1–2% per year for age-matched controls.

Cross References

- ▶ Age Decrements
- ▶ Mild Cognitive Impairment
- ▶ Normal Aging

References and Readings

Kral, V. A. (1962). Senescent forgetfulness: Benign and malignant. *Journal of the Canadian Medical Association, 86*, 257–260.

Larrabee G. J., & Crook, T. H. (1994). Estimated prevalence of age-associated memory impairment derived from standardized tests of memory function. *International Psychgeriatrics, 6*, 95–104.

Petersen, R. C., Stevens, J. C., Ganguli, M., Tangalos, E. G., Cummings, J. L., & DeKosky, S. T. (2001). Practice parameter: Early detection of dementia: Mild cognitive impairment (an evidence-based review). Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology, 56*, 1133–1142.

Smith, G., & Rush, B. K. (2006). Normal aging and mild cognitive impairment. In D. K. Attix & K. A. Welch-Bohmer (Eds.), *Geriatric neuropsychology assessment and intervention* (pp. 27–56). New York: Guilford.

Benton, Arthur (1909–2006)

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Landmark Clinical, Scientific, and Professional Contributions

- Arthur Benton was one of the pioneering figures in clinical neuropsychology. Beginning in the 1940s, he introduced and applied novel and objective assessment techniques that provided a basis for fundamental brain-behavior studies in aphasia, visuospatial abilities, hemispheric specialization, and other cognitive processes. Through the development of standardized tasks that stressed specific abilities, together with collection of data from neurological patients and normal comparison subjects, he was able to bring increased reliability and sensitivity to the mental status exam, helping to establish neuropsychology as a valuable clinical entity. He developed a number of neuropsychological tests that have been in wide use in clinical and research settings worldwide for several decades, including the Visual Retention Test, Judgment of Line Orientation, Three-Dimensional Block Construction, and Facial Recognition. He advocated a flexible approach to clinical assessment, with the content and scope of testing determined by the referral question, context, and patient abilities.

Education and Training

He received his B.A. and M.A. degrees from Oberlin College, and completed his Ph.D. at Columbia University in 1935 under the mentorship of Carney Landis, followed by clinical training at the Payne Whitney Psychiatric Clinic of New York Hospital.

Major Appointments

- Dr. Benton volunteered for military service in the US Navy in 1941, and was commissioned as a lieutenant in the medical department. His active duty ended in 1945, but he continued to serve in the US Navy Reserve for many years, eventually retiring at the rank of captain. In 1946, he accepted a position in the Psychology Department at the University of Louisville. In 1948, he became a professor at the University of Iowa, where he would remain for over 50 years. He initially was appointed Professor and Director of Graduate Training in Clinical Psychology, and then accepted a joint appointment in the departments of Psychology and Neurology in 1958. He officially retired in 1978, but remained active in research, teaching, and other professional activities for another 20 years.

Major Honors and Awards

- President, American Orthopsychiatric Association, 1965
- President, International Neuropsychological Society, 1970
- Secretary-General, Research Group on Aphasia of the World Federation of Neurology, 1971–1978
- Distinguished Professional Contribution Award, American Psychological Association, 1978
- Outstanding Scientific Contribution Award, International Neuropsychological Society, 1981
- Samuel Torrey Orton Award, Orton Dyslexia Society, 1982
- Distinguished Service and Outstanding Contribution Award, American Board of Professional Psychology, 1985
- Distinguished Clinical Neuropsychologist Award, National Academy of Neuropsychology, 1989
- Gold Medal Award for Life Achievement in the Application of Psychology, American Psychological Foundation, 1992

Biography

Arthur Benton was born in New York City on October 16, 1909. Educated at Oberlin and Columbia, he was a great historian who could trace his academic lineage to the earliest psychologists. During his military assignment to the San Diego Naval Hospital prior to beginning his academic career, he worked with neurologist Morris Bender and examined servicemen with traumatic brain injury. This experience helped convince him of the value of standardized clinical tests, and led to the development of the Benton Visual Retention Test.

During his first academic appointment at the University of Louisville, Benton cowrote with Spafford Ackerly the seminal paper on childhood-onset damage to prefrontal cortex. This detailed neuropsychological and neuroanatomical study of a single patient dispelled the notion that early damage to the brain was always followed by good recovery, and presaged later work illuminating the prefrontal cortex as a critical region underlying social and emotional behavior.

In 1948, Benton began his long career at the University of Iowa when he took the position of Professor and Director of Graduate Training in Clinical Psychology. Two years later, A. L. Sahs, Chairman of the Department of Neurology at the University of Iowa Hospitals and Clinics invited him to set up a laboratory in the hospital for the purpose of studying behavioral impairments related to brain disease, a move strongly supported by Drs. Russell Meyers (Chairman of the Division of Neurosurgery) and Maurice Van Allen (Iowa City VA Hospital). From its inception, his neuropsychology program was dedicated to the tripartite goals of scientific investigation, patient care, and student training, united by a focus on developing objective psychological measures for the impairments resulting from brain dysfunction. The beginnings of the program were quite humble, with the original neuropsychology unit being housed in a windowless $5' \times 6'$ room shared with the Department of Urology, which utilized it for "special purposes."

Benton, Arthur (1909–2006). Figure 1

The laboratory rapidly expanded, and with access to the high volume of neurological patients at the University Hospitals and other nearby institutions, Benton and his students systematically approached each of the primary domains of cognition, devising and validating tests of language, memory, attention, visual perception, visuomotor abilities, auditory recognition, tactile perception, body schema, and more. The enduring value of their empirical approach is reflected in the fact that several of these tests remain in the batteries of most neuropsychologists today.

Benton advocated a hypothesis-testing approach to neuropsychological evaluation. According to this flexible approach, hypotheses regarding the patient's condition would arise from behavioral observations, the patient's history, and performances on an initial brief battery of tests. These hypotheses would then be tested with subsequent targeted behavioral tests. "I think that we should regard neuropsychological assessment in the same way as we view the physical or neurological examination, i.e., as a logical, sequential decision-making process rather than as simply the administration of a fixed battery of tests" (Benton, 1985). He was a strict empiricist and did not hesitate to challenge popular beliefs if his data indicated otherwise. Perhaps best known was his characterization of the Gerstmann syndrome as ". . . a fiction; it is simply an artifact of defective and biased observations" (1961), based on his systematic observation that the components of the Gerstmann syndrome did not co-occur with one another any more than with deficits not considered part of the syndrome.

Benton was instrumental in bringing together the international neuropsychological community. He used his knowledge of French, German, and Italian to translate and bring to attention reports of neurological syndromes that had been largely overlooked because they were published in languages other than English. He was a visiting scholar at the University of Milan (1964), the Neurosurgical Clinic, Hospital Sainte-Anne, Paris (1968), the Hebrew University Medical School, Jerusalem (1969), the Free University of Amsterdam (1971), the University

of Helsinki (1974), the Tokyo Metropolitan Institute of Gerontology (1974), the University of Melbourne (1977), L'Ecole des Hautes Etudes, Paris (1979), the University of Victoria, British Columbia (1980), the University of Minnesota Medical School (1980), and the University of Michigan (1986).

In the context of all of his professional accomplishments, Dr. Benton's dedication to education in neuropsychology was perhaps his greatest contribution. During neuropsychology's formative years, he was instrumental in developing training standards for the field. At the first scientific session of the INS, held in Washington D.C. in 1967, he moderated an afternoon symposium on the development of a comprehensive training program in neuropsychology, and he remained active in refining these standards over the years. At the University of Iowa, he supervised 46 doctoral dissertations and 24 master's theses, and he provided consultation to leading neuropsychology centers around the world. He was known for supervision characterized by frankly honest feedback, often bruising to the student's ego, but always accompanied by sage guidance for improving the situation.

Dr. Benton officially retired in 1978, at which time the Benton Laboratory of Neuropsychology in the Department of Neurology was dedicated. His retirement was incomplete, however, as he continued to provide guidance for the neuropsychologists at Iowa and elsewhere and continued writing for more than another 2 decades. Today, the Benton Neuropsychology Laboratory at the University of Iowa Department of Neurology remains a vital program for research, training, and patient care, in the tradition established by Dr. Benton more than a half century ago.

Benton's wife, Rita, was a professor of musicology at the University of Iowa, where she was the first head of the Music Library in 1957. Arthur and Rita met in 1939 while they both were vacationing in Paris, and they married later that year. Upon Rita Benton's death in 1980, the Music Library was named in her honor. They had three children: Raymond, Abigail, and Daniel. Arthur Benton died in Glenview, Illinois on December 27, 2006 from complications of emphysema, at the age of 97.

Cross References

- ▶ ABPP
- ▶ APA
- ▶ Aphasia
- ▶ Benton Visual Retention Test
- ▶ Clinical Neuropsychology
- ▶ Facial Recognition Test
- ▶ Flexible Battery
- ▶ Frontal Lobes
- ▶ Gerstmann's Syndrome
- ▶ Hemispheric Specialization
- ▶ Hypothesis Testing Approach (to Evaluation)
- ▶ Judgment of Line Orientation
- ▶ Mental Status Examination
- ▶ Multilingual Aphasia Examination
- ▶ National Academy of Neuropsychology (NAN)
- ▶ Standardized Tests

References and Readings

Ackerly, S. S., & Benton, A. L. (1948). Report of a case of bilateral frontal lobe defect. *The Frontal Lobes; Proceedings of the Association for Research in Nervous and Mental Disease 1947*, 27 (pp. 479–504). Baltimore: Williams and Wilkins.

Benton, A. L. (1945). A visual retention test for clinical use. *Archives of Neurology and Psychiatry, 54*, 212–216.

Benton, A. L. (1955). Right-left discrimination and finger-localization in defective children. *Archives of Neurology and Psychiatry, 74*, 383–389.

Benton, A. L. (1956). The concept of pseudofeeblemindedness. *Archives of Neurology and Psychiatry, 75*, 379–388.

Benton, A. L. (1960). Motivational influences on performances in brain-damaged patients. *American Journal of Orthopsychiatry, 30*, 313–321.

Benton, A. L. (1961). The fiction of the "Gerstmann Syndrome." *Journal of Neurology, Neurosurgery, and Psychiatry, 24*, 176–181.

Benton, A. L. (1962). Behavioral indices of brain injury in school children. *Child Development, 33*, 199–208.

Benton, A. L. (1964). Contributions to aphasia before Broca. *Cortex, 1*, 314–327.

Benton, A. L. (1964). Developmental aphasia and brain damage. *Cortex, 1*, 40–52.

Benton, A. L. (1967). Problems of test construction in the field of aphasia. *Cortex, 3*, 32–58.

Benton, A. L. (1969). Development of a multilingual aphasia battery: Progress and problems. *Journal of Neurological Sciences, 9*, 39–48.

Benton, A. L. (1977). Interactive effects of age and brain disease on reaction time. *Archives of Neurology, 34*, 369–370.

Benton, A. L. (1985). Some problems associated with neuropsychological assessment. *Bulletin of Clinical Neurosciences, 50*, 11–15.

Benton, A. L., & Fogel, M. L. (1962). Three-dimensional constructional praxis. *Archives of Neurology, 7*, 347–354.

Benton, A. L., Hamsher, K. deS., Varney, N. R., & Spreen, O. (1983). *Contributions to neuropsychological assessment.* New York: Oxford University Press.

Benton, A. L., & Howell, I. L. (1941). The use of psychological tests in the evaluation of intellectual function following head injury. *Psychosomatic Medicine, 3*, 138–151.

Benton, A. L., & Van Allen, M. W. (1968). Impairment in facial recognition in patients with unilateral cerebral disease. *Cortex, 4*, 344–358.

Levin, H. S., & Benton, A. L. (1975). Temporal orientation in patients with brain disease. *Applied Neurophysiology, 38*, 56–60.

Benton Face Recognition Test

► Facial Recognition Test

Benton Faces

► Facial Recognition Test

Benton Test

► Benton Visual Retention Test

Benton Visual Form Discrimination Test

► Visual Form Discrimination

Benton Visual Retention Test

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Synonyms

Benton Test; BVRT

Description

The Benton Visual Retention Test (BVRT) is a widely used test of visual memory, visual perception, and/or visual construction. Now in its fifth edition (Sivan, 1992), the test consists of three equivalent forms (Forms C, D, and E), each composed of 10 items of visual stimuli. Most items include three geometric forms presented along a horizontal plane, making the test particularly sensitive to visual neglect (Sivan, 1992).

The following description of the BVRT was adapted from Strauss, Sherman, and Spreen (2006). The test includes four alternative methods of administration (A, B, C, and D) that assess different aspects of functioning. The most common administration (A) assesses immediate recall of a visual display. After presenting a stimulus card for 10 s, the card is removed, and the examinee is asked to draw the design from memory. Administration B follows the same procedure as A, but with a 5-s exposure interval. Administration C allows the examiner to dissociate memory functioning from perceptual and motor aspects of the task by asking the examinee to reproduce the designs while each item is in plain view. There is no time limit, but individuals who work very slowly should be encouraged to increase their speed. In Administration D, a 15-s interval is inserted between the 10-s encoding phase and the figure reproduction, allowing the examiner to assess short-term retention of visual information. Scoring consists of both the number of correct designs and the number of six different types of errors: omissions, distortions, perseverations, rotations, misplacements, and size errors. Administration time for each form is approximately 5 min. Several sets of norms are available and reflect different demographic characteristics, including age ranges and education levels (Mitrushina, Boone, Razani, & D'Elia, 2005; Strauss et al., 2006). A multiple-choice recognition administration (Administration M, with alternate forms F and G) is also sometimes used, particularly with patients who have visuoconstructional and motor coordination deficits (Amieva, Gaestel, & Dartigues, 2006). For Administration M, the examinee views a target stimulus for 10 s and, after it has been removed, is required to identify it from among four choices. Although not part of the English-language version, materials for this special administration are available in the German (Sivan & Spreen, 1996) and French (Benton, 1965) editions.

Historical Background

Dr. Arthur L. Benton developed the Visual Retention Test as a brief measure of immediate nonverbal memory to

supplement the popular auditory digit span test in neuropsychological evaluations (Benton, 1945). It was first published in 1946. Memory-for-designs tasks had appeared earlier in the century as part of larger intelligence tests, but included only a few designs and did not have separate normative data. As an addition to the digit span test, the BVRT was intended to provide a broader assessment of short-term memory, and its format was selected for its resistance to emotional influence, employment of different sensorimotor components (graphomotor versus auditory-vocal), and minimal examiner-subject interaction (freedom from interpersonal demands). The initial version included seven cards and two parallel forms. A 1955 revision increased the number of designs and alternate forms, and added norms for children aged 8–16 years. Later editions included a design copy administration and updated norms. The most recent revision was authored by Abigail Benton Sivan in 1992 and is available from its publisher, Pearson Assessments (http://pearsonassess.com).

Psychometric Data

Information on reliability and validity may be found in the manual. Test-retest reliability is 0.85. Alternate form reliability ranges from 0.79 to 0.84. There is evidence that Form C is slightly less difficult than Forms D and E under Administration A. Correlations between immediate (Administration A) and delayed (Administration D) recall are positive and range from 0.40 to 0.83, depending on the combination of forms used. Construct validity has been demonstrated through moderate correlations (0.46–0.62) of the BVRT with nonverbal subtests from the Wechsler Adult Intelligence Scales, including Block Design, Digit Symbol, and Object Assembly.

Child and adolescent normative data are included for Administrations A and C. The normative data for each method of administration are based on different standardization samples, and sample characteristics are provided for Administrations A, B, and C. (Normative data for Administration D are not included in the manual.) The standardization sample for Administration A is based on a compilation of three separate studies totaling over 1,300 participants, ranging in age from 8–69 years. (See manual for discussion of participant inclusion criteria for each of these studies.) The standardization sample for Administration B is based on 103 medical inpatients and outpatients, aged 16–60 years, with no evidence or history of brain disease. The standardization samples for Administration C are 200 medical patients with no history of brain disease for the adult norms, and 236 children, aged 6–13 years, enrolled in public schools in Iowa and Wisconsin, for the child and adolescent norms.

Clinical Uses

As it recruits a number of different cognitive functions, the BVRT is sensitive to many forms of brain damage and disease; however, its ability to discriminate among diagnoses is low (for a review, see Mitrushina et al., 2005). An individual's global performance, quantified as either the number correct score or error score, provides the best indicator of impairment. According to the manual, measures of specific error types, such as omissions, perseverations, and distortions, are not by themselves diagnostic, but may raise hypotheses for further testing. For example, a high number of perseverative errors suggests possible frontal lobe damage, particularly if supported by other test and behavioral data. Omission of peripheral figures may raise suspicion of brain damage and is most frequently associated with left hemispatial neglect as a result of damage to right parietal lobe regions. In contrast, global performance has not been found to consistently distinguish between patients with unilateral right and left brain damage. Though the BVRT is sensitive to the visuospatial disturbance that is often observed in patients with right hemisphere damage, studies have shown that individuals with unilateral left hemisphere damage can exhibit similarly poor results on Administration A (Vakil, Blachstein, Sheleff, & Grossman, 1989), as well as on copy and multiple-choice administrations (Arena & Gainotti, 1978). This indicates that memory for the BVRT designs, many of which can be verbalized, is mediated by both hemispheres. However, the presence of a delay interval may differentially affect verbally and visually encoded material and therefore help to discriminate between left- and right-sided brain damage. Participants with right hemisphere damage achieved a lower total correct score on Administration D than on Administration A, whereas individuals with left hemisphere damage had the opposite pattern of performance, benefitting from the delay. In contrast, scores from healthy participants did not differ between the two administrations (Vakil et al., 1989).

Both copy and memory administrations are highly sensitive to early dementia, and may also help to identify individuals who are at risk for developing dementia in the future. In one such study, participants with six or more errors on Administration A were nearly twice as likely to develop Alzheimer's Disease 10–15 years later, when compared to participants who had fewer errors (Kawas et al.,

2003). The BVRT also aids in identifying children with a learning disability and discriminating among types of learning disabilities, with reading deficits associated with the lowest levels of performance (Snow, 1998). Children with Attention-Deficit/Hyperactivity Disorder receiving stimulant medication have also been shown to perform more poorly on the BVRT than healthy participants (Risser & Bowers, 1993). Poorer performance is evident in a subset of patients with schizophrenia and may result, at least in part, from abnormal patterns of visual scanning and fixation related to deficient attention (Obayashi, Matsushima, Ando, Ando, & Kojima, 2003). The BVRT may also be useful in detecting malingering, which has been characterized by a greater number of errors, particularly distortion ones, than seen in neuropsychologically impaired patients (Benton & Spreen, 1961; Suhr, Tranel, Wefel, & Barrash, 1997).

In evaluating results, it is important to consider that the BVRT may be sensitive to individual differences that do not necessarily reflect neuropathology. Stratified normative data confirm that age is negatively correlated and that baseline intellectual functioning is positively correlated with the BVRT number correct score. The association with baseline intellect is strongest in the lower than average IQ ranges. Education-stratified norms are also available, and indicate a positive relationship between years of education and the number correct score (Strauss et al., 2006). The BVRT is used worldwide, and normative data have been published from more than a dozen countries (Mitrushina et al., 2005). While relatively few in number, studies involving direct cross-cultural comparisons demonstrate generally good consistency; however, caution is recommended when testing individuals with very low levels of education (Mitrushina et al., 2005). Comparisons of elderly people living in the US and Canada suggest similar levels of performance when education and/or reading ability are controlled. Results from a large Columbian sample of school-aged children did not differ from North American norms (Rosselli, Ardila, Bateman, & Guzman, 2001), suggesting that when educational quality is similar, as is increasingly more common in developed countries, cross-cultural differences, if present, are relatively small. Most studies have shown no gender differences.

Cross References

- ▶ Short Term Memory
- ▶ Visual-Motor Function
- ▶ Visuoperceptual
- ▶ Wechsler Memory Scale

References and Readings

Amieva, H., Gaestel, Y., & Dartigues, J. (2006). The multiple-choice formats (Forms F and G) of the Benton Visual Retention Test as a tool to detect age-related memory changes in population-based studies and clinical settings. *Nature Protocols, 1*, 1936–1938.

Arena, R., & Gainotti, G. (1978). Constructional apraxia and visuoperceptive disabilities in relation to laterality of lesions. *Cortex, 14*, 463–473.

Benton, A. L. (1945). A visual retention test for clinical use. *Archives of Neurology and Psychiatry, 54*, 212–216.

Benton, A. L. (1965). *Manuel pour l'application du Test Rêtention Visuelle.* Paris, France: Les Editions du Centre de Psychologie Appliquée.

Benton, A. L. (1974). *Revised Visual Retention Test: Clinical and experimental applications* (4th ed.). New York: Psychological Corporation.

Benton, A. L., & Spreen, O. (1961). Visual Memory Test: The simulation of mental incompetence. *Archives of General Psychiatry, 4*, 79–83.

Kawas, C. H., Corrada, M. M., Brookmeyer, R., Morrison, A., Resnick, S. M., Zonderman, A. B., et al. (2003). Visual memory predicts Alzheimer's disease more than a decade before diagnosis. *Neurology, 60*, 1089–1093.

Mitrushina, M. N., Boone, K. B., Razani, J., & D'Elia, L. F. (2005). *Handbook of normative data for neuropsychological assessment* (2nd ed.). New York: Oxford University Press.

Obayashi, S., Matsushima, E., Ando, H., Ando, K., & Kojima, T. (2003). Exploratory eye movements during the Benton Visual Retention Test: Characteristics of visual behavior in schizophrenia. *Psychiatry and Clinical Neurosciences, 57*, 409–415.

Risser, M. G., & Bowers, T. G. (1993). Cognitive and neuropsychological characteristics of attention deficit hyperactivity disorder children receiving stimulant medications. *Perceptual and Motor Skills, 77*, 1023–1031.

Rosselli, M., Ardila, A., Bateman, J. R., & Guzman, M. (2001). Neuropsychological test scores, academic performance, and developmental disorders in Spanish-speaking children. *Developmental Neuropsychology, 20*, 355–373.

Sivan, A. B. (1992). *Benton Visual Retention Test* (5th ed.). San Antonio, TX: Psychological Corporation.

Sivan, A. B., & Spreen, O. (1996). *Der Benton-Test* (7th ed.). Bern, Switzerland: Verlag Hans Huber.

Snow, J. H. (1998). Clinical use of the Benton Visual Retention Test for children and adolescents with learning disabilities. *Archives of Clinical Neuropsychology, 13*, 629–636.

Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (2nd ed.). New York: Oxford University Press.

Suhr, J., Tranel, D., Wefel, J., & Barrash, J. (1997). Memory performance after head injury: Contributions of malingering, litigation status, psychological factors, and medication use. *Journal of Clinical and Experimental Neuropsychology, 19*, 500–514.

Vakil, E., Blachstein, H., Sheleff, P., & Grossman, S. (1989). BVRT-scoring system and time delay in the differentiation of lateralized hemispheric damage. *International Journal of Clinical Neuropsychology, 11*, 125–128.

Ben-Yishay, Yehuda (1933–)

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Major Appointments

- 1967-present New York University Medical Center, Rusk Institute of Rehabilitation Medicine
- 1974–1976 Clinical Director of Israel Head Trauma Project, New York University Medical Center, Rusk Institute, and Israel Ministry of Defense Joint Research Project, Afeka, Israel.
- 1976–1983 Visiting Clinical Director, New York University Medical Center, Rusk Institute, and Israel Ministry of Defense Joint Research Project, Afeka, Israel.
- 1995–1997 Clinical Director, Kurt Goldstein Institute for Holistic Neuropsychological Rehabilitation Stravbing, Germany

Major Honors and Awards

- 1976 Howard A. Rusk Award for Outstanding Accomplishments in Rehabilitation
- 1982 William F. Caveness Award for Distinguished Contributions in the field of Head Injury, National Head Injury Foundation.
- 1988 Thomas J. Dean Award of Excellence in Head Injury Rehabilitation, Dallas, Rehabilitation Foundation.
- 1991 Distinguished Career Achievement Award, American Board of Medical Psychotherapists.
- 2006 Outstanding Life-Time Scientific Contributions to Rehabilitation Psychology. American Psychological Association, Division 22.

Landmark Clinical, Scientific, and Professional Contributions

- Dr. Ben-Yishay is the father of holistic brain injury rehabilitation. Initially developed in Israel for war veterans with head injuries and later transitioned to the New York University School of Medicine at the Rusk Institute, Dr. Ben-Yishay's treatment interventions with individuals with brain injuries combined contributions from neuropsychology, behavioral psychology, cognitive-behavioral psychotherapy, special education, social psychology, and psychodrama. He adapted these modalities to the needs and capabilities of his patients, systematically applying them in therapeutic community settings to reach maximal effectiveness. Through his holistic approach to the treatment of brain injury, a foundation for cognitive and neuropsychological rehabilitation was established.
- The holistic rehabilitation approach developed by Dr. Ben-Yishay includes a number of components in addition to traditional cognitive retraining, including: development of a therapeutic milieu or community, psychotherapy, regular involvement of family and caregivers, psychoeducation, and transitional work opportunities. Within the therapeutic milieu or community, persons with brain-injury not only participate in activities aimed at adaptation to and compensation for their deficits but also meet regularly with staff members to monitor their progress. Interaction with other individuals with brain injury is also an important part of the therapeutic milieu. During individual and group psychotherapy, persons with brain injury address the many adjustment issues associated with their deficits. In addition, the involvement of family and caregivers in the rehabilitation process not only provides additional support for the person with a brain injury as they complete therapies but also assists with the transition back to the community by providing realistic education and information regarding the person's progress and injury. Finally, transitional work opportunities provide important information regarding individuals' abilities outside of structured settings and help to provide additional functional goals for rehabilitation therapies.
- The work of Dr. Ben-Yishay has been researched and applied both within the United States and abroad, and premiere rehabilitation institutes around the world utilize his model of cognitive rehabilitation as the foundation for their own brain injury programs. His teaching methods are studied by students and professionals from all over the world, and he is internationally known as a clinician, teacher, researcher, and expert in the field of holistic rehabilitation.

Short Biography

Yehuda Bin-Yishay was born February 11, 1933 in Cluj, Romania. He grew up in Israel and served in the Israeli army. In 1957, he received a B.A. degree in Sociology and

Special Education from Hebrew University in Jerusalem, Israel. Then, in 1958, he came to the United States on a scholarship from the New School University in New York City. There, he studied under Kurt Goldstein. He completed an internship in Clinical Psychology in 1960 at Trenton State Hospital in Trenton, NJ. His Master's degree in Personality Psychology was completed in 1961.

After completing his Master's degree, Dr. Ben-Yishay served as the psychologist for an experimental program in the Department of Rehabilitation at the Albert Einstein College of Medicine in New York. The study tested the effectiveness of a "therapeutic community" model of rehabilitation.

In 1964, Dr. Ben-Yishay joined the faculty at New York University. He obtained his Ph.D. from New York University, following the completion of studies investigating the effects of normobaric oxygen on stroke patients' performances on neurologic, sensory-motor, and cognitive measures. While at New York University, Dr. Ben-Yishay's research over the next several years focused on three key areas: 1) rehabilitation outcome prediction studies; 2) comparisons between normal controls and brain injured individuals across a variety of measures; and 3) development and efficacy studies of cognitive rehabilitation modules.

From 1974–1977 Dr. Ben-Yishay conducted a pilot study in Israel to investigate the effects of holistic brain injury rehabilitation on Israeli war veterans. The results were impressive, and the study was followed in September of 1978 by the New York University-Rusk Head Trauma Program, a fully funded five-year research grant-based program.

Throughout his career, Dr. Ben-Yishay trained numerous rehabilitation neuropsychologists, who have gone on to institute his model of cognitive rehabilitation. Individuals such as Anne-Lise Christensen, Ph.D. and George Prigatano, Ph.D. have been greatly influenced by the work of Dr. Ben-Yishay and established programs built upon principles learned under his tutelage.

Dr. Ben-Yishay's work in the area of holistic brain injury rehabilitation has continued to the present day, and he has been recognized extensively for his work worldwide. He has receiving numerous awards and honors, including the 2006 Lifetime Scientific Contributions to Rehabilitation Psychology Award from Division 22 of the American Psychological Association.

In addition to the numerous international committee and consultant positions that he has held, Dr. Ben-Yishay has also served on a number of important editorial boards, including: *Archives of Physical Medicine and Rehabilitation, Journal of Head Trauma Rehabilitation, Brain Injury, and Neuropsychological Rehabilitation.*

Cross References

- ▶ Cognitive Rehabilitation
- ▶ Goldstein, Kurt (1878–1965)

References and Readings

Ben-Yishay, Y., & Diller, L. *A primer holistic neuropsychological rehabilitation.* New York, NY: Oxford University Press, In Press.

Prigatano, G., & Ben-Yishay, Y. (1999). Psychotherapy and psychotherapeutic intervention. In M. Rosenthal (Ed.), *Rehabilitation of the adult and child with traumatic brain injury* (3rd Ed.). Philadelphia: F.A. Davis.

Benzodiazepines

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Definition

Benzodiazepines belong to a class of medications known as sedative-hypnotics. They bind to the subtype A of the common inhibitory neurotransmitter substance, gamma-aminobutyric acid (GABA). They have wide ranging effects. Their popular use is reflected in their anxiolytic, muscle relaxant, sedative, anesthetic, and anticonvulsant properties. Due to their safety profile, benzodiazepines became very popular in the 1970s, replacing older drugs such as barbiturates and meprobamate for the treatment of anxiety symptoms, insomnia and other sleep disorders (Iversen, Iversen, Bloom & Roth, 2009), and alcohol withdrawal syndrome (Ntais, Pakos, Kyzas, & Ioannidis, 2005). However, negative effects of benzodiazepine use have also been reported. Although these "side effects" vary depending upon the original indication for benzodiazepine use, some of the unwanted effects include drowsiness, decreased concentration, memory impairment, psychomotor slowing (Buffett-Jerrott & Stewart, 2002), and postural instability (with increased risk of falls) among the elderly (Allain, Bentue-Ferrer, Polard, Akwa, & Patat, 2005). Chronic use also carries the risk of substance dependence and abuse, and cognitive impairment with prolonged use at high

doses (Stewart, 2005; Barker, Greenwood, Jackson, & Crowe, 2004). For these and other reasons, medications in this class are now more commonly used on a short-term rather than a long-term basis (Iversen et al., 2009).

Current Knowledge

As reported above, chronic use of benzodiazepines has declined, particularly for the treatment of anxiety disorders and insomnia. Tricyclic antidepressants (TCAs) and selective serotonergic agents are increasingly being prescribed over benzodiazepines for the treatment of anxiety disorders. For example, selective TCAs are reportedly as effective as benzodiazepines in the treatment of generalized anxiety disorder and certain selective serotonin reuptake inhibitors, and TCAs are effective in the treatment of panic and obsessive–compulsive disorders (Bourin & Lambert, 2002).

With respect to insomnia, benzodiazepines were the treatment of choice over barbiturates. However, negative effects such as the development of tolerance, residual daytime sleepiness, aggravation of respiratory conditions, and reduced duration of slow-wave (restorative) and REM sleep were also reported. Newer, nonbenzodiazepine hypnotic compounds such as zopiclone and zaleplon are also effective in treating insomnia, yet have fewer side effects than those of benzodiazepines (Montplaisir et al., 2003).

Cross References

► Barbiturates
► GABA

References and Readings

Allain, H., Bentu'e-Ferrer, D., Polard, E., Akwa, Y., & Patat, A. (2005). Postural instability and consequent falls and hip fractures associated with use of hynotics in the elderly. A comparative review. *Drugs in Aging, 22*, 749–765.

Barker, M. J., Greenwood, K. M., Jackson, M., & Crowe, S. F. (2004). Cognitive effects of long-term benzodiazepine use. A meta-analysis. *CNS Drugs, 18*, 37–48.

Bourin, M., & Lambert, O. (2002). Pharmacotherapy of anxious disorders. *Human Psychopharmacology: Clinical and Experimental, 17*, 383–400.

Buffett-Jerrott, S. E., & Stewart, S. H. (2002). Cognitive and sedative effects of benzodiazepine use. *Current Pharmaceutical Design, 8*, 45–58.

Iversen, L. L., Iversen, S. D., Bloom, F. E., & Roth, R. H. (2009). *Antidepressants and anxiolytics. Introduction to neuropsychopharmacology* (pp. 306–335). New York: Oxford University Press.

Montplaisir, J., Hawa, R., Moller, H., Morin, C., Fortin, M., Matte, J., Reinish, L., & Shapiro, C. M. (2003). Zopiclone and zaleplon vs benzodiazepiones in the treatment of insomnia: Canadian consensus statement. *Human Psychopharmacology: Clinical and Experimental, 18*, 29–38.

Ntais, C., Pakos, E., Kyzas, P., & Ioannidis, J. P. (2005). Benzodiazepines for alcohol withdrawal. *Cochrane Database of Systematic Reviews, 20*, CD005063.

Berg Balance Scale

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Synonyms

BBS; 7-item BBS-3P

Description

The Berg Balance Scale (BBS) is a 14-item performance observation measure that assesses balance on a scale from 0 to 4 for each item, yielding a total score range of 0–56, where higher scores indicate better balance. The BBS tests both static and dynamic balance with items meant to mimic balance challenges encountered in daily life.

Historical Background

In 1989, Berg developed the BBS to fill the need for a quantitative balance assessment tool to screen older adults for fall risk. The BBS has subsequently become the best known clinical balance instrument. Shorter versions of the BBS, such as the seven-item BBS-3P (which also has a condensed rating scale) have also been developed and validated.

Psychometric Data

The high reliability, validity, and sensitivity of the BBS, including predictive validity for fall risk, are well documented in the literature. Some authors initially dichotomized the scale, using the threshold value <45 points as an indication of fall risk. However, more rigorous study

has determined that a gradient of fall risk exists over the entire scale. A retrospective study of community-dwelling persons with stroke demonstrated that changing from 3 to 4 for the "standing on one leg" item had a sensitivity of 0.90 and a specificity of 0.50 for predicting the history of multiple falls.

Clinical Uses

The BBS is available online (Internet Stroke Center, 2007). Administration requires 10–20 min, a chair, a step, a ruler, and a stopwatch. Balance ability is sometimes grossly categorized as good, fair, or poor for score ranges from 56 to 41, 40–21 and 20–0, respectively. As stated above, a gradient of fall risk exists over the entire scale. BBS scores are used when prescribing mobility aids and treatment interventions, identifying safe and unsafe activities, and to measure treatment effect. When assessing the treatment effect for individual patients with stroke, a score change of 6 points has been shown to represent real change, beyond measurement error, with 90% confidence.

Although originally designed to screen older adults for fall risk, the BBS has subsequently been validated for persons with stroke, multiple sclerosis, and Parkinson's Disease.

Cross References

- ▶ Balance Disorders
- ▶ Multiple Sclerosis
- ▶ Parkinson's Disease
- ▶ Reliability
- ▶ Sensitivity
- ▶ Specificity
- ▶ Stroke

References and Readings

Alzayer, L., Beninato, M., & Portney, L. G. (2009). The accuracy of individual berg balance scale items compared with the total berg score for classifying people with chronic stroke according to fall history. *Journal of Neurologic Physical Therapy: JNPT, 33*(3), 136–143.

Berg, K., Wood-Dauphinee, S., & Williams, J. I. (1995). The balance scale: Reliability assessment with elderly residents and patients with an acute stroke. *Scandinavian Journal of Rehabilitation Medicine, 27*(1), 27–36.

Berg, K., Wood-Dauphinee, S., Williams, J. I., & Gayton, D. (1989). Measuring balance in the elderly: Preliminary development of an instrument. *Physiotherapy Canada, 41*, 304–311.

Blum, L., & Korner-Bitensky, N. (2008). Usefulness of the berg balance scale in stroke rehabilitation: A systematic review. *Physical Therapy, 88*(5), 559–566.

Chou, C. Y., Chien, C. W., Hsueh, I. P., Sheu, C. F., Wang, C. H., & Hsieh, C. L. (2006). Developing a short form of the berg balance scale for people with stroke. *Physical Therapy, 86*(2), 195–204.

Internet Stroke Center (2007). *Berg balance scale.* http://www.strokecenter.org/Trials/scales/berg.html. Accessed May 19, 2010.

Mao, H., Hsueh, I., Tang, P., Sheu, C., & Hsieh, C. (2002). Analysis and comparison of the psychometric properties of three balance measures for stroke patients. *Stroke, 33*(4), 1022–1027.

Muir, S. W., Berg, K., Chesworth, B., & Speechley, M. (2008). Use of the berg balance scale for predicting multiple falls in community-dwelling elderly people: A prospective study. *Physical Therapy, 88*(4), 449–459.

Stevenson, T. J. (2001). Detecting change in patients with stroke using the berg balance scale. *Australian Journal of Physiotherapy, 47*, 29–38.

Tyson, S. F., & Connell, L. A. (2009). How to measure balance in clinical practice. A systematic review of the psychometrics and clinical utility of measures of balance activity for neurological conditions. *Clinical Rehabilitation, 23*(9), 824–840.

Berger's Waves

▶ Alpha Rhythm

Best Performance Method

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Synonyms

Cognitive potential

Definition

Neuropsychologists typically do not have an opportunity to evaluate patients before the onset of neurological illness or injury. Judgments about impairment are often made by comparing obtained test scores with estimates of premorbid ability. There are several approaches to estimating premorbid level of ability. One such approach is the Best Performance Method. Using this method, data are collected from multiple sources, including, but not limited to, test scores, observations, interviews, reports from family, and historical data. After the data are collected, the data source that yields the highest level of functioning is the set standard to which all other aspects of functioning are

compared. The Best Performance Method assumes that one performance level exists for each person's cognitive abilities. A notable discrepancy between a patient's best and other performances is indicative of neuropsychological impairment. The Best Performance Method also assumes that performance should be consistent across all areas of functioning. For example, very superior intellectual and other abilities would be expected from a patient who has earned a doctoral degree in engineering. The method has been criticized by some who believe that there is a high likelihood of overestimating premorbid ability.

Cross References

- ▶ Deficit Measurement
- ▶ Premorbid Estimate
- ▶ Premorbid Functioning
- ▶ Premorbid Intelligence

References and Readings

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). The rationale of deficit measurement. In *Neuropsychological Assessment* (4th ed., pp. 86–99). New York: Oxford University Press.

Mortensen, E. L., Gade, A., & Reinisch, J. M. (1991). "Best Performance Method" in clinical neuropsychology. *Journal of Clinical and Experimental Neuropsychology, 13*, 361–371.

Beta-Interferons

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Synonyms

Avonex®; Betaseron®; Rebif®

Definition

Interferon β is a disease-modifying drug currently indicated for treatment of relapsing forms of multiple sclerosis. Its mechanism of action is complex and is presumed to inhibit immune system T-cell activation and migration into the central nervous system as well as modulate the action of some pro-inflammatory proteins (cytokines). There are three FDA approved beta interferons available in the US – Avonex® (INF-β1a), Betaseron® (INF-β1b), and Rebif® (INF-β1a). These medications are administered via injection, and each has been shown to reduce the frequency of MS relapses, reduce MRI evidence of brain lesions, and possibly reduce disability progression.

Cross References

- ▶ Multiple Sclerosis

References and Readings

National Clinical Advisory Board of the National Multiple Sclerosis Society. (2007). *Disease management consensus statement*. Retrieved February 15, 2010, from http://www.nationalmssociety.org/download.aspx?id=8

Noseworthy, J., Miller, D., & Compston, A. (2006). Disease-modifying treatments in multiple sclerosis. In A. Compston, C. Confavreux, H. Lassman, I. McDonald, D. Miller, J. Noseworthy, et al. (Eds.), *McAlpine's multiple sclerosis* (4th ed., pp. 729–802). Philadelphia: Elsevier.

Zhang, J., Hutton, G., & Zang, Y. (2002). A comparison of the mechanisms of action of interferon beta and glatiramer acetate in the treatment of multiple sclerosis. *Clinical Therapeutics, 24*, 1998–2021.

Betaseron®

- ▶ Beta-Interferons

Between-session Assignments

- ▶ Homework

Beyond a Reasonable Doubt

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Definition

Beyond a reasonable doubt is the standard of proof required in most criminal cases within an adversarial system. Generally, the prosecution bears the burden of proof and is required to prove their version of events to this standard. This means that the proposition being presented by the prosecution must be proven to the extent that there is no "reasonable doubt" in the mind

of a reasonable person that the defendant is guilty. There can still be a doubt, but only to the extent that it would not affect a "reasonable person's" belief regarding whether or not the defendant is guilty. The "shadow of a doubt" is sometimes used interchangeably with reasonable doubt, but this extends beyond the latter to the extent many believe is an impossible standard. Reasonable doubt is therefore used. If doubt affects a "reasonable person's" belief that the defendant is guilty, the jury is not satisfied beyond a "reasonable doubt." The precise meaning of words such as "reasonable" and "doubt" is usually defined within jurisprudence of the applicable country.

The standard that must be met by the prosecution's evidence in a criminal prosecution is that no other logical explanation can be derived from the facts except that the defendant committed the crime, thereby overcoming the presumption that a person is innocent until proven guilty. If the jurors or judge have no doubt as to the defendant's guilt, or if their only doubts are unreasonable doubts, then the prosecutor has proven the defendant's guilt beyond a reasonable doubt and the defendant should be pronounced guilty. The term "reasonable doubt" connotes that evidence establishes a particular point to a moral certainty and that it is beyond dispute that any reasonable alternative is possible. It does not mean that no doubt exists as to the accused's guilt, but only that no reasonable doubt is possible from the evidence presented.

Beyond a reasonable doubt is the highest standard of proof that must be met in any trial. In civil litigation, the standard of proof is either proof by a "preponderance of the evidence" or proof by "clear and convincing evidence." These are lower burdens of proof. A preponderance of the evidence simply means that one side has more evidence in its favor than the other, even by the smallest degree. Clear and convincing proof is evidence that establishes a high probability that the fact sought to be proven is true. The main reason that the high-proof standard of reasonable doubt is used in criminal trials is that such proceedings can result in the deprivation of a defendant's liberty or even in his or her death. These outcomes are far more severe than in civil trials, in which money damages are the common remedy.

Cross References

- ▶ Burden of Proof
- ▶ Clear and Convincing Evidence
- ▶ Preponderance of the Evidence

References and Readings

Denney, R. L. (2005). Criminal forensic neuropsychology and assessment of competency. In G. Larrabee (Ed.), *Forensic neuropsychology: A scientific approach.* New York: Oxford University Press.

Melton, G. B., Petrila, J., Poythress, N. G., & Slobogin, C. (2007). *Psychological evaluations for the courts: A handbook for mental health professionals and lawyers* (3rd ed.). New York: Guilford.

BG-II

- ▶ Bender Visual–Motor Gestalt Test II

BHS

- ▶ Personality Inventory for Children

BI

- ▶ Barthel Index

BIAA

- ▶ Brain Injury Association of America

Bias

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Synonyms

Partiality; Prejudice

Definition

Faust, Ziskin, and Hiers (1991) and Wedding and Faust (1989) explain chief forms of bias related to clinical

judgment and decision making in neuropsychology. First, *hindsight bias* is the tendency to believe, after the outcome of an incident is determined, that the outcome could have been more reliably predicted than is actually true. This form of bias suggests that being aware of an event via a client's clinical history may lead the clinician to conclude that they can determine the outcome of the event and make diagnostic determinations. *Confirmation bias* refers to the tendency to seek confirming evidence while failing to consider disconfirming evidence when generating diagnostic impressions. Thus, a clinician seeks to confirm initial hypotheses, while failing to gather information related to alternative hypotheses. Moreover, it has been demonstrated that clinicians tend to stop hypothesis evaluation once information in support of an initial hypothesis has been gathered, thus potentially terminating the evaluation prior to adequate consideration of competing hypotheses.

To combat against bias in neuropsychological assessment and testimony, Wedding and Faust (1989) and Sweet and Moulthrop (1999) provided a number of strategies for clinicians to consider when testifying and preparing reports. First and foremost, they recommended that clinicians be familiar with the scientific literature regarding human judgment and decision making. Moreover, they recommend that clinicians begin with consideration of the most valid information, generating alternative diagnostic hypotheses, and then gathering and considering evidence for each and providing an outline of disconfirmatory information. Thus, in the context of a neuropsychological evaluation, it is recommended that clinicians generate a list of test findings that support specific hypotheses, but also list data that disputes such hypotheses. Larrabee (2000) suggests a four-component consistency analysis for neuropsychological decision making, including asking the following four questions: (a) Are the data consistent within and between neuropsychological domains? (b) Is the neuropsychological profile consistent with the suspected etiologic condition? (c) Are the neuropsychological data consistent with the documented severity of injury? and (d) Are the neuropsychological data consistent with the subject's behavioral presentation? Several pieces of data must be analyzed in order to address the aforementioned questions: comprehensive interview, meticulous record review, and comprehensive and redundant neuropsychological tests within each domain (language, perception, sensorimotor functioning, attention/information processing, psychomotor speed, verbal and visual learning and memory, intelligence, problem solving, motivation, and personality).

References and Readings

Faust, D., Ziskin, J., & Hiers, J. B., Jr. (1991). *Brain damage claims: Coping with neuropsychological evidence.* Marina del Rey, CA: Law & Psychology Press.

Larrabee, G. J. (2000). Forensic neuropsychological assessment. In R. D. Vanderploeg (Ed.), *Clinicians guide to neuropsychological assessment* (2nd ed., pp. 301–335). Mahwah, NJ: Lawrence Erlbaum.

Sweet, J. J., & Moulthrop, M. A. (1999). Self-examination questions as a means of identifying bias in adversarial assessments. *Journal of Forensic Neuropsychology, 1*, 73–88.

Van Gorp, W., & McMullen, W. (1997). Potential sources of bias in forensic neuropsychological evaluations. *The Clinical Neuropsychologist, 11*, 180–187.

Wedding, D., & Faust, D. (1989). Clinical judgment and decision-making in neuropsychology. *Archives of Clinical Neuropsychology, 4*, 233–265.

Bicycle Drawing Test

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Definition

As the name suggests, the Bicycle Drawing Test requires patients to draw a picture of a bicycle in freehand using a pencil. It can be a useful measure of visual–spatial and visual–motor impairments, and also has been used in the assessment of hemi-neglect syndromes. Typically, the patient is asked to draw a copy of a simple line drawn picture of a bicycle. Many clinicians first ask the patient to draw a bicycle in freehand from their own memory, to assess their constructional ability in the absence of a model.

History and Clinical Evidence

The Bicycle Drawing Test is widely associated with Piaget's (Piaget, 1955) investigations of cognitive development, though similar tests seem to have been employed earlier (Poppelreuter, 1990; Veiders, 1934). Neuropsychological investigations of focal unilateral lesions (Hecaen & Assal, 1970) have demonstrated differences in performance between patients with left- and right-sided posterior brain lesions. Such comparisons have also suggested qualitative differences in the types of errors made among patients with left- and right-sided lesions (Angenent, 1971). Such findings led to the inclusion of this test as a measure of

constructional ability. One study compared the Bicycle Drawing test to the Bender–Gestalt test (Greenberg, Rodriguez & Sesta, 1994), and found bicycle drawing to be more sensitive to brain dysfunction in children with visual–spatial problems than the Bender–Gestalt test. Interestingly, improvements in bicycle drawing performance have been described in studies of patients with Parkinson's disease, multiple sclerosis, and Tourette's syndrome (Sandyk, 1994; Sandyk, 1997a, 1997b), with reversal of spatial orientation in Parkinson's patients when they received electromagnetic pulses to their brain (Sandyk, 1998). The quality and complexity of the drawing produced by children and adults has also been linked to their intellectual ability (Sharma, 1972).

Current Clinical Use

Formal scoring systems exist for the Bicycle Drawing test (Greenberg et al., 1994) providing a means for deriving quantitative results when using this test. Studies comparing the drawings of amateurs with artists have also shown that observational, experimental, and neuropsychological methods for scoring drawings can provide systemic differences in cognitive skills among individuals (van Sommers, Lange-Küttner & Thomas, 1995). Yet, most clinicians currently use the Bicycle Drawing test in conjunction with other constructional tests, including coping or freehand drawing tasks (e.g., cube, house), and examine results qualitatively for gross spatial distortion, or omissions. Other tests, such as copying of the Rey Complex Figure are now more widely used for visual–motor assessment. Yet, the bicycle drawing test is an easy-to-administer task that can yield valuable information about the visual-spatial and constructional abilities of patients. It can also detect hemi-neglect syndrome, as some patients may omit one side of the bicycle. IT can yield information about additional information on intellectual development when used in the assessment of children. Generally, it should not be used as a stand-alone test, but should rather be used in conjunction with other tests of visual constructional functioning.

Cross References

- ► Bender–Gestalt
- ► Block Design
- ► Clock Drawing
- ► Rey Complex Figure Test

References and Readings

Angenent, H. L. (1971). The development of thought structure among normal and mentally retarded children. *Nederlands Tijdschrift voor de Psychologie en haar Grensgebieden, 26*(4), 215–232.

Greenberg, G. D., Rodriguez, N. M., & Sesta, J. J. (1994). Revised scoring, reliability, and validity investigations of Piaget's Bicycle Drawing Test. *Assessment, 1*(1), 89–101.

Hecaen, H., & Assal, G. (1970). A comparison of constructive deficits following right and left hemispheric lesions. *Neuropsychologia, 8*(3), 289–303.

Piaget, J. (1955). Perceptual and cognitive (or operational) structures in the development of the concept of space in the child. *Acta Psychologica, 11*, 41–46.

Poppelreuter, W. (1990). *Disturbances of lower and higher visual capacities caused by occipital damage: with special reference to the psychopathological, pedagogical, industrial, and social implications.* Oxford/New York: Clarendon Press/Oxford University Press.

Sandyk, R. (1994). Reversal of a visuoconstructional deficit in Parkinson's disease by application of external magnetic fields: A report of five cases. *International Journal of Neuroscience, 75*(3–4), 213–228.

Sandyk, R. (1997a). Progressive cognitive improvement in multiple sclerosis from treatment with electromagnetic fields. *International Journal of Neuroscience, 89*(1–2), 39–51.

Sandyk, R. (1997b). Reversal of a visuoconstructional disorder by weak electromagnetic fields in a child with Tourette's syndrome. *International Journal of Neuroscience, 90*(3–4), 159–167.

Sandyk, R. (1998). Reversal of the bicycle drawing direction in Parkinson's disease by AC pulsed electromagnetic fields. *International Journal of Neuroscience, 95*(3–4), 255–269.

Sharma, T. R. (1972). Measuring intelligence through bicycle drawings. *Indian Educational Review, 7*(1), 1–30.

van Sommers, P. (1995). Observational, experimental and neuropsychological studies of drawing. In C. Lange-Küttner, & G. V. Thomas (Eds.) *Drawing and looking: Theoretical approaches to pictorial representation in children* (pp. 44–61). Hertfordshire: Harvester Wheatsheaf.

Veiders, E. (1934). Analyse der Fähigkeit zum räumlichen Denken. Analysis of the ability for spatial thinking. *Psychotechnisches Zeitschrift, 9*, 52–60.

Bilateral Acoustic Neurofibromatosis

► Neurofibromatosis Type 2

Bilateral Simultaneous Stimulation

► Double Simultaneous Stimulation

Bilingual Aphasia

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Definition

Bilingual aphasia is a term referring to aphasia in an individual who is bilingual or multilingual. The degree and nature of impairment vary widely, and depend on the interplay among a number of factors, including site and size of the lesion, the individual's premorbid language learning and experience history, and proficiency and immersion in each language. Current evidence suggests that the same classification/diagnosis of aphasia (e.g., fluent, nonfluent) tends to hold across both languages, with varying degrees of impairment and recovery trajectories (Paradis, 1998). In the USA, 45,000 new cases of bilingual aphasia are expected each year (Paradis, 2001).

Historical Background

Knowledge of bilingual aphasia is primarily based on a history of case studies (for reviews, see Pearce, 2005; Lorenzen & Murray, 2008), including documented cases as early and varied as a patient who could read Latin but not German (Gesner, 1770); a patient in Southern France who, after brain damage, showed a selective deficit in French but not Occitan (Lordat, 1843); and a trilingual patient who recovered French and Spanish, but lost the ability to even understand Italian (Pitres, 1895). Based on early case studies, a number of predictions have been made about patterns of impairment and recovery in bilingual individuals with aphasia, including better recovery of the mother tongue (Ribot's Law, 1882), the most familiar language (Pitres' Rule, 1895), the language dominant in the environment (Bychowsky, 1919), and the language "closest to our heart" (Minkowski, 1927).

Current Knowledge

A systematic and theoretical understanding of bilingual aphasia relies on an understanding of the bilingual language system and its neural correlates. While more similarities than differences have been observed with regard to linguistic processing and neuroanatomical correlates of language for bilinguals and monolinguals, language function of a bilingual speaker is not equivalent to that of a monolingual speaker. With two language systems instead of one, bilinguals have been shown to activate their two languages in parallel and experience interaction and interference between them. In addition, bilinguals frequently switch and translate between languages.

There is still considerable debate regarding the functional localization of language in bilinguals. The evidence to date indicates that the extent of overlap in representation of languages depends on the speaker's language proficiency and the age at which the second language was acquired. Recent imaging research suggests that different languages are most likely to share the same brain regions when the second language is acquired earlier (Paradis, 2000; Wartenburger et al., 2003), and when both languages are highly proficient (e.g., Chee et al., 1999; Golestani, Alario, Meriaux, Le Bihan, Dehaene, & Pallier, 2006; Klein, Milner, Zatorre, Zhao, & Nikelski, 1999; Perani et al., 2003). Even in languages that are structurally different from each other, at least partially shared representations have been identified (e.g., English/Chinese; English/ASL, Chee et al., 1999; Emmorey, Mehta, & Grabowski , 2007).

Additional brain areas have also been identified during bilingual language processing. Bilinguals may engage the prefrontal cortex to a larger extent when they process a less proficient language than when they process a more proficient language (e.g., Golestani et al., 2006; Indefrey, 2006; Marian et al., 2007; Perani et al., 2003; Sakai, Miura, Narafu, & Muraishi, 2004). Increased activation in dorsolateral prefrontal cortex, anterior cingulate gyrus, and supramarginal gyrus has been observed during language switching (Hernandez, Martinez, & Kohnert, 2000; Price, Green, & von Studnitz, 1999; Wang, Xue, Chen, Xue, & Donga, 2007), while increased activation in anterior cingulate gyrus and basal ganglia has been noted during translation (Price et al., 1999). Finally, bilinguals show activation of the left caudate nucleus and anterior cingulate gyrus for naming tasks in a bilingual context (Abutalebi et al., 2007). Abutalebi and Green (2007) proposed that the neural network that supports language control in bilinguals likely includes anterior cingulate cortex, prefrontal cortex, caudate nucleus, and left inferior parietal lobule.

Bilingual individuals with aphasia typically go through a variety of changes in their language abilities, where their languages are available to different degrees during the acute phase of recovery (up to 4 weeks post onset). During this phase, availability of representations may vary because of diaschisis. As impairment patterns

stabilize during the post-acute phase (up to 5 months post-onset), language impairment becomes more directly related to site of lesion and damage to specific language representations.

Clinical Variants and Recovery Patterns

Bilingual individuals with aphasia show great variability in impairment and recovery patterns. Paradis (2001), in a review of 132 cases of bilingual aphasia, found that 61% showed parallel recovery of their two languages, 18% showed differential recovery of their two languages, 7% showed blended recovery, and 5% showed selective recovery (for similar distributions on a sample of 20 Italian–Friulian patients, see Fabbro, 2001). Reports of atypical and pragmatically inappropriate language switching behaviors in bilingual individuals with aphasia have also appeared (Muñoz, Marquardt, & Copeland, 1999).

Parallel impairment refers to aphasia of the same type and severity in both languages. The two languages are impaired and recover simultaneously (relative to premorbid language proficiency). *Differential impairment* refers to aphasia of the same type in both languages (e.g., fluent vs. nonfluent) with crosslinguistic differences in severity levels. In contrast, *differential aphasia* refers to different aphasia symptoms in each language. *Differential recovery* refers to one language recovering better than the other (relative to premorbid levels). *Blended impairment* refers to the inappropriate combination of two or more languages (e.g., the patient may lose the ability to discriminate between languages). *Pathological mixing*, characterized by inadvertent and uncontrolled language switches, is typically associated with blended impairment. In contrast, *pathological fixation* is an inability to switch languages. *Antagonistic recovery* refers to a pattern where one language recovers first, and starts regressing when the other language starts to recover. *Alternating antagonism* refers to repetition of the antagonistic pattern, with the two languages alternating in availability (cycles may range from hours to months). *Selective impairment* refers to aphasia in only one language, while the other language remains intact (relative to premorbid language proficiency). In addition, a variety of deficits have been identified in bilingual aphasics' ability to translate from one of their languages to the other. An *inability to translate* is reflected in bilinguals' inability to translate either forward (from their native language to their second language) or backward (from their second language to their native language). *Paradoxical translation* is an ability to translate from one language to the other, but not the other way around. *Translation without comprehension* is a preserved ability to translate without an ability to comprehend the meaning of either translation. Finally, *spontaneous translation* is the involuntarily production of translations that cannot be inhibited.

The heterogeneity of the bilingual population makes it difficult to link language profiles and lesion site/size with specific impairment and recovery patterns in individuals with bilingual aphasia. However, a number of *linguistic factors* in impairment and recovery patterns have been identified. Naming and translation of cognate words (that share sound and meaning in the two languages, e.g., *lamp-lámpara*) is typically less impaired in bilingual individuals with aphasia than naming and translation of noncognate words (*key – llave*, Goral, Levy, & Obler, 2006; Kohnert, 2004; Roberts & Deslauriers, 1999). In general, aspects of bilinguals' languages that are more shared are also more resistant to impairment (e.g., Kiran & Tuctenhagen, 2005). Linguistic features that differ between languages (e.g., different grammatical systems) may result in differences in how symptoms of aphasia are expressed even if the same underlying deficit exists. For example, a morphologically rich language can theoretically undergo greater morphological breakdown, and morphological deficits may look more severe. Therefore, cross-linguistic differences in the symptoms and recovery patterns of bilingual aphasia frequently occur at points where the two linguistic systems diverge.

Another explanation for divergent recovery patterns in bilingual aphasia is the Cue Strength hypothesis (e.g., Wulfeck, Bates, & Capasso, 1991). According to this hypothesis, the linguistic importance of a grammatical structure or the contribution it makes to the linguistic message may account for crosslinguistic differences in syntactic deficits, with more important cues being more likely to be preserved. For example, English-speaking individuals with aphasia have been found to be more sensitive to word-order errors during grammaticality judgments while Italian-speaking individuals with aphasia were more sensitive to morphological errors. Similarly, differences in reading impairments have been found across languages with different orthographies. Readers are referred to a detailed review of current knowledge about bilingual aphasia by Lorenzen and Murray (2008).

Assessment

An important part of assessment in bilingual aphasia consists of establishing premorbid proficiency levels as accurately as possible and determining the nature and

extent of impairment in each language relative to these premorbid proficiency levels. Self-reports, questionnaires about the history of language use (e.g., code-switching), reports from family members or friends, and written or recorded samples of patients' language abilities are typically used to establish premorbid proficiency levels. Once premorbid proficiency levels have been established, it is important to assess both of the patients' languages in order to gauge their full linguistic capacity and impairments across languages.

There are currently no assessment measures for bilingual aphasia that meet *all* standards for measurement validity. The bilingual aphasia test (BAT, Paradis, 1987) is the most comprehensive tool available and provides systematic ways to assess aphasia in many languages. Tasks on the BAT are equivalent in linguistic complexity across languages, and cover assessment of linguistic levels (phonological, lexical-semantic, morphological, syntactic), linguistic skills (comprehension, formulation, repetition, judgment, lexical access), and linguistic units (words, sentences, paragraphs). The BAT assumes that the test-taker has pre-morbid proficiency in each language that is equivalent to at least 400 language-learning hours. The test is administered in each language on different days. Where it is not feasible to obtain all language versions of the BAT, its principles may be followed during assessment.

Additional tasks that may be useful in examining bilingual individuals' language impairments include the type-token ratio in each language based on comparable language samples, number of verbs and grammatical clauses per utterance, semantic acceptability, confabulation, and total number of words or utterances within a set time window (fluency measures). Preservation of links between languages may be assessed by testing participants' translation abilities from the native language to the second language and vice versa. As part of cognitive assessment, language switching behaviors may be examined. Pathological mixing can be distinguished from nonpathological mixing: For example, in nonpathological mixing, subject and verb should be in the same language, and switches should not occur on prepositions.

If, due to limitations in resources, assessment and treatment are done only in English, the clinician may obtain information on the structure of the clients' other language in order to identify crosslinguistic influences in the clients' English output. This may allow the clinician to distinguish low premorbid proficiency in English from a disorder. The *dynamic assessment* approach provides an alternative method for examining deficits in situations of low premorbid language proficiency. Dynamic assessment focuses on the ability to learn new information, rather than the ability to retrieve known information. A clinician may explain a new grammatical rule and test the client's ability to generalize it. If the client generalizes the rule easily, then weak linguistic performance is likely due to the influence of the non-target language or low proficiency in the target language, rather than aphasia.

Treatment

Assessment of both languages, together with the social communication needs of the client, will inform choice of therapy language and specific therapy goals. A primary goal in the treatment of bilingual individuals with aphasia is to maximally benefit both languages even if treatment occurs in only one language.

Treatment may be conducted to target both languages directly. During bilingual treatment, language-switching may be encouraged as a compensatory strategy to allow the client to use his/her full linguistic capacity. Translation may be used in a similar manner to aid lexical access. For example, switch-back through translation (SBT) treatment (Ansaldo & Marcotte, 2007) is a procedure where the client is cued to translate the word back into the other language whenever an inadvertent switch occurs.

If resources are only available to treat in English, the speech–language pathologist may work to identify outside resources in helping to rehabilitate the clients' other languages. Such linguistic resources may include language-specific community groups, or guidance of family members. Crosslinguistic generalization is most likely to occur when shared representations are targeted for treatment, crosslinguistic associative links are used, or similar cognitive processes are a focus of intervention (e.g., reading in alphabetic languages). Current evidence suggests that treatment in the person's weaker language may generalize to his/her stronger language, especially when treatment targets are similar across languages (Edmonds & Kiran, 2004). However, generalization from the stronger to the weaker language is less likely. Generalization across languages has been shown when cognate words are used in treatment (Kohnert, 2004), semantic features are treated (Edmonds & Kiran, 2004, 2006), and general cognitive function is treated (Kohnert, 2004). However, cross-linguistic generalization does not always occur (e.g., Galvez & Hinckley, 2003).

Future Directions

In 2000, 18% of the population older than 5 years of age (47 million people) spoke a language other than English

at home, up from 14% in 1990 and 11% in 1980 (U.S. Census, 2000). As the bilingual population grows, with special growth in older adults, the need for accommodation of bilingual individuals with aphasia will increase. Among Mexican Americans, stroke incidence is slightly higher (1.63%) than in non-Latino white peers (1.36%), and transient ischemic attacks are more frequent at younger ages (Lorenzen & Murray, 2008). The US Department of Health and Human Services found that Latino individuals were 33% less likely to receive necessary healthcare services, compared to non-Latino white peers (Lorenzen & Murray, 2008). With these changes in population dynamics, systematic evidence-based research on the efficacy of various treatment approaches for bilingual aphasia has become increasingly necessary.

One particularly promising avenue of research in bilingual aphasia is in the area of cognitive control. Language production requires cognitive control skills. These skills may be honed with bilingual experience, as bilinguals must suppress one language in favor of another language every time they speak. Balanced bilinguals have been shown to have cognitive advantages over monolinguals on nonlinguistic executive function tasks, especially as they age (Bialystok, Craik, Klein, & Viswanathan, 2004; Kave, Eyal, Shorek, & Cohen-Mansfield, 2008; Zied et al., 2004). Given the important role of the executive control system in bilinguals' ability to appropriately maintain and switch between languages, it is important to examine the integrity of the cognitive control system in bilingual individuals with aphasia. It is also possible that cognitive-control advantages may support language recovery and responsiveness to treatment in bilingual patients with aphasia.

Cross References

- ▶ Aphasia
- ▶ Aphasia Tests
- ▶ Multilingual Aphasia Examination
- ▶ Speech–Language Pathology
- ▶ Speech–Language Therapy

References and Readings

Abutalebi, J., Brambati, S. M., Annoni, J. M., Moro, A., Cappa S. F., & Perani, D. (2007). The neural cost of the auditory perception of language switches: An event-related fMRI study in bilinguals. *Journal of Neuroscience, 27*, 13762–13769.

Abutalebi, J., & Green, D. (2007). Bilingual language production: The neurocognition of language representation and control. *Journal of Neurolinguistics, 20*, 242–275.

Ansaldo, A. I., & Marcotte, K. (2007). Language switching and mixing in the context of bilingual aphasia. In J. G. Centeno, L. K. Obler, & R. T. Anderson (Eds.), *Studying communication disorders in Spanish speakers: Theoretical, research, and clinical aspects*. Clevedon, UK: Multilingual Matters.

Bialystok, E., Craik, F. I. M., Klein, R., & Viswanathan, M. (2004). Bilingualism, aging, and cognitive control: Evidence from the Simon task. *Psychology and Aging, 19*, 290–303.

Bychowsky, Z. (1919). Concerning the restitution of language loss subsequent to a cranial gunshot wound in a polyglot aphasic. In M. Paradis (Ed.), *Readings on aphasia in bilinguals and polyglots* (pp. 130–144). Montreal, QC: Didier.

Chee, M. W., Caplan D., Soon, C. S., Sriram, N., Tan, E. W., Thiel, T., et al. (1999). Processing of visually presented sentences in Mandarin and English studied with fMRI. *Neuron, 23*, 127–137.

Edmonds, L., & Kiran, S. (2004). Confrontation naming and semantic relatedness judgments in Spanish/English bilinguals. *Aphasiology, 18*, 567–579.

Edmonds, L., & Kiran, S. (2006). Effect of semantic naming treatment on crosslinguistic generalization in bilingual aphasia. *Journal of Speech, Language, and Hearing Research, 49*(4), 729–748.

Emmorey, K., Mehta, S., & Grabowski, T. (2007). The neural correlates of sign versus word production. *Neuroimage, 36*, 202–208.

Fabbro, F. (2001). The bilingual brain: Bilingual aphasia. *Brain and Language, 79*, 201–210.

Galvez, A., & Hinckley, J. J. (2003). Transfer patterns of naming treatment in a case of bilingual aphasia. *Brain and Language, 87*, 173–174.

Gesner, J. A. P. (1770). *Sammlung von Beobachtungen aus der Arzneygelehrtheit und Naturkunde* (pp. 1769–1776). Nördlingen: CG Beck.

Golestani, N., Alario, F. X., Meriaux, S., Le Bihan, D., Dehaene, S., & Pallier, C. (2006). Syntax production in bilinguals. *Neuropsychologia, 44*(7), 1029–1040.

Goral, M., Levy, E. S., & Obler, L. K. (2006). Neurolinguistic aspects of bilingualism. *International Journal of Bilingualism, 6*, 411–440.

Hernandez, A. E., Martinez, A., & Kohnert, K. (2000). In search of the language switch: An fMRI study of picture naming in Spanish-English bilinguals. *Brain and Language, 73*, 421–431.

Indefrey, P. (2006). A meta-analysis of hemodynamic studies on first and second language processing: Which suggested differences can we trust and what do they mean? In M. Gullberg & P. Indefrey (Eds.), *The cognitive neuroscience of second language acquisition* (pp. 279–304). Malden, MA: Blackwell.

Kave, G., Eyal, N., Shorek, A., & Cohen-Mansfield, J. (2008). Multilingualism and cognitive state in the oldest old. *Psychology and Aging, 23*, 70–78.

Kiran, S., & Tuctenhagen, J. (2005). Imageability effects in normal bilingual adults and in aphasia: Evidence from naming to definition and semantic priming tasks. *Aphasiology, 19*, 315–325.

Klein, D., Milner, B., Zatorre, R. J., Zhao, V., & Nikelski, J. (1999). Cerebral organization in bilinguals: A PET study of Chinese–English verb generation. *NeuroReport, 10*, 2841–2846.

Kohnert, K. (2004). Cognitive and cognate-based treatments for bilingual aphasia: A case study. *Brain and Language, 91*, 294–302.

Lordat, J. (1843). Leçons tirées du cours de physiologie de l'année scolaire 1842–1843. *Journal de la Société de médecine pratique de Montpellier, 7*, 333–353.

Lorenzen, B., & Murray, L. (2008). Bilingual aphasia: A theoretical and clinical review. *American Journal of Speech-Language Pathology, 17*, 299–317.
Marian, V., Shildkrot, Y., Blumenfeld, H. K., Kaushanskaya, M., Faroqui-Shah, Y., & Hirsch, J. (2007). Cortical activation during word processing in late bilinguals: Similarities and differences as revealed by fMRI. *Journal of Clinical and Experimental Neuropsychology, 29*, 247–265.
Marrero, M. Z., Golden, C. J., Espe-Pfeifer, P. (2002). Bilingualism, brain injury, and recovery: Implications for understanding the bilingual and for therapy. *Clinical Psychology Review, 22*, 465–480.
Minkowski, E. (1927). *La Schizophrénie.* Paris: Payot.
Muñoz, M. L., Marquardt, T., & Copeland, G. (1999). A comparison of the code-switching patterns of speakers with aphasia and neurologically normal bilingual speakers of English and Spanish. *Brain and Language, 66*, 249–274.
Paradis, M. (1987). *The assessment of bilingual aphasia.* Hillsdale, NJ: Erlbaum.
Paradis, M. (Ed.). (1995). *Aspects of bilingual aphasia.* Oxford: Pergamon.
Paradis, M. (1998). Aphasia in bilinguals: What is atypical? In P. Coppens, Y. Lebrun, & A. Basso (Eds.), *Aphasia in atypical populations* (pp. 35–66). Hillsdale, NJ: Erlbaum.
Paradis, M. (2000). Prerequisites for a study of neurolinguistic processes involved in simultaneous interpreting. A synopsis. In B. Englund Dimitrova & K. Hyltenstam (Eds.), *Language processing and simultaneous interpreting: Interdisciplinary perspectives* (pp. 17–24). Amsterdam: John Benjamins.
Paradis, M. (2001). Bilingual and polyglot aphasia. In R. S. Berndt (Ed.), *Handbook of neuropsychology* (2nd ed., pp. 69–91). Oxford: Elsevier Science.
Pearce, J. M. S. (2005). A note on aphasia in bilingual patients: Pitres' and Ribot's laws. *European Neurology, 54*, 27–31.
Perani, D., Abutalebi, J., Paulesu, E., Brambati, S., Scifo, P., Cappa, S. F., et al. (2003). The role of age of acquisition and language usage in early, high-proficient bilinguals: A fMRI study during verbal fluency. *Human Brain Mapping, 19*, 170–182.
Pitres, A. (1895). Etude sur l'aphasie chez les polyglottes. *Revue de Medicine, 15*, 873–899.
Price, C. J., Green, D. W., & von Studnitz, R. (1999). A functional imaging study of translation and language switching. *Brain, 122*, 2221–2235.
Roberts, P. M., & Deslauriers, L. (1999). Picture naming of cognate and non-cognate nouns in bilingual aphasia. *Journal of Communication Disorders, 32*, 1–22.
Sakai, K. L., Miura, K., Narafu, N., & Muraishi, Y. (2004). Correlated functional changes of the prefrontal cortex in twins induced by classroom education of second language. *Cerebral Cortex, 14*, 1233–1239.
U.S. Census Bureau: Language Use. (2000). Retrieved November 20, 2009, from http://www.census.gov/population/www/socdemo/lang_use.html
Wang, Y., Xue, G., Chen, C., Xue, F., & Donga, Q. (2007). Neural bases of asymmetric language switching in second-language learners: An ER-fMRI study. *NeuroImage, 35*, 862–870.
Wartenburger, I, Heekeren, H. R., Abutalebi, J., Cappa, S. F., Villringer, A., & Perani, D. (2003). Early setting of grammatical processing in the bilingual brain. *Neuron, 37*, 159–170.
Wulfeck, B., Bates, E., & Capasso, R. (1991). A cross-linguistic study of grammaticality judgments in Broca's aphasia. *Brain and Language, 41*, 311–336.
Zied, K. M., Phillipe, A., Karine, P., Valerie, H. T., Ghislaine, A., Arnaud, R., et al. (2004). Bilingualism and adult differences in inhibitory mechanisms: Evidence from a bilingual stroop task. *Brain and Cognition, 54*, 254–256.

Binge Drinking

► Alcohol Abuse

Binswanger's Disease

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Synonyms

CADASIL; Multi-infarct dementia; Subcortical leukoencephalopathy; Subcortical vascular dementia

Definition

Binswanger's disease (BD) is a type of subcortical vascular dementia caused by widespread, microscopic damage to cerebral white matter. The damage is usually the result of atherosclerosis (i.e., narrowing of arterial blood vessels) that reduces the supply of blood to subcortical areas of the brain, causing tissue to die. The characteristic pattern of BD-damaged brain tissue can be seen using brain imaging techniques such as computed tomography (CT) or magnetic resonance imaging (MRI). CT imaging of BD often reveals symmetric, noncontrasting hypodensities also called "leukoaraiosis," and more sensitive MRI imaging reveals periventricular hyperintensities on T2 sequences.

There is some controversy in the literature about whether BD constitutes a distinct clinical entity or simply describes the result of different neuropathologies that affect subcortical white matter (Caplan, 1995; Hachinski et al., 2006; Olsen and Clasen, 1998; Pantoni and Garcia, 1995). Although the precise cause of BD is unclear, it is frequently associated with diabetes, cardiovascular disease, previous cerebrovascular accident, malnutrition and, most notably, hypertension. The age of onset for BD is typically between ages 60 and 79 years, with men and women equally affected. Estimates about the incidence of BD range from 3–12% (Babikian and Ropper, 1987).

Current Knowledge

Neuropathology

Gross pathology of brain tissue affected by BD is characterized by gyral atrophy and widening of the sulci resulting from the loss of cerebral white matter. Lateral ventricles are also typically enlarged. Lacunar infarctions can be found in the white matter, pons, and basal ganglia as well as occasionally in the cerebellum. Microscopic pathology of BD is marked by diffuse and patchy white matter demyelination with areas of reactive gliosis and decreased nerve fibers. The small arteries of the white matter also show fibrous thickening, which is associated with hypertension and cardiovascular disease. BD is believed to result from ischemic injury of frontal lobe white matter and is distinct from the lacunar state of small vessel disease that also causes dementia but results from infarctions in the basal ganglia and internal capsule.

Clinical Symptoms

BD typically has a slow, insidious onset that eventually manifests in cognitive and motor dysfunctions related to the disruption of subcortical neural circuits. Specifically, patients exhibit executive dysfunction (e.g., impaired initiation, inhibition, monitoring of goal-directed behavior, and verbal fluency), psychomotor slowing, inattention, and short-term memory loss with poor retrieval but intact recognition (Roman, 2003). Other symptoms include changes in speech, an unsteady gait, postural instability, changes in personality or mood (including apathy, irritability, and depression), as well as urinary incontinence (Babikian and Ropper, 1987; Caplan, 1995; Lezak, Howieson, & Loring, 2004; Roman, 2003).

Treatment

Treatment of BD is often targeted at specific symptoms. For example, medications such as donepezil and memantine may be used to treat the cognitive symptoms associated with BD. Individuals with depression may be treated with antidepressant medications (e.g., serotonin-specific reuptake inhibitors (SSRIs) such as sertraline or citalopram) and individuals with agitation or disruptive behavior can be treated with atypical antipsychotic medications such as risperidone or olanzapine (Sink, Holden, & Yaffe, 2005). Other treatment interventions are often focused on reducing cardiovascular risk factors by eating a healthy diet, exercising, and not smoking or drinking too much alcohol. Controlling vascular risk factors can help improve cognition and may even help prevent the development of dementia (Roman, 2005).

Prognosis

BD is a progressive disease and there is currently no cure. The course of BD can be variable and deterioration can occur suddenly or gradually and then progress in a stepwise manner (Santamaria Ortiz and Knight, 1994).

Cross References

▶ Leukoaraiosis

References and Readings

Babikian, V., & Ropper, A. H. (1987). Binswanger's disease: a review. *Stroke, 18*(1), 2–12.

Caplan, L. R. (1995). Binswanger's disease – revisited. *Neurology, 45*(4), 626–633.

Hachinski, V., Iadecola, C., Petersen, R. C., Breteler M. M., Nyenhuis, D. L., Black S. E. et al. (2006). National Institute of Neurological Disorders and Stroke-Canadian Stroke Network vascular cognitive impairment harmonization standards. *Stroke, 37*(9), 2220–2241.

Lezak, M. D., Howieson, D. B., & Loring, D. (2004). *Neuropsychological assessment* (4th ed., p. 1016). New York: Oxford University Press.

Olsen, C. G., & Clasen, M. E. (1998). Senile dementia of the Binswanger's type. *American Family Physician, 58*(9), 2068–2074.

Pantoni, L., & Garcia, J. H. (1995). The significance of cerebral white matter abnormalities 100 years after Binswanger's report. A review. *Stroke, 26*(7), 1293–1301.

Roman, G. C. (2003). Neurological aspects of vascular dementia: Basic concepts, diagnosis, and management, In P. A. Lichtenberg, D. L. Murman, & A. M. Mellow (Eds.), *Handbook of dementia - psychological, neurological, and psychiatric perspectives* (pp. 149–171). Hoboken, NJ: John Wiley & Sons, Inc.

Roman, G. C. (2005). Vascular dementia prevention: a risk factor analysis. *Cerebrovascular Diseases, 20*(Suppl. 2), 91–100.

Santamaria Ortiz, J., & Knight, P. V. (1994). Review: Binswanger's disease, leukoaraiosis and dementia. *Age Ageing, 23*(1), 75–81.

Sink, K. M., Holden, K. F., & Yaffe, K. (2005). Pharmacological treatment of neuropsychiatric symptoms of dementia: a review of the evidence. *Jama, 293*(5), 596–608.

Biological Cycles

▶ Circadian Rhythms

Biomechanics of Injury

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Definition

An inclusive term to explore and describe the mechanical and physical factors that result in traumatic brain injury.

Current Knowledge

Biomechanical injuries typically occur without the direct impact of an outside object on the skull or brain, but rather in the context of acceleration–deceleration injuries. High-speed situations such as motor vehicle accidents and sports provide mediums for these inertia-based injuries. The structure of the skull includes sinuses and bony protective regions. Underlying brain tissue is held in suspension underneath the skull not only by the meninges, but also by a cushion of cerebral spinal fluid. Different inertial forces such as linear acceleration, rotation of the head, or massive vibration or air pressure changes in the environment can result in a wide range of potential damage to these underlying substances. These disruptions may include skull fracture, linear acceleration injury, rotational injury, and the effects of vibration of the skull and brain against one another.

Superficial or deep lesions may result in parenchymal injury depending on the type of mechanical force that occurred at the time of head trauma. Linear acceleration injuries are most often associated with superficial brain injuries such as cerebral contusions, while rotational injuries are most often associated with disruptions to deep white matter tracts and projections, and centrally located brain structures and neural networks. Consequently, rotational injuries may be more severe with regard to effects on cognition, motor skills, and functional status. Concussion with or without loss of consciousness is also a consequence of biomechanical forces and the subsequent effects on underlying brain tissue.

The biomechanics of injury differentially affect initial and long-term recovery from acquired brain injury. Understanding these different mechanical forces may help one to improve understanding of injury severity. Increased understanding of the biomechanics underlying brain injury has led to the development of protective headgear in high-speed or direct-impact sports such as biking, motor racing, football, and hockey.

Cross References

- ▶ Acceleration Injury
- ▶ Deceleration Injury
- ▶ Diffuse Axonal Injury
- ▶ Rotational Acceleration
- ▶ Traumatic Brain Injury

References and Readings

Bayly, P. V., Cohen, T. S., Leister, E. P., Ajo, D., Leuthardt, E. C., Genin, G. M. (2005). Acceleration-induced deformation of the human brain. *Journal of Neurotrauma, 22*, 845–856.

Ommaya, A. K., Goldsmith, W., & Thibault, L. (2002). Biomechanics and neuropathology of adult and pediatric head injury. *British Journal of Neurosurgery, 16*, 220–242.

Biopsy

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Definition

A biopsy is a medical examination entailing the removal of cellular tissue via a needle or surgical resection. In particular, an incisional or core biopsy involves a select sample of tissue, whereas an excisional biopsy necessitates a larger specimen. The biopsy results are typically evaluated microscopically by a pathologist, who determines if a lesion's pathology is benign or malignant. Although histological confirmation of tumor diagnosis can be achieved, a biopsy sampling error can result if the specific tissue section does not contain the most representative cellular features. When the biopsy is abnormal, the cell structure may be unusual and indicative of malignancy. However, further pathological examination is often required to make a definitive diagnosis.

Cross References

- ▶ Psychopathology
- ▶ Radiosurgery

References and Readings

Yu, X., Liu, Z., Tian, Z., Li, S., Huang, H., Xiu, B., et al. (2006). Stereotactic biopsy for intracranial space-occupying lesions: clinical analysis of 550 cases. *Stereotactic and Functional Neurosurgery, 75*, 103–108.

Biorhythms

- ▶ Circadian Rhythms

BIT

- ▶ Rivermead Behavioral Inattention Test

BJLOT

- ▶ Judgment of Line Orientation

Blast Injury

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Definition

A trauma sustained as a result of exposure to an explosion or its effects. Technically, blast injury can affect any physical system/function; its neurological effects are highlighted here.

Historical Background

Blast injuries can occur in any setting, civilian or military. However, exposure to the effects of explosive forces is much more associated with military populations, and has been since the advent of modern warfare. Awareness of the effect of blast injuries began to emerge in earnest with the phenomenon of "shell shock" during the First World War. That war exposed staggering number of soldiers to explosive injuries, far more than had previous conflicts. As a result, an ever-increasing number of military personnel presented with vague but incapacitating complaints that prevented them from returning to active (particularly frontline) duty. Initially, these symptoms were considered secondary to organic central nervous system injury. Over time however, others favored a more psychological or even intentional (i.e., malingering) explanation, citing the fact that many shell-shocked servicemen did not appear to have been as close to the explosion as would seem necessary to truly be negatively affected. The nature of the shell-shock symptoms was further obscured by the lack of diagnostic methods, absence of a clear definition of the syndrome, and even political factors (e.g., superiors being able to justify returning much-needed soldiers to frontline duty if their complaints reflected psychological or constitutional weaknesses rather than neurological/organic injuries). This debate of "psychological versus neurological" causes has continued throughout subsequent wars and is a particular focus of the present conflicts in the Middle East, given the high incidence of explosives utilized by terrorists and non-Coalition combatants.

Current Knowledge

With improvements in medical care of trauma and the development of more effective defensive equipment (i.e., body and vehicle armor), a greater number of servicemen and women injured in combat are surviving than ever before: The mortality rate for wounded personnel has declined from approximately 30% during the Second World War to approximately 10% today. As a result, a greater number of the wounded are surviving with traumatic brain injury than in the past – from under 20% during the Vietnam conflict to perhaps near 50% in the current wars in Afghanistan and Iraq. Overall, it has been estimated that possibly as many as 22% of all combat troops in Operations Iraqi Freedom and Enduring Freedom may have incurred an acquired brain injury of some degree. The majority of these combat-related brain injuries are sustained as a result of exposure to an explosion.

Explosions may cause injury through four mechanisms:

1. Primary Blast Injury

A primary injury is one sustained from exposure to the shock/pressure waves initiated by the explosion. When explosive munitions are detonated, a shockwave approaching a speed of 8000 meters/second is generated. The waves generated from a blast can cause life-threatening injuries when they strike an individual directly, or if they reflect off nearby surfaces and then come into contact with the person. The force generated is of such a magnitude that it often results in an instant fatality or in trauma to multiple body systems. Body organs that are relatively solid or fluid-filled tend to sustain a lesser degree of injury than those that are gas-filled or have a gas-fluid interface, such as the tympanic membrane, lung, and colon. Although not fully understood, research suggests that the explosion may injure the central nervous system directly, as in a concussion, but may also indirectly affect the brain. The latter case may occur when peripheral somatic areas are impacted by the blast, setting in motion events that ultimately impact the central nervous system (CNS), such as chemical/metabolic cascades, physical sequelae (i.e., cerebral infarction caused by an air embolism), and/or kinetic events (e.g., transfer of shock/pressure wave energy from the body, up the vasculature into brain tissue). It has been postulated that the severity and number of the patient's physical wounds from primary blasts often overshadow symptoms of traumatic brain injury, delaying diagnosis and treatment for these injuries.

2. Secondary Blast Injury

Secondary injuries occur when shrapnel, debris, or other objects are caught up by the blast and propelled against/into an individual. Many of these injuries are therefore penetrating in nature.

3. Tertiary Blast Injury

This type of injury is sustained when the person is caught up and propelled by the blast wind that follows the initial shock wave, and is thrown against objects, structures, the ground, other individuals, etc., often resulting in blunt force wounds.

4. Quaternary Blast Injury

Quaternary blast injuries are those that arise from the aftereffects of an explosion. Examples include being exposed to radiation, fire, chemicals, dust, or toxic substances that were precipitated by the explosive event.

Typically, an individual is exposed to more than one mechanism, making the contributions of one particular mechanism difficult to separate from others.

Approximately 60% of explosion-related injuries in combat lead to an acquired brain injury. As is the case with other etiologies, the majority of traumatic brain injuries resulting from explosions are classified as mild in nature. Similar to civilian settings, "mild" traumatic brain injury (mTBI) has not been consistently defined in the literature, which is a substantial limitation in making meaningful comparisons between studies. Despite this, definitions such as that proposed by the American Congress of Rehabilitation Medicine are coming into wider acceptance, and have largely been adopted by the military. The ACRM definition of mTBI includes at least one symptom such as less than half an hour of loss of consciousness, less than 24 hours of post-traumatic amnesia, any retrograde or anterograde amnesia, mental status changes immediately after injury, and transient/permanent neurological impairments. The literature cautions that a mild TBI from an explosion may not be equivalent to mild TBI from other etiologies (e.g., motor vehicle accidents, sports injuries), as the former may affect the brain more diffusely and tend to involve trauma to other organ systems, thereby complicating the patient's clinical presentation and recovery. However, studies have indicated that factors such as loss of consciousness and cognitive deficits do not appear to significantly differ between blast victims and those injured by other means, tentatively suggesting that knowledge gleaned from studying these other etiologies has at least some applicability to blast injury survivors.

Cognitive (e.g., memory, attention), somatic (e.g., dizziness, headache, sleep initiation/maintenance difficulties), and emotional (e.g., irritability, nervousness) symptoms are commonly seen initially. In civilian samples, mild TBI symptoms appear to resolve quickly, with most individuals showing rapid recovery within the first week. However, over one third may continue to experience significant post-concussion symptoms, and as many as 15% may continue to experience persistent symptoms after 12 months ("Persistent Post-Concussion Syndrome"). Unfortunately, post-concussion symptoms have not been consistently defined, and many point out that the constellation of symptoms present are vague and lack the specificity needed to identify them as constituting a true syndrome.

This debate over whether the more chronic symptom constellation after mild TBI reflects a true neurological syndrome has particular relevance in blast injury, as the brief history of "shell shock" above illustrates. Those

favoring a neurological position cite animal models in which direct and indirect exposure to primary blasts causes structural, chemical, and electrophysiological changes in the brain. Those weighing psychological factors more heavily in terms of causation point to the mTBI literature that indicates non-neurological factors, such as premorbid psychological coping resources and external stressors, appear to influence the development of concussion symptoms in some individuals. The fact that mTBI symptoms overlap considerably with anxiety disorders such as post-traumatic stress disorder is particularly noteworthy, given the high incidence of PTSD in military personnel who have experienced combat: Gaylord, Cooper, Mercade, Kennedy, Yoder, and Holcomb (2008) found that nearly 20% of military persons who incurred blast and burn injuries were appropriate for both mild brain injury and PTSD diagnoses. Hoge, McGurk, Thomas, Cox, Engel, and Castro (2008) reported that approximately 15% of soldiers surveyed after being returned home might meet criteria for both an mTBI and PTSD; these servicemen and women were more likely to have been exposed to a blast injury. In addition, their survey indicated that the presence of affective distress might be the major factor in maintaining chronic health difficulties, including mTBI symptomatology. A compromise position of sorts posits mTBI symptoms are likely neurological in origin but are subsequently maintained by emotional/psychological factors, and that the presence of PTSD and similar affective disturbances can complicate healing from and coping with mTBI and vice versa. The fact that PTSD symptoms can arise long after the actual trauma indicates that these emotional disturbances may influence a person at virtually any point in his or her brain injury recovery.

Treatment approaches of blast injuries have paralleled treatment efforts in mild TBI. Treatment begins with a thorough diagnostic assessment. The Armed Services have made significant improvements in their endeavors to standardize comprehensive screening and interviewing methods to identify service personnel who may have experienced an acquired brain injury, beginning on the battlefield and continuing throughout the military's medical system. Efforts have also been made to carefully screen the wounded for other symptoms (e.g., tinnitus) that place them at higher risk for having sustained a TBI in a blast. Any assessment should include a thorough medical evaluation and comprehensive interview that elicits historical information about past psychological treatment/coping, substance use, and combat exposure. Neuropsychological evaluation as early as possible would also be helpful in identifying post-concussive symptoms and clarifying the diagnostic picture, enabling education and treatment efforts to proceed more quickly.

The profile of cognitive weaknesses resulting from blast injuries is quite variable. Slowed attention and information processing speed, motor slowness, and executive and memory difficulties are common. As such, neuropsychological assessment should ideally be broad-based, with all major cognitive domains sampled. As is the case in sports concussions, the symptom picture for many individuals may evolve relatively rapidly, arguing for use of tests that have alternate forms (e.g., California Verbal Learning Test –2nd Edition, Hopkins Verbal Learning Test-Revised). Tracking somatic symptoms (e.g., Post-Concussion Scale-Revised) over time may also have utility. Because of the high degree of PTSD and other affective disorders, a thorough psychological evaluation should always be performed (e.g., MMPI-2, PAI) and observation for these symptoms should be an ongoing effort, not simply one restricted to an initial evaluation. Given the high degree of lowered effort present in civilian mTBI cases, effort testing (e.g., Test of Memory Malingering) is often advocated, with the caveat that poor performances on these tests should not automatically be interpreted as signs of intentional feigning of symptoms, but as a sign that further investigation is warranted as to the cause of the lowered effort.

After a thorough diagnostic assessment has been performed, treatments generally have proceeded along the lines advocated for mild brain injuries attributed to non-blast causes. Specifically, reassurance and education regarding the nature and general recovery of cognitive and other symptoms is delivered, and problematic symptoms are treated with medication (e.g., analgesics for pain, soporific medication for insomnia, antidepressants for affective symptoms) and other strategies (e.g., relaxation strategies for anxiety symptoms, psychotherapy for PTSD symptoms). Aggressive, evidence-based cognitive rehabilitation efforts have been advocated and research programs in this area have been proposed.

Future Directions

The research literature in blast injury is still in an early stage of development. The following is a partial list of necessary future research efforts: clarifying definitions of mTBI and post-concussive symptom constellations; separation of the effect of different blast mechanisms (e.g.,

primary, secondary) on the brain; standardization of research methodology with respect to inducing blast injuries in animal subjects; comparison of mTBI symptoms, course and recovery between blast injury survivors and those who have injuries from sports or other sources; investigation of the effect of multiple blast exposures; investigation of how PTSD/affective distress differs from and interacts with mTBI. More prospective research is clearly needed. Within neuropsychology, development of alternate forms for many tests is encouraged.

Cross References

- ▶ Concussion
- ▶ Mild Brain Injury
- ▶ Post-Concussion Disorder (Syndrome)
- ▶ Post-Traumatic Stress Disorder
- ▶ Traumatic Brain Injury

References and Readings

Batten, S., Beal, S., Bleiberg, J., et al. (2009). Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury and Defense and Veterans Brain Injury Center consensus conference on cognitive rehabilitation for mild traumatic brain injury. Washington, D.C.

Cernak, I., Wang, Z., Jiang, J., Bian, X., & Savic, J. (2001). Ultrastructural and functional characteristics of blast injury-induced neurotrauma. *The Journal of Trauma, Injury, Infection, and Critical Care, 50*, 695–706.

Gaylord, K., Cooper, D., Mercade J., Kennedy, J., Yoder, L., & Holcomb, J. (2008). Incidence of posttraumatic stress disorder and mild traumatic brain injury in burned service members: Preliminary report. *The Journal of Trauma, Injury, Infection, and Critical Care, 64*, S200–S206.

Hoge, C., McGurk, D., Thomas, J., Cox, A., Engel, C., & Castro, C. (2008). Mild traumatic brain injury in U.S. soldiers returning from Iraq. *The New England Journal of Medicine, 358*(5), 453–463.

Jackson, G., Hamilton, N., & Tupler, L. (2008). Detecting traumatic brain injury among veterans of operations enduring and Iraqi freedom. *North Carolina Medical Journal, 69*(1), 43–47.

Jones, E., Fear, N., & Wessely, S. (2007). Shell shock and mild traumatic brain injury: A historical review. *American Journal of Psychiatry, 164*, 1641–1645.

Bleeding

- ▶ Hemorrhage

Blessed Dementia Rating Scale (BDRS)

- ▶ Blessed Dementia Scale

Blessed Dementia Scale

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Synonyms

Blessed dementia rating scale (BDRS); Blessed–roth DS; Dementia scale (DS); Modified blessed dementia scale (DS); Newcastle DS; Revised dementia scale (RDS)

Description

The Blessed Dementia Scale (DS) was developed in 1968 by Blessed and colleagues in an attempt to quantify the "degree of intellectual and personality deterioration" (p. 799) in the elderly.

This rating scale consists of 22 items that reflect (1) changes in performance of everyday activities (8 items; e.g., using money and finding one's way), (2) changes in habits including self-care (3 items; i.e., eating, dressing and continence), and (3) changes in personality, interests, and drives (11 items; e.g., evaluation of rigidity and affect). A close friend or relative is asked to provide these behavior ratings of the examinee over the past six months; when unavailable, medical records can be used. The DS is scored on a 0–28 point scale, where higher numbers indicate a larger decrement in functional capacity. On everyday activity items, a score of 1 is given for total inability to perform a task, a score of ½ is given for partial, variable, or intermittent inability to perform an activity, and a score of 0 is given if the patient is able to perform the task. The changes in habits section is scored on a 4-point scale (i.e., 0–3), resulting in a stronger contribution to the total score. Personality changes

are scored 1 if present or 0 if absent (Blessed, Tomlinson, & Roth, 1968; Blessed, Tomlinson, & Roth, 1988). A total cut-off score of 4 out of 28 is typically used to differentiate patients with dementia versus those without. Scores of 4–9 indicate mild impairment, whereas scores of 10 or higher suggest moderate to severe impairment (Eastwood, Lautenschlaegar, & Corbin, 1983). Stern, Mayeux, Sano, Hauser, and Bush (1987) have suggested 15 as the threshold for moderate impairment.

The original DS also included a second section comprised of a brief battery of simple cognitive tasks, called the Information-Memory-Concentration Test (IMCT; Blessed et al., 1968; Blessed et al., 1988). Similar to other brief mental status instruments, the IMCT incorporates 12 items of information/orientation, 11 items of long-term memory, a brief test for the 5-min recall of a person's name and address, and three sequencing tasks requiring concentration (Blessed et al., 1968; Blessed et al., 1988). This sub-component is typically no longer included in the DS.

Historical Background

The original Dementia Scale (DS) evaluated informant-reported changes in behavior and daily functioning and also included cognitive tasks given to the patient. It was developed by Blessed, Tomlinson, and Roth in 1968 in an attempt to compare the deterioration of intellect and personality with underlying brain neuropathology (Blessed et al., 1968; Blessed et al., 1988). The Revised Dementia Scale (RDS) was introduced in 1988 and included only items reflecting informant-rated changes in everyday activities and habits (items 1 through 11; Erkinjuntti, Hokkanen, Sulkava, & Palos, 1988). The sensitivity and specificity of the revised scale was higher than that of the original DS, possibly due to lower dementia-specificity of the excluded items (i.e., changes in personality, interests, and drive; Lawson, Rodenburg, & Dykes, 1977). However, the 4-week test–retest reliability for the revised scale was lower (r = 0.68) than the original (r = 0.79), potentially due to the inclusion of fewer items (Erkinjuntti, et al., 1988).

Items from the DS have been included in the standardized interview with relatives that is part of the Cambridge Mental Disorders of the Elderly Examination (CAMDEX; Roth et al., 1986). Elements of this scale have also been incorporated in the standardized battery of the Consortium to Establish a Registry for Alzheimer's Disease (CERAD; Morris, Mohs, Rogers, Fillenbaum, & Heyman, 1988). Additional analysis of the scale has indicated that the items can be subdivided into four groups, each with its own score (Cognitive, items 1–7, score range 0–7; Personality Change, items 12–17, score range 0–6; Apathy/Withdrawal, 18, 20, and 21, score range 0–3; Basic Self-Care, 9–11, score range 0–3), in order to aid in interpretation (Stern, Hesdorffer, Sano, & Mayeux, 1990).

Psychometric Data

In community-dwelling individuals, test–retest reliability after 4 weeks was r = 0.79; the first 11 items show marginal reliability (r = 0.68; Erkinjuntti et al., 1988). Cole (1990) found an interrater reliability of r = 0.59 when comparing DS ratings by two independent raters who each interviewed the caretakers of 47 dementia patients.

The initial study employing the DS showed that scores increased as the presence of senile plaques increased (r = 0.77; Blessed et al., 1968). Also, the DS showed discriminative validity in identifying senile dementia patients compared with depressed, paraphrenia, delirious, and physically ill patients (Blessed et al., 1968). Others have also noted that the DS is able to discriminate between dementia patients and community residents (Erkinjuntti et al., 1988; Lam et al., 1997). When a cut-off of 4/28 was used, the DS was shown to have a sensitivity of 90% and a specificity of 84% (Erkinjuntti et al., 1988). Moderate to high correlations have been reported with other measures such as the CERAD Total score (r = 0.40; Chandler et al., 2005), the Mini-Mental Status Exam (r = 0.80; Hendrie et al., 1988), and the CAMDEX (r = 0.77; Hendrie et al., 1988). Additionally, Stern et al. (1987) reported that disease progression can be monitored using the DS; cognitive deficiencies affecting instrumental activities of daily living (e.g., handling money, remembering short lists) were evident early and worsened throughout the disease course, whereas changes in basic self-care did not occur until 4–5 years into the illness (Stern et al., 1990).

A cut-off of 1.5 on the RDS yields a sensitivity of 93% and a specificity of 97% in discriminating between demented and non-demented subjects, regardless of level of dementia (Erkinjuntti et al., 1988). The RDS is also highly correlated with the Activities of Daily Living Scale, the Instrumental Activities of Daily Living Scale, and the Functional Activities Questionnaire (Juva et al., 1997).

The DS appears minimally affected by demographic factors. Age correlates moderately with DS scores ($r = 0.31$), but when degree of dementia is taken into account, age does not have a significant effect (Erkinjuniti et al., 1988). Education appears unrelated to DS scores (Erkinjuniti et al., 1988). African–American patients score higher on the DS than white patients (Hargrove, Stoeklin, Haan, & Reed, 1998). The DS has been translated and validated in Chinese, Korean, and Czech (Lam et al., 1997; Lee et al., 1999; Vajdickova, Kolibas, Heretik, & Kosc, 1995).

Clinical Uses

The DS offers a blend of items commonly found on mental status exams, activities of daily living scales, and instrumental activities of daily living scales. It is quick and easy to administer, and additionally provides a quantification of the degree of dementia severity. As such, it is ideal for use by general practitioners and specialized medical and mental health care professionals to gauge initial status, as well as to track disease progression. The DS may also provide more useful information in a clinical setting than the MMSE and other cognitive assessment scales (Mant, Eyland, Pond, Saunders, & Chancellor, 1988) because it measures functional aspects of dementia.

Cross References

- ► Activities of Daily Living Scale
- ► Alzheimer's Disease
- ► CERAD
- ► Clinical Dementia Rating
- ► Dementia
- ► Dementia Rating Scale
- ► Lawton-Brody Instrumental Activities of Daily Living Scale

References and Readings

Blessed, G., Tomlinson, B. E., & Roth, M. (1968). The association between quantitative measures of dementia and of senile changes in the cerebral gray matter of elderly subjects. *British Journal of Psychiatry, 114*(512), 797–811.

Blessed, G., Tomlinson, B. E., & Roth, M. (1988). Blessed-Roth Dementia Scale (DS). *Psychopharmacology Bulletin, 24*(4), 705–708.

Chandler, M. J., Lacritz, L. H., Hynan, L. S., Barnard, H. D. Allen, G., Deschner, M., et al. (2005). A total score for the CERAD neuropsychological battery. *Neurology, 65*(1), 102–106.

Cole, M. G. (1990). Interrater reliability of the Blessed Dementia Scale. *Canadian Journal of Psychiatry, 35*(4), 328–330.

Eastwood, M. R., Lautenschlaegar, E., & Corbin, S. (1983). A comparison of clinical methods for assessing dementia. *Journal of the American Geriatrics Society, 31*(6), 342–347.

Erkinjuntti, T., Hokkanen, L., Sulkava, R., & Palos, J. (1988). The Blessed Dementia Scale as a screening test for dementia. *International Journal of Geriatric Psychiatry, 3*, 267–273.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). Mini Mental State: A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research, 12*(3), 189–198.

Hargrove, R., Stoeklin, M., Haan, M., & Reed, B. (1998). Clinical aspects of Alzheimer's disease in Black and White patients. *Journal of the National Medical Association, 90*, 78–84.

Hendrie, H. C., Hall, K. S., Brittain, H. M., Austrom, M. G., Farlow, M., Parker, J., et al. (1988). The CAMDEX: A standardized instrument for the diagnosis of mental disorder in the elderly: A replication with a U.S. Sample. *Journal of the American Geriatric Society, 36*(5), 402–408.

Juva, K., Maela, M., Erkinjuntti, T., Sulkava, R., Yukoski, R., Valvanne, J., et al. (1997). Functional assessment scales in detecting dementia. *Age and ageing, 26*(5), 393–400.

Lam, L. C. W., Chiu, H. F. K., Li, S. W., Chan, W. F., Chan, C. K. Y, Wong, M., et al. (1997). Screening for dementia: A preliminary study on the validity of the Chinese version of the Blessed-Roth Dementia Scale. *International Psychogeriatrics, 9*(1), 39–46.

Lawson, J. S., Rodenburg, M., & Dykes, J. A. (1977). A dementia rating scale for use with psychogeriatric patients. *Journal of Gerontology, 32*, 153–159.

Lee, D. Y., Yoon, J. C., Lee, K. U., Jhoo, J. H., Kim, K. W., Lee, J. H., et al. (1999). Reliability and validity of the Korean version of Short Blessed Test (SBT-K) as a dementia screening instrument. *Journal of the Korean Neuropsychiatric Association, 38*, 1365–1375.

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford.

Mant, A., Eyland, E. A. Pond, D. C., Saunders, N. A., & Chancellor, A. H. B. (1988). Recognition of dementia in general practice: Comparison of general practicioners' opinion with assessments using the Mini-Mental State Examination and the Blessed Dementia Scale. *Family Practice, 5*(3), 184–188.

Morris, J. C., Mohs, R. C., Rogers, H., Fillenbaum, G., & Heyman, A. (1988). Consortium to establish a registry for Alzheimer's disease (CERAD) clinical and neuropsychological assessment of Alzheimer's disease. *Psychopharmacology Bulletin, 24*(4), 641–651.

Roth, M., Tym, E., Mountjoy, C. Q., Huppert, F. A., Hendrie, H., Verma, S., et al. (1986). CAMDEX: A standardized instrument for the diagnosis of mental disorders in the elderly with special reference to early detection of dementia. *British Journal of Psychiatry, 149*, 698–709.

Stern, Y., Hesdorffer, D., Sano, M., & Mayeux, R. (1990). Measurement and prediction of functional capacity in Alzheimer's disease. *Neurology, 40*(1), 8–14.

Stern, Y., Mayeux, R., Sano, M., Hauser, W. A., & Bush, T. (1987). Predictors of disease course in patients with probable Alzheimer's disease. *Neurology, 37*(10), 1649–1653.

Vajdickova, K., Kolibas, E., Heretik, A., & Kosc, M. (1995). Application of behavioural scale in the diagnosis of dementia of advanced age. *Ceska a Slovenska Psychiatrie, 91*(1), 7–14.

Blessed–Roth DS

► Blessed Dementia Scale

Blindsight

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Short Description or Definition

Blindsight is a neuropsychological disorder that results from damage to the primary visual cortex (V1). Such localized cortical damage produces localized visual impairment in the patient's visual field contralateral to the site of the damage. Critically, despite the nominal loss of vision, patients with blindsight preserve the ability to detect and discriminate visual stimuli presented in the impaired region of their visual field. Lawerence Weiskrantz's (1986) observation of this ability to "see" stimuli in a "blind" visual field led him to refer to this disorder as "blindsight."

Categorization

There are two types of blindsight, termed Type I and Type II. Patients with Type I blindsight report no conscious awareness of stimuli presented in the damaged region of their visual field, yet preserve the ability to detect stimuli presented there. Patients with Type II blindsight, report a faint conscious perception of stimuli in the damaged region of their visual field, yet preserve the ability to detect stimuli with higher precision than their conscious perception.

Epidemiology

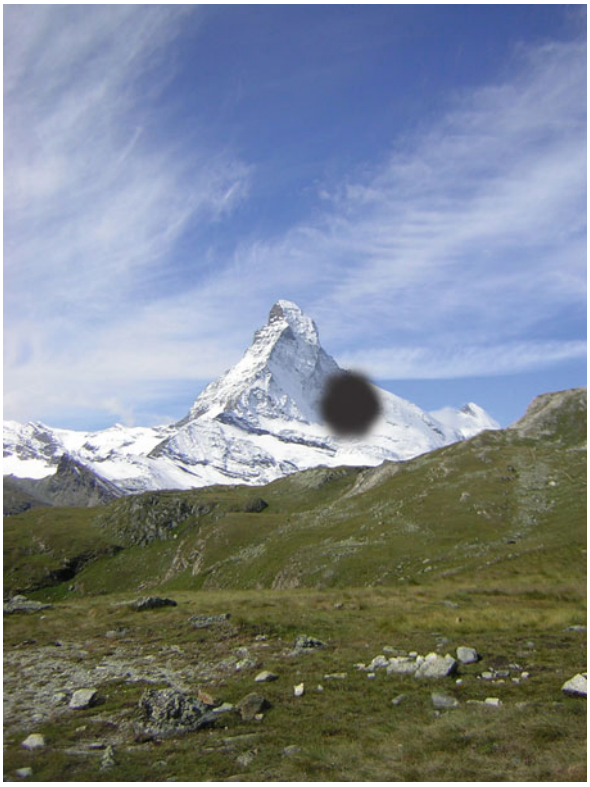
Blindsight results from brain damage to the primary visual cortex (V1) located in the posterior region of the occipital cortex, typically caused by a tumor, a hemorrhage, or some sort of brain trauma.

Natural History, Prognostic Factors, Outcomes

The first cases of blindsight were observed in war veterans with damage to their occipital lobe (Poppel et al., 1973). These veterans had no conscious perception of stimuli in the damaged portion of their visual field, yet were able to track a moving light presented there. The most extensive experimental work in this area was completed with patient DB, who was diagnosed in the 1970s. DB's case is extensively reviewed in the seminal book on blindsight (Weiskrantz, 1986) is written.

Neuropsychology and Psychology of. . .. (syndrome/illness)

The area of impaired vision in the visual field of a blindsight patient is referred to as a scotoma – defined as an island of visual loss surrounded by an area of normal visual acuity (Fig. 1). It is important to note that the visual impairment manifests in the region of the visual



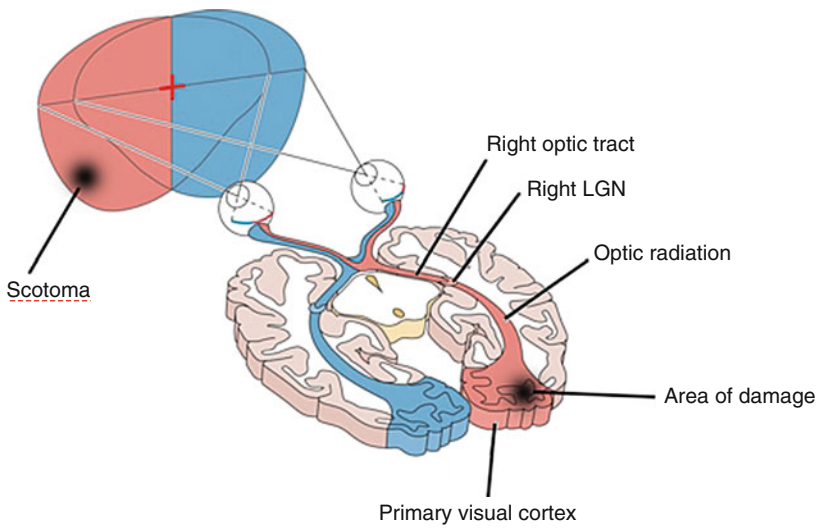
Blindsight. Figure 1 The visual perception of a scotoma

field contralateral to the hemisphere where the brain injury has occurred. For example, damage to the left hemisphere of V1 results in impairment to the right visual field. Typically, because early visual brain areas are retinotopically mapped, the extent of the damage to occipital lobe corresponds to the extent of the impairment in the visual field. For example, if an entire hemisphere's occipital lobe is ablated then the entire contralateral visual field is damaged – a condition is termed hemianopia. Likewise, if a quarter of V1 is damaged (i.e., one half of one hemisphere's occipital lobe), a quarter of the contralateral visual field is damaged – a condition is termed quadranopia. Figure 2 illustrates the pattern of visual impairment and the corresponding scotoma that arises when only a small region of occipital lobe is damaged. Although a patient with such a pattern of sparing and loss might experience blindsight in such a restricted scotoma, the most interesting cases of blindsight have been where patients have lost *all* conscious visual experience, that is, there is injury covering early visual brain areas across both hemispheres.

Note that not all patients with scotomas experience blindsight. The functional definition is that despite an absence of conscious perception, patients with blindsight retain the ability to detect and discriminate stimuli presented in their scotoma. For example, patients can localize moving and stationary stimuli using saccades or pointing. They can also discriminate line orientations, detect motion, and recent evidence suggests some patients can even differentiate between different wavelengths of color and form presented simultaneously in their scotoma (Trevethan, Sahraie & Weiskrantz, 2007). Primate studies support the claim that this unconscious perception is not subserved by islands of undamaged tissue. When the striate cortex of primates is cortically ablated, like humans, they have no conscious perception of stimuli presented in their scotoma, but do retain unconscious perception. The strongest neural evidence supporting the existence of blindsight comes from the identification subcortical connections from the Lateral Geniculate Nucleus (LGN) directly to the extrastriate cortex. These pathways, unaffected by V1 damage, are potential mediators for the unconscious visual perception observed in blindsight. The identification of this pathway has prompted a fascinating debate regarding the role of V1 in the neural representation of consciousness. Scientists have posited that if there is perception, but not conscious perception, without V1, then V1 must play a critical role as a neural correlate of consciousness; this remains an active area of research (for review see Tong, 2003).

Evaluation

Because blindsight patients experience no conscious awareness of stimuli presented in their scotoma,



Blindsight. Figure 2 The relationship between a region of damage in visual cortex (V1) and the corresponding impairment in the visual field (Adapted from Bear, Connors, & Paradiso, 2006)

experimenters rely on a two-alternative forced-choice (2AFC) procedure to diagnose and evaluate the symptoms of the disorder. The 2AFC procedure, typically used to assess behavioral performance in nonspeaking primates, presents patients with a target stimulus, a probe stimulus (matched to target), and a distractor stimulus (non-matched to target); the target can be presented either prior to or simultaneously with the paired probe and distractor. The patient must select either the probe or the distractor as matching the target. The patient is not allowed to respond, "I don't know," so in this way the task is a forced-choice. In a variant of this procedure used to assess blindsight, experimenters present an image both to the scotoma and the hemisphere of normal visual acuity then ask subjects to report whether the two stimuli are the same or different (Weiskrantz, 1986). Given that chance performance in these procedures is 50%, it is interesting to note that patients estimate their success rate on these tasks to be roughly 50%, but in reality their success rate is closer to 90%, illustrating the disconnect between conscious and unconscious vision in blindsight. Similar results have been observed using a somewhat different procedure designed to measure the unconscious perception of visual motion in blindsight patients: saccades tracking or pointing in which a moving object is presented within the visual field of the scotoma and the patient is asked to track the object with their eyes or with their finger.

Treatment

There is a period of spontaneous recovery for neurovisual lesions, typically up to 3 months post lesion, but has been reported to extend to up to a year. Following this period, active discrimination of stimuli presented in the scotoma seems to be the best strategy for improvement in humans (Sahraie, Trevethan, MacLeod, Murray, Olson, & Weiskrantz, 2006), and nonhuman primates (Dineen & Keating, 1981). As such, blindsight patients that participate as experimental subjects sometimes show large improvements in their visual discrimination abilities. For example patient DB, the first blindsight patient studied extensively (Weiskrantz, 1986), was recently retested, 30 years after his right striate cortex was surgically removed during treatment of a nonmalignant venous tumor. Patient DB can now discriminate complex circular forms presented in his scotoma, for example, he can discriminate a circle from an oval. Previously the ability to distinguish form was accounted for by DBs ability to distinguish line orientations; however, this explanation cannot account for now-present circular form discrimination (Trevethan et al., 2007). One possibility is that DB's improvement in form perception is the result of the large number of hours DB spent completing experimental testing.

One point to consider when diagnosing or treating patients with blindsight is that Type I patients have *no* conscious access to the stimuli presented in the scotoma. Consequently, experimenters should be cautious in asking for specific answers when running a 2AFC task in that this task may be distressing to a patient who experiences no conscious visual perception. That is, they may find such a task irrelevant to their personal experience.

Cross References

- ▶ Cortical Blindness
- ▶ Hemianopia
- ▶ Scotoma
- ▶ Visual Field Deficit

References and Readings

Bear, M. A., Connors, B. W., & Paradiso, M. A. (2006). *Neuroscience exploring the brain.* Baltimore, MD: Lippincott Williams & Wilkins.

Dineen, J., & Keating, E. G. (1981). The primate visual system after bilateral removal of striate cortex. Survival of complex pattern vision. *Experimental Brain Research. Experimentelle Hirnforschung. Expérimentation Cérébrale, 41*(3–4), 338–345.

Pöppel, E., Held, R., & Frost, D. (1973). Residual visual function after brain wounds involving the central visual pathways in man. *Letters to Nature, 243*, 295–296.

Sahraie, A., Trevethan, C. T., MacLeod, M. J., Murray, A. D., Olson, J. A., & Weiskrantz, L. (2006). Increased sensitivity after repeated stimulation of residual spatial channels in blindsight. *Proceedings of the National Academy of Sciences of the United States of America, 103*(40), 14971–14976.

Tong, F. (2003). Primary visual cortex and visual awareness. *Nature Reviews. Neuroscience, 4*(3), 219–229.

Trevethan, C. T., Sahraie, A., & Weiskrantz, L. (2007). Can blindsight be superior to 'sighted-sight'?. *Cognition, 103*(3), 491–501.

Weiskrantz, L. (1986). *Blindsight: A case study and its implications.* Net Library, USA: Oxford University Press.

Werner, J. S., & Chalupa, L. M. (2004). *The visual neurosciences.* Cambridge, MA: MIT Press.

Blitz-Nick-Salaam Krämpfe

▶ West Syndrome

Block Design

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Synonyms

Kohs blocks

Description

The Block Design (BD) test is a subtest from the Wechsler corpus of intelligence tests that requires the examinee to use three-dimensional blocks to construct a model from a two-dimensional stimulus card. Blocks consist of sides that are all white, all red, or diagonally half red and white. Performance is timed. Although bonus points are awarded for speed, the score is either all or none, that is, a score is awarded only if the model is correctly produced within the prescribed time limit.

Historical Background

Hutt (1925) notes that the first documented use of block construction as a psychological test was by Francis N. Maxfield, working at the University of Pennsylvania Psychology Laboratory and Clinic, who devised a "color cube" test to study "imageability in children". The procedures devised by Maxfield were also used by Clara Town (1921, cited in Hutt, 1925). Both of these researchers were interested in studying analytic problem-solving strategies in children. However, it was Samuel Calmin Kohs (1916–1960) who derived the Block Design (BD) test that was ultimately adapted by David Wechsler. It appears that Maxfield, Town, and Kohs used the same commercially available blocks, that is, all blocks were constructed with four colors – red, white, blue, and yellow. Kohs procedure differed from Maxfield and Town in that he asked children to use blocks to copy two-dimensional designs printed on stimulus cards rather than from models constructed by the examiner using identical blocks, a method adopted by Wechsler. Kohs (1920, 1923) specifically used block construction as a means to assess intelligence. Consistent with the prevailing views of the day, Kohs viewed intelligence as a unitary or global construct. The Kohs BD test consisted of a series of 17 designs (culled from a corpus of 35 original designs). Kohs (1920, 1923) clearly viewed his test as equal to the existing Binet scales in measuring general intellectual ability. He also viewed the "performance" (Kohs, 1920) or nonverbal nature of his test as a means to assess intelligence in children where it was either not possible or problematic to use language or language related tests. As adopted by Wechsler later, Kohs awarded bonus points for speed. Interestingly, a separate scoring system was also derived to measure "moves" or "each separate and distinct change in the position of the block" (Kohs, 1920). All these early researchers readily acknowledged the multidimensional aspect of their block construction procedures and commented on the qualitative features of children's block construction strategies.

Psychometric Data

Successful completion of the BD test requires a host of cognitive abilities (Kramer, Kaplan, Blusewicz, & Preston, 1991) including specific analytic and synthetic problem-solving strategies (Schorr, Bower, & Kiernan, 1982). *Analytic strategies* refer to mental segmentation of the stimulus design into individual blocks. Upon mentally dividing the blocks into segments the blocks are subsequently arranged to match each unit. This strategy might capitalize on the presence of perceptual edge cues and implicit grid information when constructing the design (Kaplan, 1988; Kaplan et al., 1991). *Synthetic strategies* emphasize the design as a whole, and may not rely on segmentation for test completion. Examinees who utilize this strategy focus primarily on the gestalt or overall form of the design. Specific BD test items tend to "pull" for one strategy versus the other. However, over reliance on either problem-solving approach will ultimately lower an examinee's test score and could be highly suggestive of either focal or lateralized neurological insult.

The BD test is often viewed as a measure of so-called "*constructional apraxia*" (Kleist, 1923, cited in Benton & Tranel, 1993) and has been naively associated with right parietal brain damage (Kaplan, 1988). Clear evidence

of the multidimensional cognitive skills necessary for optimal performance on the BD test comes from two sources: patients with cerebral disconnection (Geschwind, 1979; Kaplan, 1988); and patients with focal brain lesions. For example, patients who have undergone a commissurotomy (Geschwind, 1979; Kaplan, 1988) provided a unique opportunity to study BD problem-solving strategies because these patients serve as their own controls. Since these patients have undergone resection of the corpus callosum and the anterior commissure, sensory information cannot be transferred between the hemispheres. Illustrations provided by Geschwind (1979) and Kaplan (1988) show that when commissurotomized patients use their right hand, that is, when BD constructions are guided by the left hemisphere with no input from the right hemisphere, the inherent 2 × 2 or 3 × 3 matrix is violated and there is a tendency for blocks to pile up on the right side of the design reflecting an inattention of left hemispace suggestive right hemisphere dysfunction (Kaplan, 1988, Figures 1–2). Very different errors occur with commissurotomized patients attempting BD using their left hand, that is, when constructions are guided by the right hemisphere with no input from the left hemisphere. Now, the 2 × 2 or 3 × 3 grid matrix is rarely violated. However, blocks tend to be rotated so that the internal details of individual blocks do not match the model. Thus, Geschwind (1979) and Kaplan (1988) show that regardless of which hand is used commissurotomized patients produce zero point responses, but the underlying brain-behavior relationships responsible for these response strategies are very different. Kaplan, Palmer, Weinstein and Baker (1981) noted that similar behavior occurs in patients with focal right and left hemisphere lesions. Patients with right-sided lesions often break the 2 × 2 or 3 × 3 matrix inherent in the stimulus resulting in highly distorted responses, blocks continue to collect on the right side of hemi-space, and constructions are often initiated on the right-side with patients working from right to left. Patients with left-sided lesions respect the inherent grid configuration of the BD stimuli. These patients often make single-block, rotational errors or misalign internal details (Kaplan et al., 1991) with responses initiated on the left side of hemi-space.

Clinical Uses

Kaplan and colleagues (Kaplan, 1988; Kaplan et al., 1991) have suggested a number of additional testing and scoring procedures to extract detailed information from the BD test performance. These are listed below and are part of the WAIS-R-NI corpus (Kaplan et al., 1991).

1. *Providing Additional Blocks* – Rather than constraining the examinee's performance by providing only 4 or 9 blocks as prescribed by the Wechsler test manual, Kaplan et al. (1991) suggests presenting the patient with 9 blocks on all 4-block test items and 12 blocks on all 9-block test items. Attempting to construct designs with too few or too many blocks conveys additional information about possible spatial as well as executive impairment.
2. *Flow Charting* – Documenting the patient's performance with a flow chart is mandatory to the cogent analysis of BD test performance. Examples of the rich data which can be obtained with a flow chart are illustrated by Kaplan et al. (1991, Figure 6). As described above, focal right-hemisphere lesioned patients tend to break the 2 × 2 or 3 × 3 grid configuration of the stimulus matrix and often produce distorted responses. Kaplan et al. (1991) provides examples of BD constructions produced by right-anterior and right-posterior lesioned patients in Figures 6c and 6d, respectively (see p. 90). The 3 × 3 grid configuration is broken by both patients. However, the construction of a patient with a right-posterior lesion is measurably more distorted than the construction produced by a right-anterior lesioned patient suggesting greater perceptual-spatial impairment. Thus, as suggested many years ago by Kohs (1920, 1923) an analysis of BD "moves" provides important information.
3. *Errors Subtypes* – The WAIS-R-NI (Kaplan et al., 1991) suggests a variety of error scores that supplement the traditional total Wechsler scale score. The scoring techniques described below are designed to supplement standardized scoring procedures and help in identifying underlying brain pathology.
   (a) **Rotational errors** – scored when a block's surface coloring is incorrect. This type of internal detail error could be associated with a left hemisphere lesion.
   (b) **Broken configuration** – scored when the 2 × 2 or 3 × 3 grid matrix of the design is violated. As noted above, such errors are often seen in patients with right hemisphere lesions.

While rotational and broken configuration errors often occur in patients with circumscribed stroke, patients with epilepsy (Zipf-Williams, 2000) or brain injury (Wilde et al., 2000) lateralized to one side of the brain may also make these errors.

(c) **Orientation errors** – scored when a block(s) is incorrectly oriented, that is, when the final product or elements of the final product are shifted or misoriented about 30 degrees in relation to the model. Spatial, perceptual, or executive problems might underlie this difficulty.

(d) **Perseverations** – scored when incorrect block placements persist either within or between successive BD trials. Gross perseverative behavior is often seen in patients with frontal lobe or frontal systems lesions. Less severe perseverative behavior might occur in conjunction with rotational and broken configuration errors and may suggest dysexecutive behavior associated with a specific brain region.

(e) **Stimulus bound** – examples include instances when the examinee is drawn to build their construction either right next to or under the stimulus booklet or even pile blocks on top of the stimulus booklet. Less egregious, but no less important stimulus bound errors occur when patients are aware of but unable to self-correct errors.

(f) **Response latency** – patients with bradyphrenia may ultimately produce a correct construction and might be able to self-correct errors but may complete a correct design only after the time limit as prescribed in the test manual has passed. Such behavior might be associated with subcortical syndromes. However, slow time to completion often occurs in patients with alcohol abuse, brain injury, multiple sclerosis, or epilepsy.

(g) **Start position** – using a flow chart, documenting the start position of the first block also allows for examination of a "preferred" side, and can be indicative of lateralized brain dysfunction (Akshoomoff-Haist et al., 1989).

Block Design Use with Additional Populations:

Healthy and Pathological Aging

An observed pattern of developmental cognitive change associated with age is the relative stability of verbal abilities coupled with a significant diminution in visuospatial and constructional abilities. Evidence suggests that the BD test differentiates between younger and older adults (Kaufman, 1990; Troyer et al., 1994), but the specific cognitive functions that underlie this behavior have been debated. Joy et al. (2001) provided a comprehensive evaluation of the reported age-related decline in BD test performance and offered normative data for the clinical interpretation of BD in healthy older adults. In addition to standard pass–fail scoring, these researchers also utilized proportional scoring methods as well as the supplemental measures detailed in the WAIS-R-NI. Results confirmed a moderate negative correlation between standard BD score and age ($r = 0.455$); however, the use of proportion scores, elimination of time constraints, and termination of time-bonuses significantly reduced the documented age differences. These authors interpreted this finding as evidence for less severe age-related impairment in visuospatial and constructional abilities in healthy older adults than traditional scoring techniques suggest. In general, it is important to carefully consider the role of psychomotor slowing and error types when administering the standard block design test to healthy older adults in order to avoid differently penalizing individuals based on age. Older adults diagnosed with a neurodegenerative disorder exhibit different patterns of errors depending upon their neuropsychological profile and diagnosis. Stimulus bound errors, broken configurations, and psychornotor slowing are all more prevalent in individuals diagnosed with a dementia relative healthy older adult controls.

Cross References

- ▶ Constructional Apraxia
- ▶ Visuoconstruction

References and Readings

Akshoomoff-Haist, N. A., Delis, D. C., & Kiefner, M. G. (1989). Block constructions of chronic alcoholic and unilateral brain-damaged patients: A test of the right hemisphere vulnerability hypothesis of alcoholism. *Archives of Clinical Neuropsychology, 4*, 275–281.

Benton, A., & Tranel, A. (1993). Visuoperceptual, Visuospatial, and Visuoconstructional Disorders. In K. M. Heilman & E. Valenstein (Eds.) *Clinical Neuropsychology* (3rd ed.). New York: Oxford University Press.

Caplan, B., & Caffery, D. (1992). Fractionating block design: Development of a test of visuospatial analysis. *Neuropsychology, 6*, 385–394.

Geschwind, N. (1979). Specializations in the human brain. *Scientific American, 241*, 180–199.

Hutt, R. B. W. (1925). Standardization of a color cube test. *The Psychological Clinic, 16*, 77–97.

Joy, S., Fein, D., Kaplan, E., & Freedman, M. (2001). Quantifying qualitative features of block design performance among healthy older adults. *Archives of Clinical Neuropsychology, 16*, 157–170.
Kaplan, E. (1988). A process approach to neuropsychological assessment. In T. Boll & B. R. Bryant (Eds.), *Clinical neuropsychology and brain function: Research. Measurement, and practice: Master lectures.* Washington, DC: The American Psychological Association.
Kaplan, E., Palmer, E. P., Weinstein, C., & Baker, E. (1981). Block Design: A brain-behavior based analysis. Paper presented at the annual European meeting of the International Neuropsychological Society, Bergen, Norway.
Kaplan, E., Fein, D., Morris, R., & Delis, D. (1991). *The WAIS-R as a neuropsychological instrument.* San Antonio, TX: The Psychological Corporation.
Kaufman, A. S. (1990). *Assessing adolescent and adult intelligence.* Boston: Allyn & Bacon.
Kohs, S. C. (1920). The block design tests. *Journal of Experimental Psychology, 3*, 357–376.
Kohs, S. C. (1923). *Intelligence measurement.* New York: Macmillan.
Kramer, J. H., Kaplan, E., Blusewicz, M. J., & Preston, K. A. (1991). Visual hierarchical analysis of block design configural errors. *Journal of Clinical and Experimental Neuropsychology, 13*, 455–465.
Lezak, M., Howison, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
Schorr, D., Bower, G. H., & Kiernan, R. (1982). Stimulus variables in the block design task. *Journal of Consulting and Clinical Psychology, 50*, 479–487.
Troyer, A. K., Cullum, C. M., Smernoff, E. N., & Kozora, E. (1994). Age effects on block design: Qualitative performance features and extended-time effects. *Neuropsychology, 8*, 95–99.
Wilde, M. C., Boake, C., & Sherer, M. (2000). Wechsler Adult Intelligence Scale-Revised Block Design broker configuration errors in non-penetrating traumatic brain injury. *Applied Neuropsychology, 7*, 208–214.
Zip-Williams, E. M., Shear, P. K., Strongin, D., et al., (2000). Qualitative Block Design performance in epilepsy patients. *Archives of Clinical Neuropsychology, 15*, 149–157.

Blocq's Disease

► Astasia-Abasia

Blood Alcohol Concentration

► Blood Alcohol Level

Blood Alcohol Content

► Blood Alcohol Level

Blood Alcohol Level

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Synonyms

Blood alcohol concentration; Blood alcohol content

Definition

Measure of alcohol in the blood.

Current Knowledge

Blood alcohol levels are typically expressed as milligrams or grams of ethanol per deciliter (e.g., 100 mg/dL or 0.10 g/dL). A level of 20–30 mg/dL typically results from the ingestion of one to two drinks. One drink corresponds to 340 mL (12 oz.) of beer, 115 mL (4oz) of wine, and 43 mL (1.5 oz) of a shot. Blood alcohol levels as low as 20–80 mg/dL can lead to decreased inhibitions and decreased cognitive and motor performance, while levels of 300–400 mg/dL can lead coma or death. Blood alcohol levels typically correlate inversely with cognitive and motor performance (i.e., as blood alcohol levels increase, cognitive and motor performance decrease). Specifically, increased blood alcohol levels correlate with slower reaction time and inversely correlate with frontal executive function. Additionally, speed of cognitive performance recovers as alcohol levels return to drug-free levels; however, accuracy may continue to remain impaired.

Cross References

► Coma
► Executive Function
► Frontal Lobe

References and Readings

Domingues, S. C., Mendonca, J. B., Laranjeira, R., Nakamura-Palacios, E. M. (2009). Drinking and driving: A decrease in executive frontal functions in young drivers with high alcohol concentration. *Alcohol, 43*(8), 657–664.

Schweizer, T. A., & Vogel-Sprott, M. (2008). Alcohol-impaired speed and accuracy of cognitive functions: A review of acute tolerance and recover of cognitive performance. *Experimental and Clinical Psychopharmacology, 16*(3), 340–350.
Harrison's Online – Harrison's principles of internal medicine, 16th edn. Chapter 372: Alcohol and Alcoholism.

Blood Clot

► Hematoma

Blood Flow Studies

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Synonyms

Duplex/Doppler ultrasound (US); Vascular ultrasound

Definition

A blood flow study is a noninvasive imaging technique which is used to measure blood flow and pressure through arteries, veins, or chambers and valves of the heart. Doppler US may be used to diagnose vascular conditions such as thrombosis, vascular stenosis, valvular heart disease, peripheral vascular and aneurysmal disease. It may also be used to evaluate the condition of bypass grafts and blood flow to transplanted organs.

Current Knowledge

Blood flow studies such as Doppler US uses a transducer that sends high-frequency sound waves which bounce off of solid objects including red blood cells. The sound waves are reflected back to the transducer. Moving objects, such as the red blood cells, cause a change in pitch of the sound waves (also known as the "Doppler effect"). These reflected waves are sent to and processed by a computer which translates the waves into pictures or graphs. The images are representative of the flow of blood through the vessel.

There are four different types of Doppler US studies currently being utilized by physicians. Continuous wave Doppler is typically used at the bedside and only produces sound from the transducer which the practitioner uses to listen for blockage or stenosis of the vessel. Duplex Doppler produces both a picture of the blood vessel and a graph representing the speed and direction of blood flow (hence the name "duplex"). Color Doppler uses a computer to convert the Doppler sounds into colors and overlay those colors on an image of the blood vessel. Power Doppler is more sensitive than color Doppler in detecting blood flow. It combines the results given by color Doppler with duplex Doppler. It is commonly used to evaluate the flow of blood through vessels within solid organs.

Transcranial Doppler (TCD) is used to measure the blood flow through the brain's blood vessels. It is becoming more widely used to evaluate for embolus, stenosis, vasospasm, and the risk of stroke.

Cross References

► Doppler Ultrasound
► Regional Cerebral Blood Flow
► Transcranial Doppler

References and Readings

Blaivas, M. (2007). Ultrasound in the detection of venous thromboembolism. *Critical Care Medicine, 35*(Suppl. 5), S224–234.
Hamper, U. M., DeJong, M. R., & Scoutt, L. M. (2007). Ultrasound evaluation of the lower extremity veins. *Radiologic Clinics of North America, 45*(3), 525–547.
Rubens, D. J., Bhatt, S., Nedelka, S., & Cullinan, J. (2006). Doppler artifacts and pitfalls. *Radiologic Clinics of North America, 44*(6), 805–835.
Weber, T. M., Lockhart, M. E., & Robbin, M. L. (2007). Upper extremity venous Doppler ultrasound. *Radiologic Clinics of North America, 45*, 513–524.

Blood Oxygen Level-Dependent

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Synonyms

BOLD

Definition

BOLD imaging is a version of magnetic resonance imaging that depends on the different magnetic properties of oxygenated versus deoxygenated hemoglobin and thus, indirectly, on variations in local tissue perfusion. The utility of BOLD imaging for functional magnetic resonance imaging (fMRI) also depends on the physiological phenomenon by which metabolically active cerebral tissue "demands" more perfusion than less-active tissue. Thus, populations of neurons that are particularly active during a cognitive or motor task actually elicit a relative surplus of perfusion which, in turn, results in an increase in the ratio of oxygenated to deoxygenated hemoglobin, detectable as a change in the BOLD signal.

Historical Background

As early as 1890, Roy and Sherrington noted that regional cerebral blood flow increased in areas of neural activity. This increase in perfusion became detectable in vivo with the advent of positron emission tomography (PET), in which radioactive tracers are injected and their emitted radiation detected. It was not until the discovery of BOLD contrast by Ogawa and colleagues at Bell Laboratories, however, that it was possible to measure these neurally mediated changes in flow without radiation exposure using MRI and, hence, to allow a broader range of studies with normal control subjects and with repeated studies of the same individuals.

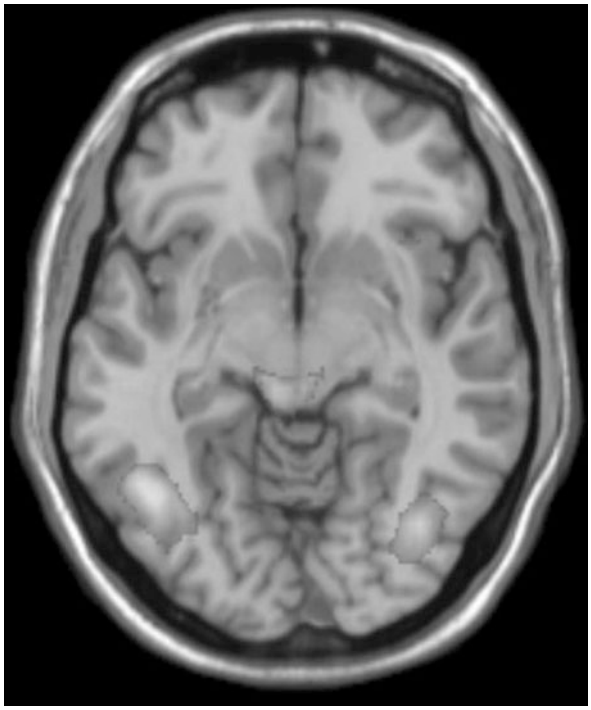
Current Knowledge

Because of its dependence on the state of oxygenation of the blood, the BOLD signal is several steps removed from the typical phenomenon of interest: changes in neural activity. All measures of cerebral blood flow are indirect, in the sense that they can be influenced by cardiovascular factors (e.g., changes in cardiac output, vascular resistance) as well as changes in metabolic demand by neural (as well as nonneural tissue, e.g., glia) tissue. But BOLD has the additional limitation that it represents the relationship between oxygen delivery and oxygen extraction, rather than oxygen consumption itself – a more direct measure of tissue metabolic activity. In addition, there is a delay of several seconds between the changes in neural activity and the changes in associated blood flow. Thus, BOLD imaging typically involves mathematical modeling of the "hemodynamic response function" (the increase and subsequent return to baseline of flow associated with a neural event) to allow the BOLD signal to be related to specific behavioral and cognitive events that are assumed to require neural processing. The hemodynamic response function can be modeled in a normative sense (i.e., the shape of the blood flow response in a "typical" organism) or in the individual subject.

Although the BOLD signal is not a direct measure of neural activity, a number of studies that have simultaneously recorded direct measures of cerebral blood flow or actual neuronal firing rates have suggested that, in most circumstances at least, there is a reasonably tight coupling of neural activity and the BOLD signal. Less research, however, has been conducted on animals or humans with known abnormalities of cardiovascular function, so that the possibility remains that the coupling assumptions underlying the use of this method are violated in certain research contexts.

The BOLD signal provides relative information about changes in flow, rather than an absolute measure of perfusion in cc/gm/min as can be obtained from PET, or perfusion MRI. However, when contrasted to or subtracted from some control condition, the change in the BOLD signal can help to localize the brain areas that are specifically active in the condition of interest when compared to the control condition.

BOLD imaging studies in human neuroscience most often make use of one of the two common experimental designs: blocked or event-related (Fig. 1). In the blocked design, the subject is asked to perform a particular cognitive or motor task in blocks that alternate with other blocks of a contrasting task or rest. The BOLD signal is then statistically averaged across the two types of blocks, and a measure of the difference between them is mapped onto each voxel in the MR image, thus showing those areas of the brain that had the largest change in BOLD signal between the two conditions. A contrasting rest block is typically used when one is interested in the brain areas involved in all aspects of the task, although this method has been criticized because there is no standardization of the subject's mental activity during rest. Alternatively, if one is interested in the brain areas involved with a specific task process, one might alternate the experimental task with a control task that shares most but not all of the features of the experimental task. For example, if a research subject performs alternating blocks of finger tapping in response to a visual signal versus viewing the visual signal without tapping, areas involved in the perception of the visual signal will tend to be canceled out across conditions whereas neural networks specifically involved in the tapping response will be highlighted. In this way, a wide range of cognitive and



Blood Oxygen Level-Dependent. Figure 1 This axial slice of the brain shows the areas of most significant BOLD activation across 18 control subjects, obtained while they attended to three randomly moving visual stimuli. Higher visual areas in the occipital cortices (motion areas V5/MT+) and superior colliculi show the greatest activation

motor tasks have been studied in normal subjects as a way of localizing the neural networks involved in their performance; and in patient subjects, as a way of exploring how that localization may have been altered by pathology or recovery.

In event-related BOLD designs, experimental trials of different types can be delivered in a random sequence and averaged in a time-locked fashion. The timing between trials is sometimes "jittered" (i.e., randomly varied) so that, even though the hemodynamic responses from individual trials overlap, their individual effects can be separately modeled (deconvolved), by incorporating the known temporal spacing between them.

More recently, the BOLD signal has been used to understand how activities in different parts of the brain are interrelated. Modern neuroscience posits the presence of distributed neural networks, rather than focal regions, supporting specific cognitive and motor processes. Since components of these neural networks may be separated by considerable distances, it is of interest to understand how they communicate with each other in the performance of specific mental activities. By assessing how strongly changes in the BOLD signal in different regions are correlated over time, one can derive a measure of "functional connectivity," assessed either at rest or during the performance of specific tasks. Measures of functional connectivity do not specify the actual anatomical connections between regions, but merely demonstrate the degree to which their activity levels are linked over time.

With any of these experimental designs, the BOLD signal must also be mapped to an anatomical model of the brain. This is done by modeling the signal separately in each voxel of the MRI image and then contrasting the signal in each voxel between the experimental conditions of interest. This may require additional manipulations, such as warping each subject's image to a standard template, "smoothing" the signal so that the activity in collections of voxels rather than individual voxels is highlighted, and deriving statistical maps that code the reliability of the change of interest across brain regions and individual subjects. Several forms of computer software are available for processing raw fMRI data into analyzable maps and statistical results.

Although BOLD and other functional MRI techniques are extremely powerful research tools, they incorporate a large number of data transformations and assumptions between the raw signal acquisition and interpretations at the level of brain activity and behavior. As noted above, conclusions reached by these techniques can be undermined by alterations in the coupling between neural activity and blood flow, by failure to accurately understand the cognitive and motor processes required by the task, and by invalid application of the many analytical and statistical methods that transform the measured BOLD signal into statistical maps of brain activity. Thus, investigators seeking to use these methods typically need extensive training in their application.

Cross References

- ▶ Functional Imaging
- ▶ Magnetic Resonance Imaging

References and Readings

Buxton, R. B., Uluda, K., Dubowitz, D. J., & Liu, T. T. (2004). Modeling the hemodynamic response to brain activation. *Neuroimage, 23*(Suppl. 1), S220–233.

Jamie Shorey, History of functional MRI. Retrieved October 15, 2007 from http://www.ee.duke.edu/~jshorey/MRIHomepage/fmri.html

Huettel, S. A., Song, A. W., & McCarthy, G. (2004). *Functional magnetic resonance imaging.* Suderland, MA: Sinauer Associates.

Kida, I., & Hyder, F. (2006). Physiology of functional magnetic resonance imaging: Energetics and function. *Methods in Molecular Medicine, 124*, 175–195.

Logothetis, N., & Pfeuffer, J. (2004). On the nature of the BOLD fMRI contrast mechanism. *Magnetic Resonance Imaging, 22*(10), 1517–1531.

Nair, D. G. (2005). About being BOLD. *Research – Brain Research Reviews, 50*(2), 229–243.

Stephan, K. E., Harrison, L. M., Penny, W. D., & Friston, K. J. (2004). Biophysical models of fMRI responses. *Current Opinion in Neurobiology, 14*(5), 629–635.

Steward, C. A., Marsden, C. A., Prior, M. J., Morris, P. G., & Shah, Y. B. (2005). Methodological considerations in rat brain BOLD contrast pharmacological MRI. *Psychopharmacology, 180*(4), 687–704.

Blood Thinner

► Warfarin (Coumadin)

Blood-Brain Barrier

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Synonyms

Cerebral microvasculature

Definition

The blood-brain barrier (BBB) maintains brain homeostasis by regulating the movement of compounds across the endothelium of cerebral capillaries.

Current Knowledge

BBB serves to maintain brain homeostasis by regulating the influx and efflux of compounds to and from the brain. The presence of a barrier was first documented by Paul Ehrlich in the late nineteenth century. However, it was not until the advent of the electron microscope that the makeup of the BBB was begun to be understood. The brain microvascular endothelium comprises the BBB. In contrast to endothelium from other vascular beds, the morphologic features unique to the brain microvascular endothelium, such as tight junctions, increased electrical resistance, and lack of pinocytic vesicles, provide limited and selective access to this highly specialized organ. Only lipophilic molecules less than 600 Da can passively diffuse through the BBB. This protects the brain from toxins, microorganisms (i.e., bacteria) and peripheral neurotransmitters. This selective barrier can potentially limit entry of large substances required for normal brain function, including insulin, amino acids, and glucose. In order to circumvent this problem, the BBB has developed highly specialized transport mechanisms on both the luminal and albuminal membrane surfaces, such as Na-K-Cl cotransporter, γ-glutamyl transpeptidase (GGTP), and the GLUT-1 glucose transporter. The protective BBB can be at a disadvantage in that it prevents the entry of pharmacologic agents that are often hydrophilic.

Also unique to brain microvascular endothelium is their intimate association with astrocytes, forming the glia limitans. Astrocytes are thought to participate in the induction and maintenance of the endothelial BBB phenotype. In vitro studies have shown that astrocytes cocultured with endothelial cells can induce BBB phenotypic features, including tight junctions and increased electrical resistance. Astrocytic membranes and supernatant from astrocytic cultures share similar inductive properties. The mechanisms of this induction and the inductive factor(s) have yet to be fully elucidated.

In addition to its regulatory role, studies of the BBB are beginning to emerge to demonstrate its function in establishing a unique brain milieu. In vitro BBB models have shown decreased tissue plasminogen activator and then anticoagulant protein thrombomodulin expression and increased plasminogen activator inhibitor-1 expression by brain endothelium compared with endothelium from the periphery. These findings suggest a procoagulant environment in the brain that may predispose the brain to strokes.

Cross References

► Neuroglia

Blood-Filled Dilatation

► Aneurysm

Blue Spot

► Locus Ceruleus

BNT

► Boston Naming Test

Body Schema

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Synonyms

Somatognosis

Definition

General term for the personal awareness of one's body, including the location and orientation of its various parts and their relative motion in space and time, as well as its functional integrity.

Current Knowledge

Although usually taken for granted, to effectively carry out normal motor activities one needs to appreciate both the static and kinetic state of the body as a whole as well as its individual parts. This information is derived from a number of sensory feedback loops, including signals from receptors in the muscles, tendons, ligaments and the skin (proprioceptive, kinesthetic, and tactile information), the inner ear or vestibular sense (orientation, direction, and speed of movement of the head), and vision. Perhaps as a result of collective experiences with such discrete sensory input, it has been suggested that individuals eventually develop what might be considered a superordinate sense of one's own body, independent of its movement in space or time. This knowledge, at least to some extent, transcends one's own body and allows insights into bodies in general. Because awareness of body schema is such a fundamental operation of the central nervous system, it almost functions at a subliminal level. One is normally only aware of its operation when it becomes dysfunctional.

Disorders of body schema, known as *asomatognosias*, can take on various guises. Although relatively rare, *autotopagnosia* represents what might be considered the quintessential body schema disturbance. This deficit involves difficulties in identifying body parts and/or appreciating their relative relations to one another. Care should be taken to differentiate asomatognosia from unilateral neglect or anomia. In the former, the deficit is restricted to one side of the body; in the latter, difficulties with naming extend beyond just parts of the body. More commonly, autotopagnosia is restricted to difficulty identifying individual fingers, especially the middle three. The deficit is usually bilateral and will frequently involve not only difficulties with regard to the patient's own fingers, but also those of the examiner or pictorial representations of a hand. Deficits are often found whether tested visually or tactually and whether verbal or nonverbal (e.g., matching to a model) responses are required. Unilaterally expressed deficits in finger recognition using only tactile stimulation likely reflect a more basic somatosensory disturbance.

Right-left disorientation, the inability to reliably distinguish the right from the left sides of one's body in the absence of a more generalized aphasic disorder, is another commonly cited example of a disturbance of body schema. As with finger agnosia, difficulties extend beyond the patients themselves to include problems with extrapersonal right-left discriminations. *Anosognosia* and *anosodiaphoria* (a milder form of anosognosia), along with *unilateral neglect* or *hemi-inattention* are sometimes viewed as specialized forms of a body schema disorder. One major difference is that these latter syndromes are generally limited to one side of the body, whereas autotopagnosia, the more restricted finger agnosia, and right-left disorientation affect both sides of the body. The one notable exception to this rule is *Anton's syndrome*, a form of anosognosia in which the patient denies blindness where both right and left visual fields are involved. While there is some potential variability with regard to localizations of lesions, asomatognosia, when bilaterally expressed, is most commonly associated with lesions of the left parietal region, typically involving the inferior parietal lobule. Unilateral neglect or hemi-inattention syndromes may occur following anterior or posterior lesions of either hemisphere, although they are most common following right posterior lesions. Anton's syndrome is typically associated with bilateral lesions involving the posterior cerebral arteries.

Cross References

► Allesthesia
► Anosodiaphoria
► Anosognosia

- ▶ Autotopagnosia
- ▶ Cortical Blindness
- ▶ Finger Agnosia
- ▶ Hemiinattention
- ▶ Right-Left Disorientation

References and Readings

Adair, J. C., Schwartz, R. L., & Barrett, A. (2003). Anosognosia. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (4th ed., pp. 185–214). New York: Oxford University Press.

Denburg, N. L., & Tranel, D. (2003). Acalculia and disturbances of body schema. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (4th ed., pp. 161–184). New York: Oxford University Press.

Goldenberg, G. (2003). Disorders of body perception and representation. In T. E. Feinberg & M. J. Farah (Eds.), *Behavioral neurology and neuropsychology* (2nd ed., pp. 285–294). New York, NY: McGraw-Hill.

Prigatano, G. P., & Schacter, D. L. (Eds.). (1991). *Awareness of deficit after brain injury: Clinical and theoretical issues.* New York: Oxford University Press.

BOLD

- ▶ Blood Oxygene Level-Dependent

Bonnet Syndrome

- ▶ Charles Bonnet Syndrome

Bonnevie–Ullrich Syndrome

- ▶ Turner Syndrome

Borderline Schizophrenia

- ▶ Schizotypal Personality Disorder

Borreliosis

- ▶ Lyme Disease

Boston Diagnostic Aphasia Examination

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Synonyms

BDAE

Description

Boston Diagnostic Aphasia Examination (3rd ed.) (BDAE-3) Authors: Harold Goodglass, Edith Kaplan, Barbara Barresi, 2001, Publisher: Pro-Ed, 8700 Shoal Creek Blvd, Austin, TX 78757-6897, http://www.proedinc.com (also available through Psychological Assessment Resources (http://www.parinc.com)).

The BDAE-3 test kit includes test manual, stimulus cards, and test booklets for Standard and Short forms, and the 60-item Boston Naming Test with record booklets. The manual is provided within the test kit.

The Boston Diagnostic Aphasia Examination (BDAE) is a comprehensive, multiple subtests instrument for investigating a broad range of language impairments that are common consequences of brain damage. It is designed as a comprehensive measure of aphasia. The examination provides materials and procedures to evaluate five language-related sections and an additional section on praxis. The five language domains include conversational and expository speech, auditory comprehension, oral expression, reading, and writing. In addition to individual subtest scores, the test yields three broader measures: the Severity Rating Scale (a rating of the severity of observed language/speech disturbance), the Rating Scale Profile of Speech Characteristics (a rating of observed speech characteristics and of scores in two main language domains), and the Language Competency Index (a composite score of language performance on BDAE-3 subtests). The extended version includes a sixth section, "Praxis," which examines natural and conventional gestures, use of pretend objects, and bucco-facial and respiratory movements. Suggestions for administering, scoring, and interpreting performance on subtests in the BDAE are given in the test manual, as well as directions for plotting and interpreting patient profiles. Percentiles or standard scores can be derived for each subtest.

Administration

The 44-page test booklet provides instructions for test administration. This booklet is available at the back of the hardbound text by Goodglass, Kaplan, and Barresi (2001a) published by Lippincott Williams & Wilkins, which is included in the complete test package. The Short Form and Extended Form items are specified in the test booklet and are also presented in different typeface; the Short Form items are presented in bold typeface, and the Extended Form items appear in italics. The Standard administration includes all of the bold Short Form items in addition to regular typeface items.

Historical Background

The BDAE is designed to meet three goals: to enable diagnosis of aphasia syndromes, to measure the breadth and severity of aphasic disturbance, and to provide a comprehensive assessment of language to guide therapy. Initially published by Goodglass and Kaplan in 1972, it was revised in 1983 and again in 2001. Changes from the previous edition include the addition of abbreviated and expanded testing formats, incorporation of the Boston Naming Test, addition of a Language Competence Index and clarification of scoring procedures and definitions. The revision also was designed to integrate recent advances in neurolinguistics research, including methods to assess narrative and discourse complexity, category-specific dissociations in lexical production/comprehension, syntax comprehension, and analysis of grapheme-phoneme conversion during reading. The ultimate goal for the authors in developing the test was clinical utility.

The BDAE-3 consists of more than 50 subtests that can be administered in three different formats: Standard, Short, and Extended. The Standard format most closely resembles earlier versions of the BDAE. The new Short Form of the test provides a brief assessment. The extended version offers a comprehensive neurolinguistic profile that includes evaluation of spontaneous narrative, processing of word categories, syntax comprehension, and reading/writing. The BDAE-3 allows both a quantitative and a qualitative evaluation of language. The examination is based on an assumption that the nature of the aphasic deficit is determined by (1) organization of language in the brain, (2) the location of the lesion causing the aphasia, and (3) interactions among parts of the language system.

The BDAE has been adapted and translated for use in many languages including Spanish, French, German, Italian, Dutch, Hindi, Finnish, Mandarin Chinese, Japanese, and Portuguese.

Psychometric Data

Norms

Standardization of the BDAE-3 is based on an aphasic population referred concurrently by field examiners working in inpatient, outpatient, and private practice settings. Means and standard deviations for the BDAE-3 subtests for aphasic subjects are provided in the test manual. The number of subjects varies from a maximum of 85 to a low of 31, depending on the subtest. Means are also provided for 15 normal subjects who, on average, failed less than one item per subtest.

Reliability

Kuder–Richardson reliability coefficients for subtests reflect variability, ranging between <0.65 and < 0.95 with about two thirds of the coefficients reported in the manual (Goodglass et al., 2001a), ranging from 0.90 upwards. Since test–retest reliability is difficult if not impossible to attain with patients with aphasia, the current reliability coefficients demonstrate very good internal consistency in terms of what the items within the subtests are measuring (Goodglass et al., 2001a). For most subtests, correlations are very high between the Short and Standard Forms (>0.90; Goodglass et al., 2001a).

Validity: A correlation matrix was obtained for all the scores in the battery and the correlation coefficients 0.60 or greater are displayed in the manual (Goodglass et al., 2001a), with severity partialled out, showing intercorrelations between BDAE-3 subtests for the standardization sample. Based on these, "a number of sharply defined clusters" are indicated by the authors (p. 16). Although no current studies have examined the factor structure of the BDAE-3, based on data for earlier versions of the BDAE, Goodglass and Kaplan (1972) found a strong general language factor, and factors covering spatial-quantitative-somatagnostic, articulation-grammatical fluency, auditory comprehension, and paraphasia domains. Goodglass and Kaplan (1983) described a second factor analysis using a sample of 242 aphasic patients, concluding that auditory comprehension, repetition-recitation, reading, and writing were factors of equal importance. Similar findings in normal individuals were reported by Pineda et al. (2000).

Correlations between earlier versions of the BDAE and other measures have been described. For example, the

BDAE oral apraxia task has been correlated with other articulation tasks (Sussman et al., 1986); correlations for the auditory comprehension measure on the BDAE and the Token Test and with respective measures of the Porch Index of Communicative Ability (PICA) have been reported (Divenyl & Robinson, 1989). Brookshire and Nichols (1984) found the BDAE auditory comprehension subtest did not predict auditory paragraph comprehension of independent standardized material.

Goodglass and Kaplan designed the BDAE to assess various components of language function for the purpose of discriminating among different patterns of CNS lesions indicative of types of aphasia. Studies to date have not determined decision rules for the diagnosis of individual subtypes of aphasia (Crary et al., 1992; Reinvang & Graves, 1975).

Ecological validity of the BDAE for predicting progress in speech therapy has been described by various authors (Davidoff & Katz, 1985; Helm-Estbrooks & Ramsberger, 1986; Marshall & Neuberger, 1994).

Clinical Uses

The BDAE is derived from samples of adult patients with stroke and therefore is most useful when assessing inpatient or outpatient adult populations with language impairments resulting from strokes, but it can be used effectively with persons who have sustained traumatic brain injury. The BDAE offers a comprehensive look at language function from a neuropsychological perspective. Complete administration of this battery requires approximately 90 min. The Short Form requires approximately 40–60 min. The BDAE is one of the most popular batteries for use by speech–language pathologists for evaluation of aphasia and other neurologic language impairments. In addition to its strength as a comprehensive assessment of language, the BDAE provides useful instructions for observing and recording specific types of error responses (e.g., ▶ paraphasia) found in individuals with aphasia, reflecting what has been termed the "Boston school" approach to aphasia classification. The detailed examination of conversational and expository speech is an important and unique aspect of the BDAE and is well described in the manual (Goodglass et al, 2001a).

Cross References

- ▶ Anomic Aphasia
- ▶ Aphasia
- ▶ Boston Naming Test
- ▶ Broca's Aphasia
- ▶ Conduction Aphasia
- ▶ Praxis
- ▶ Wernicke's Aphasia

References and Readings

Brookshire, R. H., & Nicholas, L. E. (1984). Comprehension of directly and indirectly stated main ideas and details in discourse by brain-damaged and non-brain-damaged listeners. *Brain and Language, 21*, 21–36.

Crary, M. A., Wertz, R. T., & Deal, J. L. (1992). Classifying aphasias: Cluster analysis of Western Aphasia Battery and Boston Diagnostic Aphasia Examination. *Aphasiology, 6*, 29–36.

Davidoff, M., & Katz, R. (1985). Automated telephone therapy for improving comprehension in aphasic adults. *Cognitive Rehabilitation, 3*, 26–28.

Divenyl, P. L., & Robinson, A. J. (1989). Nonlinguistic auditory capabilities in aphasia. *Brain and Language, 37*, 290–326.

Goodglass, H., & Kaplan, E. (1972). *Boston Diagnostic Aphasia Examination (BDAE)*. Philadelphia: Lea & Febiger.

Goodglass, H., & Kaplan, E. (1983). *The assessment of aphasia and related disorders* (2nd ed.). Philadelphia: Liea & Febiger.

Goodglass, H., Kaplan, E., & Barresi, B. (2001a). *The assessment of aphasia and related disorders* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.

Goodglass, H., Kaplan, E., & Barresi, B. (2001b). *Boston diagnostic aphasia examination* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.

Helm-Estabrooks, N., & Ramsberger, G. (1986). Aphasia treatment delivered by telephone. *Archives of Physical Medicine and Rehabilitation, 67*, 51–53.

Marshall, R. C., & Neuburger, S. I. (1994). Verbal self-correction and improvement in treated aphasia clients. *Aphasiology, 8*, 535–547.

Pineda, D. A., Rosselli, M., Ardila, A., Mejia, S. E., Romero, M. G., & Perez, C. (2000). The Boston Diagnostic Aphasia Examination-Spanish Version: The influence of demographic variables. *Journal of the International Neuropsychological Society, 6*, 802–814.

Reinvang, I., & Graves, R. (1975). A basic aphasia examination: Description with discussion of first results. *Scandinavian Journal of Rehabilitation Medicine, 7*, 129–135.

Spreen, O., & Risser, A. H. (Eds.). (2003). *Assessment of aphasia*. New York: Oxford University Press.

Sussman, H., Marquardt, T., Hutchinson, J., & MacNeilage, P. (1986). Compensatory articulation in Broca's aphasia. *Brain and Language, 27*, 56–74.

Boston Naming Test

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Synonyms

BNT

Description

Boston Naming Test-2 (BNT-2)
Authors: Kaplan, Edith, Goodglass, Harold, Weintraub, Sandra
Second edition 2001
Publisher: Pro-Ed, 8700 Shoal Creek Blvd, Austin, TX 78757–6897
http://www.proedinc.com
Also available as part of the revised BDAE-2 (Goodglass et al., 2000) from Pro-Ed.

The Boston Naming Test (BNT), consisting of 60 black and white line drawings of objects, is a measure of confrontation naming that takes into account the finding that patients with dysnomia often have greater difficulties with the naming of low frequency objects. Thus, instead of a simple category of anomia, naming difficulties may be rank ordered along a continuum. Items on the BNT are ordered according to their ability to be named, which is thought to be correlated with their frequency. This type of picture-naming vocabulary test is useful in the examination of children with learning disabilities and the evaluation of adults with brain injury or dysfunction. When used in conjunction with the Boston Diagnostic Aphasia Examination, inferences can be drawn regarding language facility and possible localization of cerebral damage.

Administration

The Boston Naming Test assesses naming abilities of children, aphasic adults, and normal adults. The drawings are shown to the examinee one at a time, and the examinee is asked to name each of them. Familiarity decreases as the test progresses. Following presentation of each picture stimulus, two types of cues may be presented when there is an error response: a "stimulus cue" (descriptive) and a "phonemic cue." A stimulus cue is presented when the subject clearly misperceives the picture or indicates a lack of recognition of the picture. A phonemic cue is presented after each error response, including following a stimulus cue. The subject is given up to 20 s to respond following each stimulus presentation, including the cues. All responses are recorded as a "correct response" or as an error with the actual error response recorded for later coding by type. Types of cues presented are recorded as "stimulus cue" or "phonemic cue"; response latencies in seconds are also recorded. The total correct score is the sum of the accurate responses presented spontaneously and following a stimulus cue. Correct responses following a phonemic cue are not included in the total correct score.

Historical Background

The test was originally published by Kaplan and colleagues in 1978 as an experimental version with 85 items. It was revised to a 60-item test in 1983. The current version (BNT-2) retains the same 60 items and includes a short 15-item version as well as a multiple-choice version. Short-forms of the BNT have been developed to reduce test time. These include: Fastenau et al. (1998), Graves et al. (2004), Lansing et al. (1999), Mack et al. (1992), Saxton et al. (2000), Teng et al. (1989), and Williams et al. (1989). The 15-item short-form 4 (Mack SF4) developed by Mack et al. (1992) was adopted by the authors of the BNT-2 and can be found at the beginning of the stimulus booklet and answer sheet. The new BNT-2 also includes a multiple-choice version that can be administered following the standard presentation, specifically to further assess the subject's recognition of the lexicon for items previously missed. The BNT-2 is available separately and as part of the revised BDAE-2.The BNT has been adapted and translated for use in at least a dozen languages including a 30-item adaptation for Spanish-speaking people in the United States.

Psychometric Data

Reliability, Validity, and Norms:

Reliability

Internal consistency for the 60-item form has been reported to range between 0.78 and 0.96. Reliability coefficients have been lower for the abbreviated versions; for example, the Mack SF4 version ranges between 0.49 and 0.84. Test–retest reliability is high over short intervals. For longer time intervals, such as 11–12 months, test–retest reliability was marginal to high; for example, in a healthy, elderly Caucasian adult population, test–retest reliability ranged between 0.62 and 0.89 (Mitrushina & Satz, 1995); and high retest reliability (0.92) in a normal or neurologically stable adult population (Dikmen et al., 1999).

Validity

The BNT has been shown to correlate highly with other language-related measures; including the Visual Naming

Test of the Multilingual Aphasia Examination (Axelrod et al., 1994; Schefft et al., 2003); as well as with measures of intelligence; including the Verbal Comprehension Factor of the WAIS-R and the Standard Raven Progressive Matrices in children aged 6–12 years (Storms et al., 2004).

Poor performance on the BNT has been described in subjects with neurologic disease; including: left-hemisphere and brainstem strokes, anoxia, multiple sclerosis, Parkinson's disease, Alzheimer's disease, and closed head injuries.

Norms

The norms available in the test booklet are limited to small groups of adults ranging in age between 18 and 79 (N = 178) and of children ranging in age between 5–0 years and 12–5 years (N = 356). Information about geographical region, ethnicity, or time reference for this normative data is not provided.

Cross-sectional studies suggest that age (Heaton et al., 2004; Ivnik et al., 1996; MacKay et al., 2005; Mitrushina et al., 2005) and verbal intelligence affect the BNT scores (Killgore & Adams, 1999; Steinberg et al., 2005; Tombaugh & Hubley, 1997). Gender has been reported to be unrelated to BNT performance (Henderson et al., 1998; Ivnik et al., 1996; Lucas et al. 2005; Riva et al., 2000). Other studies suggest men outperform women in older samples, possibly because of male-biased items (Randolph et al., 1999). Reading vocabulary is strongly correlated with BNT performance (Graves & Carswell, 2003; Senior et al., 2001). Geographic region and ethnicity have been shown to affect performance (Heaton et al., 2004; Lucas et al., 2005). Linguistic background also affects test scores according to Roberts et al. (2002).

In the literature can be found a number of normative reports for adult English speakers (see pp. 905–907, Strauss, Sherman, & Spreen, 2006). For example, Heaton et al. (2004) reviewed studies over a 25-year period and presented age, gender, and educational norms for two ethnicity groups: Caucasians and African Americans. Mitrushina et al. (2005) compiled data from 14 studies, comprising a total of 1,684 educated participants with above-average intelligence who were administered the 60-item version. Their data was presented in 5-year increments, ranging from ages 25 to 84 years. The data is considered to be similar to those provided by Kaplan et al. (2001) and may overestimate expected performance for individuals with lower educational and intellectual levels. Ivnik et al. (1996) provided age-corrected norms for 663 primarily Caucasian individuals older than 55 years of age, derived from the Mayo Older Americans Normative Studies (the MOANS projects). Raw scores are converted to age-corrected scaled scores having a mean of 10 and a standard deviation (SD) of 3 (Strauss et al., 2006). Additional studies have expanded the utility of the MOANS project by providing age- and IQ-adjusted percentile equivalents of MOANS Age-adjusted BNT scores, for individuals over 55 years (Steinberg et al., 2005); age- and education-adjusted normative data based on African Americans from the Mayo Older African American Normative studies (MOAANS) project (Lucas et al., 2005) (Strauss et al., 2006).

Data on BNT norms for children is limited. The test authors provide norms for ages 5 years 0 months (5–0) through 12–5, based on small groups of participants. The data were collected in 1987 and the normative data are believed to be largely from Caucasian boys and girls who were attending public and private schools and living with middle class families in suburban or urban areas of the northeastern United States. There is a gap in the BNT norms for adolescents between the ages of 14 and 17 years.

Clinical Uses

The BNT, a visual confrontation naming test, is recommended as a supplement to the Boston Diagnostic Aphasia Examination. It can be used to assess naming abilities of children, aphasic adults, and typical adults, although there is limited and poorly described normative data, and no test–retest reliability for children.

Cross References

► Anomia
► Boston Diagnostic Aphasia Examination

References and Readings

Axelrod, B. N., Ricker, J. H., & Cherry, S. A. (1994). Concurrent validity of the MAE visual naming test. *Archives of Clinical Neuropsychology, 9*, 317–321.

Dikmen, S. S., Heaton, R. K., Grant, I., & Temkin, N. R. (1999). Test–retest reliability and practice effects of expanded Halstead–Reitan

neuropsychological test battery. *Journal of the International Neuropsychological Society, 5*, 346–356.
Fastenau, P. S., Denburg, N. L., & Mauer, B. A. (1998). Parallel short forms for the Boston Naming Test: Psychometric properties and norms for older adults. *Journal of Clinical and Experimental Neuropsychology, 20*, 828–834.
Goodglass, H., Kaplan, E., & Barresi, B. (2000). The assessment of aphasia and related disorders (3rd ed.). Philadelphia: Lea & Febiger.
Graves, R. E., Bezeau, S. C., Fogarty, J., & Blair, R. (2004). Boston Naming Test Short Forms: A comparison of previous forms with new item response theory based forms. *Journal of Clinical and Experimental Neuropsychology, 26*, 891–902.
Heaton, R. K., Miller, S. W., Taylor, M. J., & Grant, I. (2004). *Revised comprehensive norms for an expanded Halstead Reitan Battery: Demographically adjusted neuropsychological norms for African American and Caucasian adults,* Lutz, FL: PAR.
Henderson, L. W., Frank, E. W., Pigatt, T., Abramson, R. K., & Houston, M. (1998). Race, gender and educational level effects on Boston Naming Test scores. *Aphasiology, 12*, 901–911.
Ivnik, R. J., Malec, J. F., Smith, G. E., Tangalos, E. G., & Peterson, R. C. (1996). Neuropsychological test norms above age 55: COWAT, BNT, MAE Token, WRAT-R Reading, AMNART, Stroop, TMT, and JLO. *The Clinical neuropsychologist, 10*, 262–278.
Kaplan, E., Goodglass, H., & Weintrab, S. (1983). The Boston naming test. Philadelphia: Lea & Febiger.
Killgore, W. D. S., & Adams, R. L. (1999). Prediction of Boston Naming Test performance form vocabulary scores: Preliminary guidelines for interpretation. *Perceptual and Motor Skills, 89*, 327–337.
Lansing, A. E., Ivnik, R. J., Cullum, C. M., & Randolph, C. (1999). An empirically derived short form of the Boston Naming Test. *Archives of Clinical Neuropsychology, 14*, 481–487.
Lucas, J. A., Ivnik, R. J., Smith, G. E., Ferman, T. J., Willis, F. B., Petersen, R. C., 7 Graff-Radford, N. R. (2005). Mayo's Older African Americans Normative Studies: Norms for Boston Naming Test, Controlled Oral Word Association, Category Fluency, Animal naming, Token Test, WRAT-3 Reading, Trail Making Test, Stroop Test, and Judgment of line Orientation. *The Clinical Neuropsychologist, 19*, 243–269.
Mack, W. J., Freed, D. M., Williams, B. W., & Henderson, V. W. (1992). Boston Naming Test: Shortened version for use in Alzheimer's disease. *Journal of Gerontology, 47*, 164–168.
MacKay, A., Connor, L. T., & Henderson, V. W. (2005). Dementia does not explain correlation between age and scores on Boston Naming Test. *Archives of Clinical Neuropsychology, 20*, 129–133.
Mitrushina, M., & Satz, P. (1995). Repeated testing of normal elderly with the Boston Naming Test. *Aging Clinical and Experimental Research, 7*, 123–127.
Mitrushina, M. M., Boone, K. B., Razani, J., & D'Elia, L. F. (2005). *Handbook of normative data for neuropsychological assessment* (2nd ed.). New York: Oxford University Press.
Neils, J., Baris, J. M., Carter, C., Dell'aira, A. L., Nordloh, S. H., & Weiler, E. (1995). Effects of age, education, and living environment on Boston naming test performance. *Journal of Speech and Hearing Research, 38*, 1143–1149.
Nicholas, L. E., Brookshire, R. H., MacLennan, D. L., Schumacher, J. G., & Porrazzo, S. A. (1988). The Boston naming test: Revised administration and scoring procedures and normative information for non-brain-damaged adults. *Clinical Aphasiology, 18*, 103–115.
Randolph, C., Lansing, A., Ivnick, R. J., Cullum, C. M., & Hermann, B. P. (1999). Determinants of confrontation naming performance. *Archives of Clinical Neuropsychology, 14*, 489–496.
Roberts, P. M., Garcia, L. J., Desrochers, A., & Hernandez, D. (2002). English performance of Proficient bilingual adults on the Boston Naming Test. *Aphasiology, 16*, 635–645.
Saxton, J., Ratcliff, G., Munro, C. A., Coffery, C. E., Becker, J. E., Fried, L., & Kuller, L. (2000). Normative data on the Boston Naming Test and two equivalent 30-item short-forms. *The Clinical Neuropsychologist, 14*, 526–534.
Schefft, B. K., Testa, S. M., Dualy, M. F., Privitera, M. D., & Yeh, H. S. (2003). Preoperative assessment of confrontation naming ability and intrictal paraphasia production in unilateral temporal lobe epilepsy. *Epilepsy and Behavior, 4*, 161–168.
Steinberg, B. A., Beiliauskas, L. A., Smith, G. E., Langellotti, C., & Ivnik, R. J. (2005). MAYO's Older Americans Normative Studies: Age- and IQ-adjusted norms for the Boston Naming Test, the MAE Token Test, and the Judgment of Line Orientation Test. *The Clinical Neuropsychologist, 19*, 280–328.
Storms, G., Saerens, J., & De Deyn, P. P. (2004). Normative data for the Boston Naming Test in native Dutch-speaking Belgian children and the relation with intelligence. *Brain and Language, 91*, 274–281.
Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsyschological tests: Administration, norms, and commentary* (3rd ed. pp. 901–915). New York: Oxford University Press.
Teng, E. L., Wimer, C., Roberts, E., Damasio, A. R., Eslinger, P. J., Folstein, M. F., Tune, L. E., Whitehouse, P. J., Bardolph, E. L., Hui, H. C., & Henderson, V. W. (1989). Alzheimer's dementia: Performance on parallel forms of the Dementia Assessment Battery. *Journal of Clinical and Experimental Neuropsychology, 11*, 899–912.
Tombaugh, T. N., & Hubley, A. (1997). The 60-item Boston Naming Test: Norms for cognitively intact adults aged 25 to 88 years. *Journal of Clinical and Experimental Neuropsychology, 19*, 922–932.
Van Gorp, W., Satz, P., Kiersch, M., & Henry, R. (1986). Normative data on the Boston naming test for a group of normal older adults. *Journal of Clinical and Experimental Neuropsychology, 8*, 702–705.
Welch, L. W., Doineau, D., Johnson, S., & King, D. (2002). Educational and gender normative data for the Boston naming test in a group of older adults. *Brain and Language, 53*(2), 260–266.
Williams, B. W., Mack, W., & Henderson, V. W. (1989). Boston naming test in Alzheimer's disease. *Neuropsychologiea, 27*, 1073–1079.

Boston Process Approach

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Synonyms

BPA

Description

Born out of the work of A. R. Luria (e.g., Higher Cortical Function in Man, 1966), the Boston Process Approach

(BPA) to neuropsychological assessment is a method of exploring the patient's approach to a task and the process involved in attaining a specific test score (Loring, 1999). Its aim is to provide a more accurate characterization of neuropsychological function and dysfunction and the nervous system components involved than is achieved with a purely psychometric approach (Kaplan, 1988; Strauss, Sherman, & Spreen, 2006).

Current Knowledge

According to Edith Kaplan (1988, 1990), the "achievement" oriented approach to assessment is flawed, in that it assumes that the scores obtained are reflective of an underlying unitary mechanism. As an example, two individuals could arrive at a similar score via distinctly different processes that are dependent on distinctly different neural structures and/or pathways. The inherent loss of data that occurs by focusing on composite or total scores resulted in the development of an approach that focused on how a specific result was obtained. This avenue of investigation led to the Boston Process Approach (BPA). In addition to careful observation of the strategies used during the completion of a task, the BPA emphasizes the importance of demographic variables (e.g., age, gender, socioeconomic status, education, and occupational status), medical history, and mental health history, because each of these variables can influence a patient's performance.

The BPA differs from the fixed and flexible battery approaches to testing in that the final score is deemphasized; that is, whether a response is right or wrong is less important than how it was attained (Kaplan, 1990). In addition, the test may be administered differently from the standardized approach, and additional measures may be introduced to better understand the component processes that influence or are involved in the performance of a particular task. Modified materials may also be used to gain a better understanding of the errors or unusual approaches that were noted on a task (Milberg, Hebben, Kaplan, 1986).

Although right or wrong answers are deemphasized in the BPA, it is essential for the qualitative observations to be quantifiable and subjected to statistical analyses (Kaplan, 1990). This approach contributed to the development of a wide variety of measures including the Wechsler Intelligence Scale for Children, 4th Edition Integrated (Wechsler, Kaplan, Kramer, Morris, 2004) and California Verbal Learning Test, 2nd Edition (Delis, Kramer, Kaplan, & Ober, 2000). Each of these measures provides a number of standard scores, enabling comparisons with a normative sample. In addition, they provide the clinician with a way of understanding the patient's approach to the task. For example, on the CVLT-II, whether the examinee makes use of semantic categories (e.g., types of animals) to aid in recall of the word list is considered and quantified.

Despite the growing popularity of the BPA, there have been criticisms. As outlined by Strauss et al. (2006), criticisms of the approach include insufficient norms, limited information about reliability and validity, and problems with readministration due to nonstandard initial administration.

Cross References

► Hypothesis Testing Approach

References and Readings

Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000). *California verbal learning test. Adult version manual* (2nd ed.). San Antonio, TX: The Psychological Corporation.

Kaplan, E. (1988). A process approach to neuropsychological assessment. In T. Boll, & B. K. Bryant (Eds.), *Clinical neuropsychology and brain function: Research, measurement, and practice.* Washington, DC: American Psychological Association.

Kaplan, E. (1990). The process approach to neuropsychological assessment of psychiatric patients. *Journal of Neuropsychiatry, 2*, 72–87.

Loring, D. W. (1999). *INS dictionary of neuropsychology.* New York: Oxford University Press.

Luria, A. R. (1966). *Higher cortical function in man.* New York: Basic Books.

Milberg, W. P., Hebben, N., & Kaplan, E. (1986). The Boston process approach. In I. Grant, & K. M. Adams (Eds.), *Neuropsychological assessment of neuropsychiatric disorders.* New York: Oxford University Press.

Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). New York: Oxford University Press.

Wechsler, D., Kaplan, E., Fein, D., Kramer, J., Morris, R., et al. (2004). *Wechsler intelligence scale for children integrated* (4th ed.). San Antonio, TX: Harcourt Assessment.

BPA

► Boston Process Approach

BPRS

► Brief Psychiatric Rating Scale

Brachytherapy

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Synonyms

Internal radiation therapy

Definition

Brachytherapy is a form of radiation therapy, and often indicated for the treatment of specific, recurrent brain tumors and head or neck cancers. The procedure involves the placement of radioactive (e.g., iridium-192, palladium-103, or iodine-125) seeds inside or adjacent to a targeted lesion. The primary advantage of brachytherapy is that the treatment allows for a higher radioactive dose to be delivered to the tumor bed without damaging the surrounding, healthy brain tissue (Sneed, Prados, Phillips, Weaver, & Wara, 1992). In particular, High Dose Rate brachytherapy utilizes catheters to mitigate exposure and accelerate the treatment time. Intracavitary brachytherapy is another subtype that involves the use of a balloon catheter which delivers localized radiation therapy to the affected area. Following the completion of radiotherapy, the radiation source and balloon catheter are then removed. Brachytherapy is a safe procedure, although reported side effects include infection, seizures, and headaches.

Cross References

► Radiation Therapy

References and Readings

Sneed, P. K., Gutin, P. H., Prados, M. D., Phillips, T. L., Weaver, K. A., Wara, W. M., et al. (1992). Brachytherapy of brain tumors. *Stereotactic and Functional Neurosurgery, 59*, 157–165.

Bradykinesia

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Definition

Bradykinesia is a slowness of movement. It is often seen in parkinsonian individuals and is a cardinal feature of Parkinson's disease. It can be seen in movements of small muscles when an individual is asked to rapidly open and close a hand, tap a finger, or move an arm back and forth to grab an object. It can involve any limb in isolation, such as decreased arm swing during gait evaluation, or the entire body at once, evident in the abnormal stillness of a patient with Parkinson's disease. It may fluctuate during the day depending on fatigue and medication levels in the case of Parkinson's disease.

Cross References

► Parkinson's Disease

References and Readings

Fahn, S., & Jankovic, J. (Eds.). (2007). Parkinsonism: Clinical features and diagnosis. In *Movement disorders* (pp. 79–100). Philadelphia: Churchill Livingstone Elsevier.

Brain Abscesses

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Definition

Brain abscesses are an intracranial mass of immune cells, pus (i.e., collection of dead neutrophils), and other materials stemming from a bacterial or fungal infection.

Current Knowledge

Etiology

Brain abscesses may arise by direct infection of organisms, local extension from adjacent focal areas, or distribution by way of the bloodstream. Moreover, they form as an inflammatory response to bacteria or fungal infections within the brain. This inflammatory response leads to a localization of infected brain cells, immune cells, and microorganisms within an area of the brain (Kumar, Abbas, & Fausto, 2004). This area becomes encapsulated by an abscess wall, which is formed by adjacent cells to prevent further infection of neighboring structures. This results in the formation of an encapsulated, purulent (pus-filled) mass within the brain. While this inflammatory response can serve to protect the brain from further injury, it can also have significant negative consequences. If the abscess ruptures, it can lead to inflammation of the ventricles (i.e., fluid-filled cavities containing cerebral spinal fluid) within the brain in addition to inflammation of the meninges (i.e., membranes that surround the brain and spinal cord). If the brain begins to swell, the mass may raise intracranial pressure and promote progressive herniation within the brain, which can be fatal (Kumar et al., 2004).

Symptoms

Clinically, cerebral abscesses can be devastating and often lead to an increase in intracranial pressure and localized deficits (Kumar et al., 2004). Additionally, symptoms associated with brain abscesses can develop slowly (i.e., within a 2-week period) or suddenly. A nonexhaustive list of symptoms may include the following: headaches, gait disturbances, disequilibrium, changes in mental status, vomiting, and stiffness/aching of the neck, shoulders, or back.

Prognosis

If brain abscesses are left untreated, death is the most likely outcome. On the other hand, treatment can significantly reduce the mortality rate to about 10 percent. Earlier treatment predicts a better outcome, although long-term neurological deficits may persist despite all intervention approaches (Kumar et al., 2004 and http://www.nlm.nih.gov/medlineplus/ency/article/000783.htm).

Treatment

Brain abscesses are treated as medical emergencies and may require hospitalization. If the infectious agent is bacterial in nature, antibiotics are usually the treatment of choice. However, if the infection is determined to be of fungal origin, then antifungal medications may be prescribed. Surgery is usually indicated if intracranial pressure continues to increase, medications fail to reduce the size of the abscesses, or the abscesses are at risk of rupture (www.nlm.nih.gov/medlineplus/ency/article/000783.htm).

Cross References

- ▶ Brain Swelling
- ▶ Cyst
- ▶ Inflammation

References and Readings

Kumar, V., Abbas, A., & Fausto, N. (2004). *Robbins and Cotran pathalogic basis of disease* (7th ed.). Philadelphia, PA: W.B. Saunders Company.

See also http://www.nlm.nih.gov/medlineplus/ency/article/000783.htm.

Brain Arteriovenous Malformation

- ▶ Arteriovenous Malformation (AVM)

Brain Attack

- ▶ Stroke

Brain Commissures

- ▶ Commissures, Cerebral

Brain Death

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Synonyms

Death

Definition

Brain death is the irreversible loss of all brain function. Including the lack of capacity for consciousness and respirations (Presidents Commission for the Study of Ethical Problems in Medicine, 1981). Brain death is equivalent to traditional circulatory death, which is defined by cessation of tissue perfusion and the absence of pulses. However, with brain death the heart will continue to beat and spinal cord reflexes may persist for a short time (Canadian Neurocritical Care Group, 1999).

Current Knowledge

History of the Definition of Brain Death

In 1959, Mollaret and Goulon first introduced the term *coma dépassé* (beyond coma) to describe irreversible brain damage (Mollaret & Goulon, 1959). The modern scientific concept of brain death is largely based on this original description of 23 comatose patients who exhibited loss of brainstem reflexes, respirations, and flat electroencephalograms (EEG). Several years later, the Harvard ad hoc committee formalized the definition of brain death using neurological criteria and published their landmark article in 1968. These publications helped to define current practice guidelines, now widely accepted by clinicians, involved in the diagnosis of brain death.

Criteria for the Diagnosis of Brain Death in Adults

The determination of brain death is largely a clinical diagnosis. Any experienced physician should be able to make the diagnosis; however, in some states, a specialist in the field of neuroscience is required to make the assessment. Certain criteria should be met before a diagnosis of brain death is considered in order to determine the presence of unequivocal neurologic devastation. These include interpreting relevant imaging studies and excluding the presence of conscious altering drugs. (Table 1, Wijdicks, 2000).

When the assessment for neurologic devastation is complete, a focused and methodical clinical examination should follow with emphasis on the documentation of coma, absence of brainstem reflexes, and demonstration of apnea following maximal stimulation of respiratory centers (Table 2).

In some instances, the clinical determination of brain death is not possible because of a patients' extreme hemodynamic or respiratory instability. In these cases, certain confirmatory testing can be completed to make the diagnosis. These often include cerebral angiography, transcranial doppler, electroencephalography (EEG), or

Brain Death. Table 1 Assessment of Neurologic Devastation

Clinical or radiographic evidence of catastrophic and irreversible brain injury
Exclusion of drug intoxication, sedatives, or paralytic agents
Correction of severe electrolyte, acid-base, or endocrine disturbances
Core body temperature >32°C

Brain Death. Table 2 Clinical criteria for brain death

1. Coma, profound state of unconsciousness
2. Pupils fixed at midposition and dilated
3. Absence of papillary response to light
4. Absence of pupil movement with head manipulation or injection of cold water into the EAC (external auditory canal)
5. Absence of motor response
6. Absence of corneal and gag reflexes
7. Absence of coughing in response to tracheal suctioning
8. Absence of respiratory drive at $Paco_2$ 60mmHg or 20mmHg above patients baseline[a]

[a]$Paco_2$ is the partial pressure of arterial carbon dioxide
Reprinted from Wijdicks (2001) with permission

nuclear imaging. These tests are not required for the standard diagnosis of adult brain death.

References and Readings

Ad Hoc Committee of the Harvard Medical School. (1968). A definition of irreversible coma. Report of the Ad Hoc Committee of the Harvard medical school to examine the definition of brain death. *Journal of the American Medical Association, 205*, 337–340.

Canadian Neurocritical Care Group. (1999). Guidelines for the diagnosis of death. *Canadian Journal of Neurological Sciences, 26*, 64–66.

Mollaret, P., Goulon, M. (1959). Le coma dépassé. *Revue Neurologique, 101*, 3–15.

President's Commission for the Study of Ethical Problems in Medicine. (1981). Guidelines for the determination of death. *Journal of the American Medical Association, 246*, 2184–2186.

Wijdicks, E. F. (2000). *Brain death.* Philadelphia: Lippincott Williams & Wilkins.

Wijdicks, E. F. (2001). The Diagnosis of Brain Death. *New England Journal of Medicine, 344*, 1215–1221.

Brain Disease

► Encephalopathy

Brain Hemorrhage

► Hemorrhagic Stroke

Brain Imaging

► Neuroimaging

Brain Injury

► Head Injury
► Penetrating TBI
► Traumatic Brain Injury (TBI)

Brain Injury Association of America

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Synonyms

BIAA

Membership

The Brain Injury Association of America (BIAA) consists of more than 40 state affiliates as well as hundreds of local chapters and support groups. A portion of the individuals involved at these various levels subscribe to the national mailing list which includes the names of approximately 19,000 individuals. Approximately two-thirds of the list are traumatic brain injury (TBI) survivors and their family members while the remaining represent a wide variety of professional providers and researchers (G. Ayotte, personal communication, November, 2008).

Major Areas or Mission Statement

BIAA Mission Statement: "Creating a better future through brain injury prevention, research, education, and advocacy."

Landmark Contributions

The BIAA, formerly the National Head Injury Foundation, was founded in 1980 by Marilyn and Marty Spivack and other family members of brain injury survivors. Among BIAA's landmark contributions was its success in securing Congressional approval of the 1996 Traumatic Brain Injury Act (PL 104–166), later re-authorized as Title XIII of the Children's Health Act of 2000 (PL 106–310), and most recently as the S. 793 TBI Act of 2008. The original bill created the Federal TBI program to address the struggles of many persons with TBI in gaining access

to appropriate community-based care. It is the only Federal law that addresses the millions of Americans who suffer permanent disability as a result of traumatic brain injury. The law allowed the Health Resources and Services Administration (HRSA) to develop a grant program for states to aid in the treatment of persons with TBI. The bill authorized appropriations to the National Institutes of Health (NIH) for TBI research and to the Centers for Disease Control and Prevention (CDC) for a public information campaign regarding the consequences and prevention of brain injury. The ability to achieve successive appropriation bills has been due in part to the work of the BIAA and others to persuade approximately 100 members of Congress to join the Congressional Brain Injury Task Force. The task force was established in 2001 to provide guidance to Congress and other federal agencies regarding policies relevant to the care and treatment of those with traumatic brain injury.

In 1992, the BIAA was integral in shaping the Defense and Veterans Head Injury Program, later renamed the Defense and Veterans Brain Injury Center (DVBIC). This organization's mission is to develop and provide advanced TBI evaluation and treatment for veterans as well as active duty military personnel and their dependents. The program has lead to the development of six additional Department of Defense and Veterans Affairs treatment sites that offer specialized care for military survivors of brain injury. The DVBIC includes a number of collaborating partners to provide medical care, clinical research, and brain-injury education. It was named the TBI operational component of the recently established Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury.

In 1999, the US Supreme Court decided the case of *L.C. & E.W. vs. Olmstead*. Justice Ruth Bader Ginsburg delivered the opinion of the Court, holding that under Title II of the Americans with Disabilities Act "states are required to place persons with mental disabilities in community settings rather than in institutions when the State's treatment professionals have determined that community placement is appropriate, the transfer from institutional care to a less restrictive setting is not opposed by the affected individual, and the placement can be reasonably accommodated, taking into account the resources available to the State and the needs of others with mental disabilities." Although the case was not specific to brain-injury survivors, the ruling allowed the BIAA and others to advocate for these individuals, many of whom had been inappropriately placed in psychiatric hospitals or other institutional settings that were not designed for their unique needs. The ruling tasked the states to plan reforms in treatment, transportation, housing, education, and social support, in order to integrate brain injury survivors (among others) into the least restrictive setting possible. To aid state agencies working toward compliance, the BIAA partnered with Independent Living Research Utilization to provide regional training workshops regarding the content of the Olmstead decision, its intended effect, and potential strategies to meet the community needs of individuals with cognitive disabilities who might otherwise be wrongly placed in institutional settings.

The BIAA in coordination with the Brain Trauma Foundation, the American Association of Neurological Surgeons, and other professional contributors, developed and published *Guidelines for the Management of Severe Brain Injury* (2000). The BIAA was also involved in authoring *Management and Prognosis of Penetrating Brain Injury* (Aarabi et al. 2001). These publications were created to provide up-to-date, evidence-based guidelines and protocols to improve the outcome of TBI patients.

As part of its ongoing legislation efforts to increase private insurance, Medicare, and TriCare coverage for cognitive rehabilitation, the BIAA published *Cognitive Rehabilitation: The Evidence, Funding, and Case for Advocacy of Brain Injury* (Katz, Ashley, O'Shanick, and Connors, 2006), which included ten recommendations to increase access and delivery of cognitive rehabilitation services across the nation.

In 1996, the BIAA founded the American Academy for the Certification of Brain Injury Specialists (AACBIS) which to date has certified 2,800 members. The mission of AACBIS is to improve the care provided to individuals with brain injury through enhanced education and training of their health care providers. Training is provided by volunteers and based primarily upon *The Essential Brain Injury Guide* (American Academy for the Certification of Brain Injury Specialists, 2007), which was also published by the BIAA. Certification is granted to those with appropriate work experience who have successfully completed the training and written examination.

Major Activities

The BIAA has demonstrated a long-term commitment to shaping public policy and partnering with governmental agencies. It has repeatedly worked to preserve and expand rehabilitation options for persons with brain injury, particularly Medicare and Medicaid beneficiaries. It has worked to secure federal funding for research and public

education. The BIAA remains active in disability advocacy and has provided consultation and assistance in developing numerous legislative proposals that benefit those who have sustained brain injury. Encouraging private/public partnerships, particularly in the care of military service-related brain injury, has been a crucial area of intervention. Public policy initiatives that enhanced trauma care, child abuse prevention, transportation safety, education, and respite care were among the association's 2007 accomplishments (G. Ayotte, personal communication, November, 2008)

The BIAA views brain injury prevention and awareness as a primary component of its mission. The association has distributed information kits, produced public service announcements, and provided access to subject matter experts for a number of media outlets. The BIAA also publishes *TBI Challenge!*, a quarterly newsletter with a distribution that includes 25,000 households. The BIAA hosts/co-hosts a number of educational meetings and conferences, maintains a comprehensive website, and publishes the National Directory of Brain Injury Rehabilitation Services to provide survivors and/or family members with helpful information about services in their community. On an annual basis the BIAA responds to over 100,000 requests for assistance through either its national information call center or its website. Each March the BIAA circulates 10,000 information kits to mark Brain Injury Awareness Month.

Cross References

► Traumatic Brain Injury

References and Readings

Aarabi, B., Alden, T. D., Chestnut, R. M., Downs J. H., Ecklund, J. M., Eisenberg, H. M., et al. (2001). Management and prognosis of penetrating brain injury. *Journal of Trauma, Injury, Infection and Critical Care, 51*, S1–S86.

American Academy for the Certification of Brain Injury Specialists (2007). *The essential brain injury guide* (4th ed.). McLean, VA: Brain Injury Association of America.

Brain Injury Association of America. (n.d.). *Brain injury association USA home page*. Retrieved December 20, 2008, from http://www.biausa.org

Brain Injury Association of America (2005). *Academy of certified brain injury specialists (ACBIS)*. Retrieved December 20, 2008, from http://www.aacbis.net/index.html

Brain Trauma Foundation, American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care (2000). Guidelines for the management of severe traumatic brain injury. *Journal of Neurotrauma, 17*, 451–627.

Defense and Veterans Brain Injury Center. (n.d.). *Defense and veterans brain injury center: Home of defense and veterans head injury program*. Retrieved December 20, 2008, from http://www.dvbic.org

Katz, D. I., Ashley, M. J., O'Shanick, G. J., & Connors, S. H. (2006). *Cognitive rehabilitation: The evidence, funding, and case for advocacy of brain injury*. McLean, VA: Brain Injury Association of America.

Brain Plasticity

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Definition

Plasticity refers to the brain's ability to change its structure in response to development, the environment, or injury.

Current Knowledge

Brain plasticity, or neuroplasticity, refers to the brain's ability to change in response to development, to the environment (including learning), and in response to injury or aging. While it was once conceptualized that once the brain ceases development, that it would then be resistant to change, or in effect be static. Research over the last several decades has demonstrated that the brain continues to be capable of change, or restructuring, throughout the life span. While much research with respect to brain plasticity focuses on outcomes following injury, brain plasticity also refers to developmental changes that occur in the brain throughout the life span, including synaptic changes that occur in response to the acquisition of new learning and memories. As such, plasticity is seen as an intrinsic property of the central nervous system in that the brain is constantly restructuring and reorganizing in response to new learning. Specifically, studies of learning new behaviors, such as learning Braille, result in the rapid, but transient onset of cortical enlargement that gives way to a more stable but less dramatic cortical enlargement associated with plasticity.

Research has demonstrated that a variety of factors affect plasticity of neural reorganization and proliferation. One such environment includes the richness of the environment in which an organism is grown. Specifically, organisms that grow in a richly stimulating environment in which a variety of experiences are encountered have greater plasticity than individuals who are reared in less-

stimulating environments. Empirical experimentation in humans and animals alike have demonstrated that the dendrite length as well as the density of synapses in organisms with enriched motor and sensory environments surpass those raised in less-stimulating environments. However, these differences appear only to exist with early learning environments, as adolescents and adults show no such sensitivity to environmental factors. Both gender and hormone differences also appear to play a role in neural plasticity, with specific respect to cortical areas; for example, while males are more sensitive to experience related to the visual cortex, females are more sensitive to development in the hippocampal area.

However, the concept of brain plasticity may be best understood by examining the processes by which the brain changes in response to injury. Responses to injury may result in the loss of a previously held behavior, release of a previously suppressed behavior, the assumption of a function by a neighboring neuronal network, or the development of new behaviors (which may be adaptive or maladaptive). Physiologically, reorganization can occur by changing the balance between excitatory and inhibitory synaptic and membrane responses as well as by strengthening or weakening synaptic connections. Brain plasticity can also involve the growth of new dendrites and axons to form new synaptic connections. Research suggests that the age of onset of the injury is critical in the development of new connections, as long connections are more difficult to form in the mature brain, whereas the young brain may be more capable of forming long connections due to the existence of excess connectivity. Changes in connectivity can occur through the strengthening or weakening of synaptic density or the rearrangement of synaptic connections. While the concept of neuroplasticity does at times result in recovery of adaptive behavioral changes, plasticity may also lead to unmasking of previous suppressed and maladaptive behaviors as well as the development of dysfunctional behaviors.

Brain plasticity may occur via multiple difference mechanisms. Perhaps the most common, and best understood, mechanism includes the expansion of a specified area of circuitry or the recruitment of either a local or distal area of circuitry. Such reorganization of function is a common post-injury response and occurs shortly following the injury and continues to develop years following the injury as the organism adapts. Remodeling or reorganization occurs both at the cortical and subcortical level, and can occur both within and between functional modalities. For example, much literature exists examining the reorganization of neuronal circuitry in response to blindness. Early onset blindness results in the functional loss of a large area of the brain. The learning of alternative communication techniques, such as Braille, requires the adaptation of new behavior; research has demonstrated that individuals who have learned Braille have larger sensory maps related to the finger pad used in reading as compared to the contralateral equivalent or as compared to individuals who do not read Braille. Furthermore, not only do blind individuals develop enlarged corresponding sensory maps, it has also been demonstrated that the occipital, or visual, cortex is subsequently recruited for tactile information processing, as well as auditory information processing.

The advancement of technology has furthered our understanding of the existence of, and mechanisms behind, neuroplasticity. The ability to specify and characterize brain function via visualization of glucose and oxygen metabolism has allowed for exploration of mechanisms of plasticity. Processes of functional neuroimaging, including positron emission tomography as well as functional magnetic resonance imaging allows for indirect visualization of synaptic activity; experimentation involving tasks being performed while the brain is being imaged allows for examination of synaptic changes. Magnetic resonance spectroscopy is thought to be a promising technique that allows for analysis of the connection between neurochemical changes and behaviors. Electroencephalography and magnetoencephalography allows to direct measurement of neuronal activity; however, it lacks structural specificity. Transcranial magnetic stimulation also allows for direct analysis of neural activity by temporarily suppressing brain regions, allowing for direct assessment of brain-behavior relationships in conjunction with functional neuroimaging techniques.

References and Readings

Galaburda, A., & Pascual-Leone, A. (2003). Mechanisms of plasticity and behavior. In T. E. Feinberg & M. J. Farah (Eds.), *Behavioral neurology & neuropsychology* (2nd ed., pp. 57–70). New York, NY: McGraw-Hill.

Kolb, B. (1995). *Brain plasticity and behavior.* Mahwah, NJ: Erlbaum.

Kolb, B., Gibb, R., & Robinson, T. E. (2003). Brain plasticity and behavior. *Current Directions in Psychological Science, 12*(1), 1–5.

Kolb, B., & Whishaw, I. Q. (1998). Brain plasticity and behavior. *Annual Review of Psychiatry, 49*, 43–64.

Nelson, C. A., & Luciana, M. (2001). *Handbook of developmental cognitive neuroscience.* Boston, MA: Massachusetts Institute of Technology.

Brain Reserve

► Cognitive Reserve

Brain Reserve Capacity

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Synonyms

Global reserve; Reserve

Definition

Brain reserve capacity is the brain's resilience to pathological damage or changes. The greater the brain reserve capacity, the less likely an individual will demonstrate behavioral disturbance associated with a disease.

Historical Background

Research has attempted to understand the role of various factors involved in cognitive decline. Frequent central nervous system disorders occur in the elderly, which increase the likelihood of cognitive decline. Age, in itself, is a factor known to alter cognitive functioning. Brain reserve capacity is the brain's ability to effectively manage the increasing changes in normal aging and to cope with pathological damage.

Post-mortem examination of elderly individuals provides evidence that that there is a discrepancy between the clinical manifestation of Alzheimer's disease and the neuropathology of the disorder (Katzman et al., 1998). Specifically, this early study found that a subset of individuals whose brains were found to have a high degree of pathology associated with Alzheimer's disease did demonstrate minimal clinical symptoms associated with the disease. Interestingly, the results from this study suggested that the weight of the brains in this subset of patients was higher. These patients were also found to have more neurons. It was subsequently concluded that perhaps these patients' larger brains and their possession of more neurons were protective against dementia symptoms. While subsequent studies have been inconclusive, many studies have suggested that head circumference, brain volume, intracranial volume, and genetic influences also play an important role in brain reserve capacity.

Current Knowledge

Research consistently demonstrates that the underlying neuropathology is not consistent with behavioral disturbance caused by dementia. Brain reserve capacity partially explains this phenomenon. It appears that symptomatic behaviors are less likely to be prevalent in individuals with greater brain reserve capacity. Research has also consistently found that cognitive reserve capacity, that is the lifestyle approaches that encourage cognitive activity, plays an important role in functional ability despite neuropathological changes. It appears plausible that brain reserve capacity, such as increased amount of neurons and neuronal connections, is at least in part due to behaviors that encourage cognitive reserve capacity, such as education and occupation. The interplay of cognitive activity and brain reserve capacity is being carefully studied in an attempt to understand the relationship.

Future Directions

Identifying factors that increase the likelihood of brain reserve capacity has the possibility of being an invaluable tool toward improving the quality of elderly people's lives and potentially reducing the risk of developing Alzheimer's disease. The National Institute of Aging and other federally funded programs have invested millions of dollars to better understand the factors in improving brain reserve capacity. Studies will continue to better understand factors that increase brain cells, synaptic connections, and other neurophysiological markers. The implementation and advancement of technology will assist in providing a clearer understanding of these factors as well.

Cross References

► Alzheimer's Disease
► Cognitive Reserve

References and Readings

Katzman, R., Terry, R., DeTeresa, R., Brown, T., Davies, P., Fuld, P., et al. (1988). Clinical, pathological and neurochemical changes in dementia: a subgroup with preserved mental status and numerous neocortical plaques. *Annals of Neurology, 23*, 138–144.

Stern, Y. (2006). *Cognitive Reserve, Theory and Application*. Series Editor L. Bieliauskas. New York, NY: Psychology Press.

Brain Storming

► Hemodynamic Response

Brain Swelling

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Definition

Expansion of the size of the brain that occurs following head trauma and brain injury.

Current Knowledge

Together with brain edema, brain swelling can elevate intracranial pressure. Once intracranial pressure is elevated, oxygen, glucose, and blood have difficulty reaching all portions of the brain. Blood vessels are no longer efficient in carrying blood, oxygen, and nutrients throughout the brain. As a consequence, increased intracranial pressure complicates the degree of brain injury and also the brain's natural response to trauma.

Brain swelling can occur in 15–20% of severe brain injuries. The exact mechanism that leads to brain swelling is poorly understood, but once trauma is sustained, the brain tissue swells to compress harder and harder against the rigid skull. Brain swelling must be managed emergently following brain injury because patients experiencing brain swelling are at a higher risk of death. The brain may swell to a point in which portions of the brain herniate through openings in the skull. Brain swelling may compress the brainstem, the area of the brain that maintains consciousness, and critical life functions, such as cardiac function and respiration. Methods of managing brain swelling involve administering medications to constrict blood vessels, drilling a burr hole or conducting decompressive craniotomy to relieve pressure, temporarily removing a portion of the skull in a decompressive craniectomy to relieve pressure (and replacing the skull fragment once pressure is normalized), placing an external drain to relieve pressure and excess fluid from the surface of the brain, placing the patient on artificial respirator so that carbon dioxide does not accumulate in the brain, inducing hypothermia, and using an electronic intracranial pressure monitor with a valve to adjust pressure over time.

Cross References

► Edema
► Intracranial Pressure

References and Readings

Marmarou, A. (2007). A review of progress in understanding the pathophysiology and treatment of brain edema. *Neurosurgery Focus, 22*, E1.

Brain Tumor

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Definition

An abnormal mass of tissue in which some cells (glial or non-glial) grow and multiply uncontrollably. A tumor can be benign or malignant. It is associated with damage or mutation to the TP53 gene on human chromosome 17. P53 regulates the cell cycle and functions in tumor suppression. A tumor can cause damage by increasing pressure in the brain, by shifting the brain or pushing against the skull, and by invading and damaging nerves and healthy brain tissue. Some tumors may be truly indolent in their growth, growing so slowly that they are present for an unknown length of time because symptoms are less gross and disruptive. Those that are actively growing may be more likely to present with the following symptoms, depending on tumor locus: headaches; nausea or vomiting; seizures or convulsions; difficulty in thinking, speaking, or finding words; personality changes; weakness or paralysis in one part or one side of the body; loss of balance; vision changes; confusion and disorientation; and memory loss (Levin, Leibel, & Gutin, 2001; Price, Goetz, & Lovell, 2007).

Cross References

► Astrocytoma
► Glioma
► Neuroblastoma
► Neurocytoma

References and Readings

Levin, V. A., Leibel, S. A., & Gutin, P. H. (2001). Neoplasms of the central nervous system. In V. T. DeVita, S. Hellman, & S. A. Rosenberg (Eds.), *Cancer: principles and practice of oncology* (pp. 2100–2160). Philadelphia, PA: Lippincott, Williams, & Wilkins.

Price, T. R. P., Goetz, K. L., & Lovell, M. R. (2007). Neuropsychiatric aspects of brain tumors. In S. C. Yudofsky & R. E. Hales (Eds.), *The American psychiatric publishing textbook of neuropsychiatry and behavioral neurosciences* (5th ed., pp. 735–764). Washington, DC: American Psychiatric Association.

Brain with Excessive Weight

► Megalencephaly

Brainstem Auditory Evoked Potentials (BAEP)

► Brainstem Auditory Evoked Responses

Brainstem Auditory Evoked Responses

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Synonyms

Auditory brainstem responses (ABR); Auditory brainstem response audiometry; Auditory evoked response (AER); Brainstem auditory evoked potentials (BAEP); BAER; Brainstem response (BSR)

Definition

Brainstem auditory evoked responses (BAER) test the function of the auditory nerve and auditory pathways of the brain by measuring the electrophysiologic responses to repeated clicks presented to each ear. The response time of electrical waves generated from different anatomical parts of the brain-ear system are plotted as summarized below (Lew, Lee, Pan, & Chiang, 2007):

Wave I: Cochlear nerve (CN VIII)
Wave II: Cochlear nucleus (CN VIII)
Wave III: Superior olivary complex
Wave IV: Lateral lemniscus
Wave V: Inferior colliculus

Waveform delays may indicate an abnormality in function along the auditory pathway. Unilateral delays suggest a lesion to cranial nerve VIII along its pathway or in the brainstem. BAER may be abnormal in acoustic neuroma, demyelinating disease, migraine headaches, multiple sclerosis, brainstem tumor, brainstem stroke, or brain injury of various etiologies. Common uses of BAER include acoustic neuroma detection, multiple sclerosis diagnosis, and intraoperative monitoring during cerebellopontine angle tumor resection. Magnetic resonance imaging (MRI) may provide greater anatomic detail and would be preferable for detecting a small lesion. However, BAER may be particularly useful in an individual who cannot undergo MRI.

Current Knowledge

BAER results are generally not affected by the effect of anesthesia and medications, or peripheral vestibular pathology. BAER is sometimes used for prognostic purposes after brain injury, but its use is limited for this purpose. Complete absence of responses is considered an ominous sign (Lew et al., 2007), and abnormal BAER may confirm suspicion of brainstem injury, while normal BAER simply indicates preservation of the auditory pathways through the brain. BAER does not reveal information about damage that may have occurred elsewhere in the brain, and thus, normal BAER does not necessarily predict good outcome (Lew et al., 2007; Zafonte, Hammond, & Peterson, 1996).

Cross References

► Evoked Potentials

References and Readings

Huszar, L. (2006). Clinical utility of evoked potentials. *eMedicine*. Retrieved July 9, 2007 from http://www.emedicine.com/neuro/topic69.htm

Lew, H. L., Lee, E. H., Pan, S. S. L., & Chiang, J. Y. P. (2007). Electrophysiologic assessment techniques: evoked potentials and electroencephalography. In N. D. Zasler, D. I. Katz, R. D. Zafonte (Eds.), *Brain Injury Medicine* (p. 158). New York: Demos.

Zafonte, R. D., Hammond, F. M., & Peterson, J. (1996). Predicting outcome in the slow to respond traumatically brain-injured patient: Acute and subacute parameters. *NeuroRehabilitation, 6*, 19–32.

Brainstem Glioma

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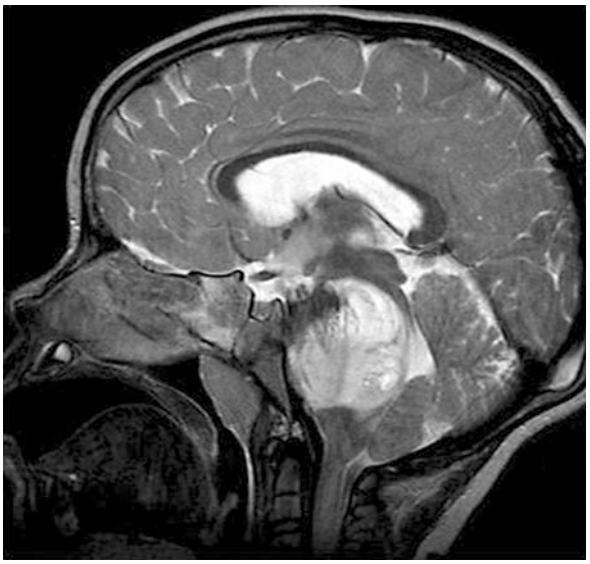
Synonyms

Midbrain glioma; Pontine glioma

Definition

Brainstem gliomas are highly aggressive tumors of the central nervous system occurring more frequently in children than in adults (Fig. 1). These type of tumors often originate from the left side and typically involve one of three anatomical locations within the brain stem. Pontine brainstem gliomas are associated with the poorest prognosis for survival, while tectal and cervicomedullary gliomas are associated with longer survival. Tectal brainstem gliomas are often associated with hydrocephalus as a result of compression of the fourth ventricle. Typical manifestations of cervicomedullary tumors include dysphagia, unsteadiness, nasal speech, and sensory loss in the face. Pontine brainstem gliomas are associated with cranial nerve or long tract symptoms, including problems with the control of facial muscles, ocular movements, and swallowing. Diffuse brainstem gliomas, once thought to be a single entity, are now thought to comprise a group of tumors with varying courses and outcomes. Brainstem gliomas can also occur in the cervicomedullary junction, pons, midbrain, and tectum; prognosis is worse and very grim for diffuse brainstem gliomas. Diffuse brainstem gliomas do not typically enhance on MRI. They are not responsive to radiotherapy, and treatment is usually limited to chemotherapy.



Brainstem Glioma. Figure 1 Picture credit: Michael Phillips and Peter C. Fisher

References and Readings

Albright, L., Pollack, I., Adelson, P., Humphreys, R., George, T., Painter, M., et al. (2007). *Principles and practice of pediatric neurosurgery* (2nd ed.). New York: Thieme.

Donaldson, S., Laningham, F., & Fisher, P. (2006). Advances toward an understanding of brainstem gliomas. *Journal of Clinical Oncology, 24*(8), 1266–1272.

Brainstem Response (BSR)

► Brainstem Auditory Evoked Responses

Brainstem Strokes

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Definition

A stroke that is caused by ischemia or hemorrhage in the midbrain, pons, or medulla is called a "brainstem stroke." There are many brainstem stroke clinical syndromes, the

presentation of each depending on the specific location in the brain stem that is involved. Most brainstem stroke syndromes result from ischemia due to partial blockage or complete occlusion of arteries in the vertebrobasilar system located at the posterior region of the brain.

Current Knowledge

Localization of the brainstem lesion can usually be made by recognizing the specific pattern of clinical deficits and understanding the anatomical basis for these clinical manifestations. Many of these strokes cause dysfunction of one or more of the many cranial nerves that originate from the brain stem. The specific clinical dysfunction, typically involving head and neck functions, localizes the tissue injury to the side that is ipsilateral to the clinical deficit. Some also involve motor or sensory deficits of the body, which localize the injury to the side that is contralateral to the clinical deficit. When cerebellar signs such as ataxia and discoordination are present in association with other brainstem findings, this localizes the lesion to the ipsilateral side, and usually to the pons. When unilateral facial and contralateral body sensory deficits exist, this also localizes the lesion to the brain stem. Other symptoms, such as vertigo, double vision, nausea, and selected tremors, are also reflective of dysfunction of certain specific brainstem structures. Because the brain stem also contains the life support centers that control respiration, blood pressure, and heart rate, a brainstem stroke has the potential to be fatal.

In its most severe form, an infarction of the ventral pons can interrupt the function of all motor pathways, causing locked-in syndrome, in which the patient can receive and understand sensory stimuli, but has no motor control, resulting in complete total body paralysis and inability to speak, while maintaining awareness and sensation.

MRI scanning usually facilitates the diagnosis of brainstem stroke.

Cross References

- ▶ Basilar Artery
- ▶ Cerebrovascular Disease
- ▶ Lacunar Infarction
- ▶ Locked-In Syndrome
- ▶ Posterior Cerebral Artery
- ▶ Posterior Communicating Artery
- ▶ Pure Motor Stroke
- ▶ Thalamic Hemorrhage
- ▶ Vertebrobasilar System

References and Readings

Chua, K., & Kong, K. (1996). Functional outcome in brain stem stroke patients after rehabilitation. *Archives of Physical Medicine and Rehabilitation, 77*, 194–197.

Dauby, J-D. (1996). *The diving bell and the butterfly: A memoir of life in death*. New York: Vintage.

Nelles G., et al. (1998). Recovery following lateral medullary infarction. *Neurology, 50*, 1418–1422.

Breathing Tube

- ▶ Endotracheal Tube

BRI

- ▶ Sick Building Syndrome

BRIEF

- ▶ Behavior Rating Inventory for Executive Functions

Brief Cognitive Rating Scale

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Synonyms

BCRS

Description

The Brief Cognitive Rating Scale (BCRS; Reisberg & Ferris, 1988) is used to assess functional and cognitive abilities in both normal aging and progressive dementia. The BCRS is part of the Global Deterioration Scale Staging System (GDS; Reisberg, Ferris, de Leon, Crook, 1993) which is composed of three separate rating scales that include the GDS, the Functional Assessment Staging (FAST; Reisberg,

1988), and the BCRS. The BCRS provides objective ratings of a number of domains that include various cognitive functions as well as functional abilities, mood, and behavior, and is made up of two parts. Part I includes ratings for Concentration, Recent Memory, Remote Memory, Orientation, and Functioning and Self Care, while Part II allows for ratings of Speech and Language Abilities, Motoric Capacities, Mood and Behavior, Praxis Ability, Calculation Ability, and Feeding Capacity.

Each of the domains is rated on a 1–7 point scale that ranges from normal (rating of 1) to profound impairment (rating of 7). For each domain, a behavioral anchor is provided for each point on the rating scale. The authors provided examples of questions that might be used to elicit information needed to complete the BCRS as well as guidelines for scoring each domain. Ratings are completed based on interviews with the patient and an informant who is knowledgeable regarding the patient's day-to-day activities and functioning. Interviews may be conducted in person or over the telephone. The BCRS has been translated into a number of languages including Chinese, French, Polish, Spanish and Swedish, among others.

Current Knowledge

From a psychometric standpoint, the BCRS has excellent interrater reliability when completed by trained clinicians, and test-retest reliability is also high (Foster et al., 1988; Reisberg et al., 1989). Validity studies indicate the BCRS has strong correlations with the GDS, the Mini-Mental State Examination, some neuropsychological measures of memory abilities, as well as measures of activities of daily living and quality of life. Relationships have also been demonstrated between BCRS scores and neuropathology measured using a variety of techniques (EEG, PET, SPECT, neurological examination). BCRS scores are sensitive to the progression of Alzheimer's disease, with significant declines in scores as the disease progresses (Ihl, Frölich, Dierks, Martin, Maurer, 1992). Thus, the BCRS is useful in both research and clinical settings where it can provide valuable information regarding progression of cognitive decline, as well as the impact that such decline has on behavior and function.

Cross References

- ▶ Alzheimer's Dementia
- ▶ Alzheimer's Disease
- ▶ Mini-Mental State Examination
- ▶ Multi-Infarct Dementia

References and Readings

Foster, J. R., Sclan, S., Welkowitz, J., Boksay, I., & Seeland, I. (1988). Psychiatric assessment in medical long-term care facilities: Reliability of commonly used rating scales. *International Journal of Geriatric Psychiatry, 3*, 229–233.

Ihl, R., Frölich, L., Dierks, T., Martin, E., & Maurer, K. (1992). Differential validity of psychometric tests in dementia of the Alzheimer type. *Psychiatry Research, 44*, 93–106.

Reisberg, B. (1988). Functional assessment staging (FAST). *Psychopharmacology Bulletin, 24*, 653–659.

Reisberg, B., & Ferris, S. H. (1988). Brief cognitive rating scale (BCRS). *Psychopharmacology Bulletin, 24*, 629–636.

Reisberg, B., Ferris, S. H., de Leon, M. J., & Crook, T. (1988). Global deterioration scale (GDS). *Psychopharmacology Bulletin, 24*, 661–663.

Reisberg, B., Ferris, S. H., Steinberg, G., Shulman, E., de Leon, M. J., & Sinaiko, E. (1989). Longitudinal study of dementia patients and aged controls. In M. P. Lawton, & A. R. Herzog (Eds.), *Special research methods for gerontology* (pp. 195–231). Amityville, NY: Baywood.

Reisberg, B., Sclan, S. G., de Leon, M. J., Kluger, A., Torossian, C., Shulman, E., Steinberg, G., Monteiro, I., McRas, T., Mackell, J., & Ferris, S. H. (2007). The GDS staging system. In A. J. Rush (Ed.), *Handbook of psychiatric measures* (pp. 431–435). Washington, DC: American Psychiatric Publishers.

Brief Evaluation

- ▶ Neuropsychological Screening Examination

Brief Psychiatric Rating Scale

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Synonyms

BPRS

Definition

The Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1962, 1988) consists of a series of 18 items

assessing the following psychiatric symptoms: somatic concern, anxiety, emotional withdrawal, conceptual disorganization, guilt feelings, tension, mannerisms and posturing, grandiosity, depressive mood, hostility, suspiciousness, hallucinatory behavior, motor retardation, uncooperativeness, unusual thought content, blunted affect, excitement, and disorientation. The instrument takes approximately 5–10 min to complete, following an interview with the patient. The clinician rates each item on a scale ranging from 1 (*not present*) to 7 (*extremely severe*). The inventory is geared toward serious psychopathology. While the ratings on individual items are meaningful, the BPRS can yield an overall score, and sets of items can be grouped into categories.

Current Knowledge

The BPRS has acceptable psychometric properties and enjoys widespread clinical use. Several authors have developed versions of the BPRS with behavioral anchors to improve reliability, or training methods to accomplish the same (Ventura, Green, Shaner, & Liberman, 1993). Using a behaviorally anchored approach and no specific interview format or rater training, Lachar et al. (2001) obtained weighted kappa agreement between psychiatrists on the majority of items at above 0.75. Scores on the BPRS are highly correlated with those of other similar instruments although, as with all such clinician-rated scales, relationships with external criteria are modest (Mortimer, 2007).

Initially developed for use with psychiatric populations, the BPRS is one of the most common scales used to track outcomes in schizophrenia research (Dunayevich, Sethuraman, Enerson, Taylor, & Lin, 2006). It has been used effectively with individuals having other psychiatric disorders (e.g., bipolar disorder; Picardi et al., 2008), as well as those with neurological conditions having psychotic or other psychiatric symptoms, including Alzheimer's disease, dementia with Lewy bodies, and Parkinson's disease (Cummings, Raman, & Thai, 2007; Devanand, 1998; Tariot, Profenno, & Ismail, 2004). It has also been used in a modified form with persons with traumatic brain injury (Levin et al., 1987). In neuropsychological practice, the BPRS can enable the clinician to organize and quantify observations of psychotic symptoms or other seriously disordered behavior, both as part of an evaluation and in tracking the course of the clinical condition over time.

The BPRS is in the public domain, and may be found in Overall & Gorham (1988). A behaviorally anchored format may be found in Woemer, Mannuzza, and Kane (1988). Versions are also widely available online, including at: http://www.public-health.uiowa.edu/icmha/outreach/documents/BPRS_expanded.PDF.

Cross References

- ▶ Affective Disorder
- ▶ Alzheimer's Dementia
- ▶ Alzheimer's Disease
- ▶ Anxiety
- ▶ Clinical Interview
- ▶ Dementia with Lewy Bodies
- ▶ Parkinson's Dementia
- ▶ Parkinson's Disease
- ▶ Psychosis
- ▶ Psychotic Disorder
- ▶ Structured Clinical Interview for DSM-IV (SCID)
- ▶ Traumatic Brain Injury

References and Readings

Cummings, K. R., Raman, R., & Thai, L. (2007). Quetiapine for agitation or psychosis in patients with dementia and parkinsonism. *Neurology, 68*, 1356–1363.

Devanand, D. P. (1998). A randomized, placebo-controlled dose-comparision of a trial of haloperidol for psychosis and disruptive behaviours in Alzheimer's disease. *American Journal of Psychiatry, 155*, 1512–1520.

Dunayevich, E., Sethuraman, G., Enerson, M., Taylor, C. C., & Lin, D. (2006). Characteristics of two alternative schizophrenia remission definitions: Relationship to clinical and quality of life outcomes. *Schizophrenia Bulletin, 86*, 300–308.

Lachar, D., Bailley, S. E., Rhoades, H. M., Espandas, A., Aponte, M., Cowan, K. A., et al. (2001). New subscales for an anchored version of the Brief Psychiatric Rating Scale: Construction, reliability and validity in acute psychiatric admissions. *Psychological Assessment, 13*, 384–395.

Levin, H. S., High, W. M., Goethe, K. E., Sisson, R. A., Overall, J. E., Rhoades, H. M., Eisenberg, H. M., Kalisky, Z., & Gary, H. E. (1987). The neurobehavioural rating scale: Assessment of the behavioural sequelae of head injury by the clinician. *Journal of Neurology, Neurosurgery and Psychiatry, 50*, 183–193.

Mortimer, A. M. (2007). Symptom rating scales and outcome in schizophrenia. *British Journal of Psychiatry Supplement, 50*, s7–s14.

Overall, J. E., & Gorham, D. R. (1962). The brief psychiatric rating scale. *Psychological Reports, 10*, 790–812.

Overall, J. E., & Gorham, D. R. (1988). The Brief Psychiatric Rating Scale (BPRS): Recent developments in ascertainment and scaling. *Psychopharmacology Bulletin, 24*, 97–99.

Picardi, A., Battisti, F., de Girolamo, G., Morosini, P., Norcio, B., Bracco, R., et al. (2008). Symptom structure of acute mania: A factor study of the 24-item Brief Psychiatric Rating Scale in a national sample of

patients hospitalized for a manic episode. *Journal of Affective Disorders, 108*, 183–189.
Tariot, P. N., Profenno, L. A., & Ismail, M. S. (2004). Efficacy of atypical antipsychotics in elderly patients with dementia. *Journal of Clinical Psychiatry, 65*(Suppl. 11), 11–15.
Ventura, J., Green, M. F., Shaner, A., & Liberman, R. P. (1993). Training and quality assurance on the Brief Psychiatric Rating Scale. *International Journal of Methods in Psychiatric Research, 3*, 221–244.
Woerner, M. G., Mannuzza, S., & Kane, J. M. (1988). Anchoring the BPRS. *Psychopharmacology Bulletin, 24*, 112–117.

Brief Symptom Inventory

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Synonyms

BSI; BSI-18

Description

The BSI (Derogatis & Melisaratos, 1983) is a shortened, 53-item version of the Symptom Checklist-90 (SCL-90; Derogatis, Lipman, & Covi, 1973) that measures emotional-behavioral functioning in nine dimensions: somatization, obsessive–compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism. Four additional items not specific to any one domain load on several different dimensions. In addition, three global indices – Global Severity Index (GSI), Positive Symptom Total (PST), and Positive Symptom Distress Index (PSDI), provide more general assessment of psychological well-being. The BSI-18 (Derogatis, 2000), an 18-item shortform of the BSI intended as a screen for psychiatric disorders and psychological distress, consists of three six-item subscales: somatization, depression, and anxiety. A GSI also can be calculated.

Not intended for use as diagnostic tools, both instruments are designed to identify psychological symptoms in medical populations, psychiatric patients, and community non-patients, with separate norms for males and females. Written at a sixth grade reading level and available in over 24 languages, these self-report measures can be hand- or computer-administered to individuals ages 13 and older (BSI) or 18 and older (BSI-18). Respondents rate the extent to which a specific problem has distressed them in the past 7 days (although evaluations over other time intervals may be specified), using a 5-point scale (0 = *not at all* to 4 = *extremely*). Administration is straightforward, taking 4 (BSI-18) to 8–10 min (BSI) to complete.

Scores are determined by summing values for each symptom dimension and then dividing by number of items endorsed in the respective dimension. A GSI can be calculated for either measure by adding the scores for all subscales, as well as the additional items (in the BSI), and then dividing by number of responses. For the BSI only, the PST is determined by counting the number of items endorsed with a positive (nonzero) response, and the PSDI is derived by dividing the sum of the item values by the PST. Raw scores can be converted to standardized T scores, generating a profile that graphically illustrates a respondent's current psychological symptom presentation. Interpretation of the BSI can be done at three levels: global scores, primary symptom dimensions, and discrete symptoms (Derogatis & Melisaratos, 1983). Computer administration, scoring, and interpretation programs are available for both instruments.

Advantages of the BSI and the BSI-18 are that they can be completed quickly and used for repeated assessments. Also, both measures are reported to be sensitive to mild-to-severe psychological distress, making them useful with many populations. Limitations include typical concerns associated with self-report measures, such as possible patient response bias and over or underreporting, as well as limited utility with some medical populations (e.g., neurologic), given the paucity of acceptable norms and concerns that certain items on the BSI may be closely related to physical and cognitive symptoms (Slaughter, Johnstone, Petroski, & Flax, 1999).

Historical Background

In response to the need for briefer evaluation tools relevant in a variety of assessment settings, including medical and industrial research studies, the BSI and BSI-18 were derived from the SCL-90, a 90-item checklist that includes the same nine symptom dimensions and three global indices as the BSI. The SCL-90, itself, was developed in 1973 (Derogatis et al., 1973) and was derived from the

Hopkins Symptom Checklist (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974). The BSI was published in 1983 (Derogatis & Melisaratos, 1983), and the BSI-18 followed in 2000 (Derogatis, 2000).

Psychometric Data

The most recent BSI manual (Derogatis, 1993) provides normative data from four samples: (1) 1,002 heterogeneous psychiatric outpatients; (2) 914 adult non-patients; (3) 423 psychiatric inpatients; and (4) 2,408 adolescent non-patients. The BSI-18 was normed on two samples: (1) 1,134 community adults and (2) 1,543 adult cancer patients. Additional norms have been developed for such diverse samples as older adults (Hale, Cochran, & Hedgepath, 1984), college students (Cochran & Hale, 1985), individuals with spinal cord injury (Heinrich, Tate, & Buckelew, 1994), British psychiatric outpatients (Ryan, 2007) and community-dwelling adults (Francis, Rajan, & Turner, 1990), Israeli adolescents (Canetti, Shalev, & De-Nour, 1994), and others (see Derogatis, 1993). Internal consistency coefficients are strong for both the BSI (0.71–0.83) and BSI-18 (0.74–0.89). Test–retest reliability also is good, ranging from 0.68 to 0.91 (BSI) and 0.68 to 0.84 (BSI-18).

BSI and BSI-18 manuals cite a variety of studies supporting validity in a range of settings and populations, including psychoneuroimmunology, psychopathology, pain assessment and management, HIV research, student mental health, and general clinical studies. For example, in symptomatic adults, convergent validity between the BSI and the Minnesota Multiphasic Personality Inventory (MMPI) was shown to be 0.30–0.72 (Derogatis & Melisaratos, 1983) for the Wiggins content scales (i.e., 13 scales tapping content areas such as social maladjustment and authority conflict) and Tryon cluster scales (i.e., seven scales assessing conceptual clusters such as social introversion and bodily symptoms). Convergent validity also has been demonstrated between the BSI and several other scales in predicting affective status among chronic pain patients (Kremer, Atkinson, & Ignelzi, 1982). High correlations were also found between the SCL-90 and the BSI (0.92–0.96; Derogatis, 1993) and BSI-18 (0.91–0.96; Derogatis, 2000). In terms of predictive validity, the BSI has been shown to be a good predictor of psychopathology in several populations, including a community unipolar depression cohort (Amenson & Lewinsohn, 1981), drug-using adults (Buckner & Mandell, 1990), and the elderly (Hale et al., 1984). Screening studies completed with medical cohorts (e.g., Kuhn, Bell, Seligson, Laufer, & Lindner, 1988) found the BSI to be a strong and reliable predictor of psychological distress; whereas, the BSI-18 has been demonstrated to predict levels of distress in cancer patients (e.g., Recklitis, Parsons, Shih, Mertens, & Robison, 2006).

Some concern has been raised about the factor structure and discriminant validity of the BSI and BSI-18. For example, Boulet and Boss (1991) found that BSI subscales correlated with nonanalogous scales on the MMPI, suggesting poor discriminant validity. In terms of factor structure, moderate to high intercorrelations were found among BSI subscales, with one factor explaining over 70% of the variance in a principal-components analysis (Boulet & Boss, 1991). Several cross-cultural studies of the BSI-18 (e.g., Asner-Self, Schreiber, & Marotta, 2006) found only the GSI (versus the three subscales) to be a valid indicator of psychological distress. These researchers suggested that the BSI and BSI-18 assess the degree, but not the exact nature of psychopathology, and therefore GSI scores should be considered the most useful indicator of psychological distress derived from the measures.

Clinical Uses

The BSI and BSI-18 are widely used measures of psychological distress employed with a variety of populations including inpatient and outpatient medical and psychiatric patients, individuals receiving treatment for substance abuse, and college students. Individuals who are of extremely low intelligence, delirious, psychotic, or have motivation to distort their responses are not good candidates for either measure. Given factor-structure concerns noted above (e.g., Boulet & Boss, 1991), the instruments may be used most appropriately as screening tools to alert clinicians to elevated levels of psychological distress, rather than as diagnostic indicators. According to the BSI manual (Derogatis, 1993), the measures are "most useful in clinical and research settings where time is a major limiting variable".

Both measures can be used as onetime assessments or administered repeatedly to evaluate treatment efficacy or trends over time. Both are reported to have been used successfully in primary care settings to assess significant changes in psychological distress and symptoms in patients with medical problems. The measures can be used in nonclinical populations (e.g., to assess caregiver distress) as well.

Both the BSI and BSI-18 may be useful tools for inclusion in neuropsychological assessments, given their

brevity and utility for repeated administrations. The BSI-18 was reported to be a valid screening measure for the overall level of psychological distress in both inpatients and outpatients with traumatic brain injury (Meachen, Hanks, Millis, & Rappaport, 2008). However, the observation that most items in the BSI obsessive–compulsive scale are more reflective of cognitive complaints (e.g., concentration and memory problems) than classic obsessive–compulsive disorder traits (Slaughter et al., 1999) highlights an issue of particular concern to neuropsychologists. Given the overlap between many items and cognitive and physical symptoms, clinicians are urged to interpret elevations cautiously and remain vigilant against misusing scale names for diagnostic purposes. It is crucial not to rely on psychiatric interpretations of elevated scales in neurological patients who have no history of emotional difficulties; instead, BSI and BSI-18 item responses might best be examined individually and used to guide treatment.

Cross References

- ▶ Beck Anxiety Inventory
- ▶ Beck Depression Inventory
- ▶ Brief Psychiatric Rating Scale
- ▶ Center for Epidemiologic Studies-Depression
- ▶ Geriatric Depression Scale
- ▶ Hamilton Rating Scale of Depression
- ▶ Millon Clinical Multiaxial Inventory
- ▶ Minnesota Multiphasic Personality Inventory
- ▶ Personality Assessment Inventory
- ▶ Self-Report Measures
- ▶ Symptom Checklist-90-Revised
- ▶ Zung Self-Rating Depression Scale

References and Readings

Amenson, C. S., & Lewinsohn, P. M. (1981). An investigation into the observed sex difference in prevalence of unipolar depression. *Journal of Abnormal Psychology, 90*, 1–13.

Asner-Self, K. K., Schreiber, J., & Marotta, S. A. (2006). A cross-cultural analysis of the Brief Symptom Inventory-18. *Cultural Diversity and Ethnic Minority Psychology, 12*, 367–375.

Boulet, J., & Boss, M. W. (1991). Reliability and validity of the brief symptom inventory. *Psychological Assessment, 3*, 433–437.

Buckner, C. J., & Mandell, W. (1990). Risk factors for depressive symptomatology in a drug-using population. *American Journal of Public Health, 80*, 580–585.

Canetti, L., Shalev, A. Y., & De-Nour, A. K. (1994). Israeli adolescents' norms of the Brief Symptom Inventory (BSI). *Israel Journal of Psychiatry and Related Sciences, 31*, 13–18.

Cochran, C. D., & Hale, W. D. (1985). College student norms on the Brief Symptom Inventory. *Journal of Clinical Psychology, 41*, 777–779.

Derogatis, L. R. (1993). *Brief Symptom Inventory (BSI): Administration, scoring, and procedures manual* (3rd ed.). Minneapolis, MN: National Computer Systems.

Derogatis, L. R. (2000). *Brief Symptom Inventory-18 (BSI-18): Administration, scoring, and procedures manual.* Minneapolis, MN: National Computer Systems.

Derogatis, L. R., Lipman, R. S., & Covi, L. (1973). SCL-90: An outpatient psychiatric rating scale: Preliminary report. *Psychopharmacology Bulletin, 9*, 13–28.

Derogatis, L. R., Lipman, R. S., Rickels, K., Uhlenhuth, E. H., & Covi, L. (1974). The Hopkins Symptom Checklist (HSCL): A self-report inventory. *Behavioral Science, 19*, 1–15.

Derogatis, L. R., & Melisaratos, N. (1983). The Brief Symptom Inventory: An introductory report. *Psychological Medicine, 13*, 595–605.

Francis, V. M., Rajan, P., & Turner, N. (1990). British community norms for the Brief Symptom Inventory. *British Journal of Clinical Psychology, 29*, 115–116.

Hale, W. D., Cochran, C. D., & Hedgepath, B. E. (1984). Norms for the elderly on the Brief Symptom Inventory. *Journal of Clinical and Consulting Psychology, 52*, 321–322.

Heinrich, R. K., Tate, D. G., & Bucklew, S. P. (1994). Brief Symptom Inventory norms for spinal cord injury. *Rehabilitation Psychology, 39*, 49–56.

Kremer, E. F., Atkinson, J. H., & Ignelzi, R. J. (1982). Pain measurement: The affective dimensional measure of the McGill Pain Questionnaire with a cancer pain population. *Pain, 12*, 153–163.

Kuhn, W. F., Bell, R. A., Seligson, D., Laufer, S. T., & Lindner, J. E. (1988). The tip of the iceberg: Psychiatric consultations on an orthopaedic service. *International Journal of Psychiatry in Medicine, 18*, 375–382.

Meachen, S. J., Hanks, R. A., Millis, S. R., & Rapport, L. J. (2008). The reliability and validity of the Brief Symptom Inventory-18 in persons with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 89*, 958–965.

Recklitis, C. J., Parsons, S. K., Shih, M., Mertens, A., & Robison, L. L. (2006). Factor structure of the Brief Symptom Inventory-18 in adult survivors of childhood cancer: Results from the childhood cancer survivor study. *Psychological Assessment, 18*, 22–32.

Ryan, C. (2007). British outpatient norms for the Brief Symptom Inventory. *Psychology and Psychotherapy: Theory, Research, and Practice, 80*, 183–191.

Slaughter, J., Johnstone, G., Petroski, G., & Flax, J. (1999). The usefulness of the Brief Symptom Inventory in the neuropsychological evaluation of traumatic brain injury. *Brain Injury, 13*, 125–130.

BRIEF-A

- ▶ Behavior Rating Inventory for Executive Functions

BRIEF-P

- ▶ Behavior Rating Inventory for Executive Functions

BRIEF-SR

► Behavior Rating Inventory for Executive Functions

Bristol Activities of Daily Living Scale

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Synonyms

Bristol ADL scale (BADLS); Revised bristol activities of daily living scale (BADLS-R)

Description

The Bristol ADL scale is an informant-rated measure that covers 20 ADLs, both basic and instrumental. Items are rated on a four-point scale (from totally dependent to totally independent, with an additional "not applicable" option).

Historical Background

The BADLS was developed specifically for use in people with dementia, as existing scales were felt to be insensitive to change in this group, having been designed for healthy older adults or people with physical disabilities. Initially, 22 items were included based on the rationale that they appeared in at least two existing ADL measures. Caregivers of people with dementia completed the questionnaire by mail, including feedback on the relevance and importance of the items and response options. Some modifications were made to the scale, with the next version incorporating different response options. Two items on which participants scored at floor and ceiling respectively were removed, leading to the final 20-item version. Bucks and Haworth (2002) stated that the measure is regularly used in 58% of memory clinics in the United Kingdom, but that a revision was needed in order to increase sensitivity to mild cognitive impairment and to reflect changes in understanding of disability (particularly in light of the 2001 WHO framework) since the scale was developed. Bucks and Haworth (2002) also stated that studies evaluating a revised BADLS are underway, but no papers reporting these studies have been published to date (information correct as of 02.06.09).

Psychometric Data

The 22-item preliminary version of the BADLS had good test–retest reliability (r = 0.95, for kappa scores for individual items see Bucks et al., 1996), and evidence of its validity was found through correlations between the BADLS and MMSE scores (r = 0.55), and between BADLS and observed performance ratings (r = 65). The final 20-item version of the BADLS, completed by 50 caregivers of people with dementia (mixed diagnoses), found estimates of reliability and validity consistent with the previous version, with BADLS–MMSE scores correlating at 0.67. Principal components analysis identified a four-factor structure consisting of instrumental ADLs (7 items explaining 40.3% of variance), self care (6 items explaining 10.3% of variance), orientation (5 items explaining 7.5% of variance), and mobility (2 items explaining 7% of variance). Byrne et al. (2000) found that the BADLS was a good measure of change in ADL proficiency over time in people with Alzheimer's disease (AD) receiving anticholinesterase inhibitors, as judged by its correlations with MMSE and ADAS-Cog scores, and sensitivity of 74% and specificity of 65% in detecting improvement/stability versus decline, in comparison with clinician-rated judgments.

A recent systematic review of 12 instrumental ADL scales for persons with dementia (Sikkes, de Lange-de Klerk, Pijnenburg, Scheltens and Uitdehaag, 2009) concluded that the BADLS was of "moderate quality," the highest rating awarded in the review, which was given to only two measures, BADLS and the Disability Assessment for Dementia.

Clinical Uses

Wicklund et al. (2007) noted that the Bristol ADL scale is heavily weighted towards basic ADLs rather than instrumental ADLs, so this should be borne in mind when considering using it. Nonetheless, the BADLS has been used as a primary or secondary outcome measure in a number of clinical trials, including those of

pharmaceutical and psychosocial interventions in people with dementia. Recent examples include open-label and controlled trials on the safety of aspirin (AD2000 Collaborative Group, 2008) and neuroleptic treatments (Ballard et al., 2008) in people with AD, a comparison of cholinesterase inhibitor and glutamate agonist treatment in moderate-severe AD (Jones et al., 2009), and RCTs of reminiscence therapy (Woods et al., 2009) and interpersonal psychotherapy (Burns et al., 2005) for people with Azheimer's disease and other dementias. Bucks and Haworth (2002) have noted that completing the questionnaire may be in itself helpful for caregivers, as it can help them to understand the effects of dementia in real-life terms.

Cross References

- ▶ Alzheimer's Disease Cooperative Study ADL Scale
- ▶ Disability Assessment for Dementia
- ▶ Lawton–Brody IADL Scale
- ▶ The Activities of Daily Living Questionnaire

References and Readings

AD2000 Collaborative Group. (2008). Aspirin in Alzheimer's disease (AD2000): A randomised open-label trial. *Lancet Neurology, 7*, 41–49.

Ballard, C., Lana, M. M., Theodoulou, M., Douglas, S., McShane, R., Jacoby, R., et al. (2008). A randomised, blinded, placebo-controlled trial in dementia patients continuing or stopping neuroleptics (the DART-AD trial). *PLoS Medicine, 5*, 587–599.

Bucks, R. S., Ashworth, D. L., Wilcock, G. K., & Siegfried, K. (1996). Assessment of activities of daily living: Development of the Bristol Activities of Daily Living Scale. *Age and Ageing, 25*, 113–120.

Bucks, R. S., & Haworth, J. (2002). Bristol Activities of Daily Living Scale: A critical evaluation. *Expert Reviews in Neurotherapeutics, 2*, 669–676.

Burns, A., Guthrie, E., Marino-Francis, F., Busby, C., Morris, J., Russell, E., et al. (2005). Brief psychotherapy in Alzheimer's disease. *British Journal of Psychiatry, 187*, 143–147.

Byrne, L. M. T., Wilson, P. M. A., Bucks, R. S., Hughes, A. O., & Wilcock, G. K. (2000). The sensitivity to change over time of the Bristol Activities of Daily Living Scale in Alzheimer's Disease. *International Journal of Geriatric Psychiatry, 15*, 656–661.

Jones, R., Sheehan, B., Phillips, P., Juszczak, E., Adams, J., Baldwin, A., et al. (2009). DOMINO-AD protocol: Donepezil and memantine in moderate to severe Alzheimer's disease – a multicentre RCT. *Trials, 10*, 57.

Sikkes, S. A. M., de Lange-de Klerk, E. S. M., Pijnenburg, Y. A. L., Scheltens, P., & Uitdehaag, B. M. J. (2009). A systematic review of Instrumental Activities of Daily Living scales in dementia: room for improvement. *Journal of Neurology Neurosurgery and Psychiatry, 80*, 7–12.

Wicklund, A. H., Johnson, N., Rademaker, A., Weitner, B. B., & Weintraub, S. (2007). Profiles of decline in activities of daily living in non-Alzheimer's dementia. *Alzheimer's Disease and Associated Disorders, 21*, 8–13.

Woods, R. T., Bruce, E., Edwards, R. T., Hounsome, B., Keady, J., Moniz-Cook, E. D., et al. (2009). Reminiscence groups for people with dementia and their family carers: Pragmatic eight-centre randomised trial of joint reminiscence and maintenance versus usual treatment: A protocol. *Trials, 10*, 64.

Bristol ADL Scale (BADLS)

- ▶ Bristol Activities of Daily Living Scale

Broadmann Area 17

- ▶ Cuneus

Broadmann Areas 18, 19

- ▶ Extrastriate

Broca, Pierre Paul (1824–1880)

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Name and Degrees

Pierre Paul Broca received a bachelor's degree of letters (a subject that includes history and literature) in 1840 from his hometown Sainte-Foy-la-Grande College, and subsequently received a Bachelor of Science degree in mathematics. He began his study of medicine in 1841 at the age of 17 at the Faculty of Medicine at the University of Paris. He completed his formal medical training there in 1848. Broca was studious and progressed rapidly during his medical studies. He was named *externe* of the Faculty of Medicine hospitals following a competitive application in 1843, *interne* in 1844, and *interne laureate*, with a 1-year extension in 1847. He competed successfully to receive the positions of Anatomy Assistant of the Faculty of Medicine in 1846 and Prosector of Anatomy to the Faculty in 1848. His mentors included such renowned individuals as François Leuret at the Bicêtre, Philippe Ricord at the Hôpital du Midi, Langier at the Hôpital Beaujon, Pierre Nicolas Gerdy at the Faculty of Medicine (Paris), and Philippe-Frédéric Blandin at the Hôtel-Dieu.

Major Appointments

- Following the completion of his medical studies, Broca served as a lecturer at the Faculty of Medicine in Paris. He lectured on topics in anatomy and surgery until a formal competition for a professorship at the Faculty of Medicine opened in 1853. That year, at the age of 29, Broca successfully competed to achieve the distinction of Professor Agrégé and Chirurgien du Bureau Central (also known as Chirurgien des Hôpitaux or Surgeon of the Hospitals) at the Faculty of Medicine. In 1867, he was selected to chair the *pathologie externe*. Broca received the distinction of a professor of clinical surgery in 1868. That same year, he was elected a member of the Académie de Médicine, and resigned his chair of the *pathologie externe* in order to accept the chair of Clinical Surgery. He held this position until his death. During his tenure as the chair of Clinical Surgery, Broca worked in several Parisian hospitals, including the Hôpital St. Antoine, the Pitié, the Hôtel des Cliniques, and the Hôpital Necker.

Major Honors and Awards

- Broca became widely recognized throughout France for his work. In 1865, he was elected president of the Paris Société de Chirurgie. In 1868, he was appointed to the Académie de Médicine and named a member of the French Legion of Honor. He was elected to a permanent seat in the French Senate in 1880, just before his death. Broca also received many posthumous honors, including the eponymous naming of Broca's area, Broca's aphasia, and the diagonal band of Broca.

Landmark Clinical, Scientific, and Professional Contributions

Early Career

- Although Broca is best known for his work on speech and the localization of brain functions, he first established his reputation as a physician and scientist using the microscope to study diseases (Finger, 2004). He described in detail the histology of articular cartilage (the type of cartilage that covers the end of bones), the histology of Rickets, and demonstrated that muscular dystrophy is primarily a disease of the muscles. Broca was among the first to use a microscope to show that cancer cells can penetrate the venous and lymphatic systems as they metastasize. In the early 1850s, he performed the first experiment in Europe using hypnotism as surgical anesthesia.
- Broca was a prolific writer. He wrote several medical classics early in his career, including an extensive treatise on brain aneurysms, *Des aneurysms et de leur traitment*, published in 1856 and a memoir on cancer, *Memoir sur l'anatomie pathologique du cancer*, published in 1853.

Speech and Localization of Function

- Broca's most well-known observations were made in 1861 and the several years that followed. During that time, there was considerable fervor regarding the plausibility of cortical localization of functions, as Franz Joseph Gall's early nineteenth-century phrenology had fallen out of favor. Gall, based on his observations of skull shape, placed the ability to speak and recall words in the inferior aspect of the frontal lobes. Jean Baptiste Bouillaud followed Gall's localizationist views and hypothesized that the anterior lobes of the brain contained a center for speech production. Bouillaud even offered a prize to the first individual who reported a case with loss of speech without lesion to the frontal lobes.
- In February 1861 at a meeting of the Société d'Anthropologie de Paris, a heated debate ensued when Pierre Gratiolet, another well-respected scientist of the time, proposed total brain volume as a meaningful correlate of intelligence, and indicated that the functions of all parts of the brain were essentially identical. These views were vehemently opposed by Bouillaud and his student and son-in-law Simon Alexandre Ernest Aubertin. Broca was present during these debates as the secretary of the Société d'Anthropologie. As he considered the views presented, Broca eventually sided with Bouillaud and Aubertin and became less willing to accept the idea that all parts of the cerebral hemispheres function in the same way (Finger, 2000).
- On April 12, 1861, a Monsieur Leborgne was admitted to Broca's surgical service at the Bicêtre for cellulitis and gangrene of the right leg. Affected by epilepsy since childhood, Leborgne developed considerable limitations in his ability to speak along with right hemiparesis at age 30. He could comprehend speech and communicate using pantomime, but his speech output was limited to the monosyllabic phrase "tan," which became his nickname.

- Leborgne died due to complications from the cellulitis and gangrene on April 17, 1861. Broca quickly completed an autopsy and presented his preliminary findings at the meeting of the Société d'Anthropologie de Paris the following day. He reported atrophy of both hemispheres of Leborgne's brain, with extensive softening of left-frontal areas originating from the third left-frontal convolution. He further examined Leborgne's brain specimen and presented his in-depth findings at the Société in August 1861. His presentation included a description of Leborgne's epilepsy and physical difficulties, including right-sided paralysis and loss of speech, along with a description of the growth of a lesion from the third left-frontal convolution to other areas of the brain. Over time, the area of the third left-frontal convolution became known as "Broca's area" as a result of this case.
- Later in 1861, Broca's surgical service was referred a Monsieur Lelong who had fractured his left femur after a fall. Lelong was an 84-year-old man who had been admitted to the Bicêtre 8 years previously for "senility." In the spring of 1860, Lelong had suddenly collapsed and fallen unconscious. Upon recovery, he was able to produce only four French words: "*oui*," "*non*," "*toujours*," and "*trios*," though his pantomimes were nearly always correct (Lee, 1981). Lelong lived for only 12 days after he was referred to Broca's service. Following Lelong's death, Broca performed an autopsy and found lesions in the second and third frontal convolutions. Presenting the findings from the case of Lelong to the Société d'Anthropologie de Paris in November 1861, Broca indicated that the findings confirmed those from his study of Leborgne, and hypothesized Lelong's left-frontal lesion was due to an old hemorrhage that had occurred at the time Lelong lost his speech in 1860 (Lee, 1981).
- Broca called the inability to produce language in the context of intact comprehension, as seen in the cases of Leborgne and Lelong, "aphémie" (Armand Trousseau subsequently renamed such disturbances "aphasia" in 1864). Broca published several additional cases of aphémie with lesions to the left hemisphere. For example, in 1863, he published a series of eight cases showing primarily left-frontal lesions with language production deficits. In an 1865 manuscript, Broca firmly asserted that the left hemisphere is the dominant seat for language production.
- Broca's declaration that the left hemisphere is predominantly responsible for language is among the clearest and most dramatic examples of localization of neural functions. Broca continues to receive the primary credit for reporting on the localization of language functions, although there were others who may have preceded him. In 1836, Marc Dax presented a work indicating that disturbances in language production were due to lesions of the left hemisphere. Dax's work remained largely unknown until his son Gustave Dax presented and published his deceased father's work in the years after 1863 (see Buckingham, 2006 and Finger, 2000).

Surgery

Broca focused heavily on the relation between the skull and the brain. In June 1871, he treated a 38-year-old laborer who was kicked in the left-frontal region of the skull by a horse. There was no fracture; nevertheless, the patient showed difficulties with speech production after approximately 1 month. He eventually lost his full ability to express himself through speech and lapsed into a coma. Broca suspected an abscess in the area of the third frontal convolution and performed a craniotomy at this approximate location based on his hypothesis. He successfully drained the abscess, but the patient slipped back into coma after approximately 11 h and died. Autopsy revealed a left-sided, predominantly frontal, meningoencephalitis (Jay, 2002). This surgery based on Broca's findings regarding the localization of speech functions is likely the first practical application of the theory of cortical localization (Finger, 2004; Stone, 1991).

Anthropology

From approximately 1866 until his death, Broca focused the majority of his efforts on the advancement of anthropology. Indeed, due to his interest in anthropology and the remains of early humans, he did not write any papers at all on speech and the brain after 1877 (Finger, 2000). Broca's initial interest in anthropology was piqued after serving on a commission examining excavations in the cemetery of the Celestins in 1847. The discovery of Neanderthal Man in 1856, the publishing of Charles Darwin's controversial ideas in *On the Origin of Species* in 1859, and the subsequent controversy on the origins of man furthered Broca's desire to study anthropology.

Much of Broca's anthropological research focused on the comparative study of skulls and the cranium circumference across ethnic groups. He devised various instruments, standardized techniques and methods to examine the structure and topography of the brain based on measurements from prehistoric craniums. He invented at least 27 instruments to determine the relation between the

brain and skull, including a goniometer (instrument to measure angles), craniograph (instrument used to depict the outline of the skull), and several stereographic instruments (Cowie, 2000).

In 1869, Broca published the first description of the Gibraltar skull. Discovered in 1848, the Gibraltar skull was among the earliest skeletal remains identified as belonging to the early species of *Homo sapiens neanderthlensis*. Broca was also fascinated with the topic of neolithic trephination, the process, whereby a hole is scraped or drilled into the skull. His interest in trephination began in 1867 after he examined an Incan skull with cross-hatched cuts. He hypothesized that the operations were performed to treat "internal maladies" in children (Finger, 2004), and was among the first to speculate that trephination was a therapeutic practice that was survived postoperatively based on the signs of inflammation at the wound margins (Cowie, 2000).

Broca was very active in the study of anthropology during the final years of his life. Indeed, he was known to spend many hours per day in the École d'Anthropologie he founded. During the last 2 decades of his life, Broca published over 240 papers and monographs on anthropological topics (Schiller, 1992), including a five-volume work entitled, *Mémoires d'Anthropologie*, published in 1871.

Short Biography

Pierre Paul Broca was born on June 28, 1824, in Sainte-Foy-la-Grande, a small town near Bordeaux, France. He was raised under the Calvinist Protestant tradition. His maternal grandfather, in addition to serving as mayor of Bordeaux during the French revolution, was a pastor. His mother, Annette Thomas, was the sister of a Protestant minister. His father, Jean Pierre (known as Benjamin) Broca, was a physician who served for several years as a surgeon in the French Army and was present at the Battle of Waterloo (Finger, 2000).

Following his undergraduate education, Broca sought to study engineering at the École Polytechnique in Paris (Schiller, 1992). The death of his only sibling, a sister named Léontine, in 1840, along with pressure from his parents to remain closer to home and follow his father's career path, led to his decision to change his course of study to medicine in 1841.

Broca showed considerable interest in the scientific societies that prevailed in Paris both during his study of medicine and throughout his career. He joined the Société Anatomique (Anatomical Society) in 1847 and the Société de Chirurgie (Surgical Society) in 1849. He also founded several societies, schools, and laboratories. In 1848, he established a society of free-thinking individuals, many of whom were sympathetic to Charles Darwin's controversial theories. He started the anthropology laboratory at the École des Hautes Études in Paris in 1858, the Société d'Anthropologie de Paris, the first-known anthropological Société in the world, in 1859, the Revue d'Anthropologie in 1872, and the École d'Anthropologie in Paris in 1876. Broca held leadership positions in many of these societies, including secretary of the Société Anatomique, general secretary of the Société d'Anthropologie de Paris, president of the Société de Chirurgie, and director of the École d'Anthropologie.

Broca married Lugol Augustine, the daughter of Dr. Jean Guillaume Auguste Lugol, who propagated the use of iodine in the treatment of disease, on July 6, 1857. The couple had three children, one daughter and two sons. Just as Broca followed in his father's footsteps by becoming a physician, his two sons succeeded him as well-respected medical scientists.

Pierre Paul Broca died on July 9, 1880 in Paris at the age of 56. Autopsy showed that all organs were apparently sound, although some have speculated his cause of death to be heart disease (Finger, 2000, 2004). Following his autopsy one of his students remarked, "We shall probably not be far from the truth in attributing the catastrophe to cerebral exhaustion, arising from too protracted a course of severe intellectual exertion" (Memoir of Paul Broca, 1881). He was buried at the Montparnasse Cemetery in Paris. In his life, Broca published over 500 books and articles. His influence on ideas regarding cortical localization, speech, and anthropology endures today. Indeed, many scientists would agree that the foundations of modern neuropsychology and cognitive neuroscience were laid by Pierre Paul Broca (Dronkers et al., 2007).

Cross References

- ▶ Aphasia
- ▶ Broca's Aphasia
- ▶ Localization
- ▶ Speech
- ▶ Tan
- ▶ Wernicke's Aphasia

References and Readings

Buckingham, H. W. (2006). The Marc Dax (1770–1837)/Paul Broca (1824–1880) controversy over priority in science: Left hemisphere specificity for seat of articulate language and for lesions that cause aphemia. *Clinical Linguistics & Phonetics, 20*, 613–619.

Clower, W. T., & Finger, S. (2001). Discovering trepanation: The contributions of Paul Broca. *Neurosurgery, 49*, 1417–1425.
Cowie, S. E. (2000). A place in history: Paul Broca and cerebral localization. *Journal of Investigative Surgery, 13*, 297–298.
Dronkers, N. F., Plaisant, O., Iba-Zizen, M. T., & Cabanis, E. A. (2007). Paul Broca's historic cases: High resolution MR imaging of the brains of Leborgne and Lelong. *Brain, 130*, 1432–1441.
Finger, S. (1994). *Origins of neuroscience.* New York City: Oxford University Press.
Finger, S. (2000). *Minds behind the brain.* New York City: Oxford University Press.
Finger, S. (2004). Paul Broca (1824–1880). *Journal of Neurology, 251*, 769–770.
Greenblatt, S. H. (1970). Hughlings Jackson's first encounter with the work of Paul Broca: The physiological and philosophical background. *Bulletin of the History of Medicine, 44*, 555–570.
Grodzinsky, Y., & Amunts, K. (Eds.). (2006). *Broca's region.* New York City: Oxford University Press.
Hothersall, D. (2004). *History of psychology* (4th ed.). New York City: McGraw-Hill.
Jay, V. (2002). Pierre Paul Broca. *Archives of Pathology and Laboratory Medicine, 126*, 250–251.
Lee, D. A. (1981). Paul Broca and the history of aphasia: Roland P. Mackay Award Essay, 1980. *Neurology, 31*, 600–602.
Memoir of Paul Broca (1881). *The Journal of the Anthropological Institute of Great Britain and Ireland, 10*, 242–261.
Schiller, F. (1983). Paul Broca and the history of aphasia. *Neurology, 33*, 667.
Schiller, F. (1992). *Paul Broca: Founder of French anthropology, explorer of the brain.* New York: Oxford University Press.
Schreider, E. (1966). Brain weight correlations calculated from original results of Paul Broca. *American Journal of Physical Anthropology, 25*, 153–158.
Stone, J. L. (1991). Paul Broca and the first craniotomy based on cerebral localization. *Journal of Neurosurgery, 75*, 154–159.

Broca's Aphasia

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Synonyms

Anterior aphasia; Expressive aphasia; Motor aphasia

Short Description or Definition

It is a type of aphasia that is characterized by speech that is effortful, sparse, and halting, and impaired repetition, with relatively intact language comprehension. The spoken output of individuals with Broca's aphasia often is described as telegraphic, as it contains primarily content words and lacks functors, bound morphemes, and other grammatical elements. Paraphasic errors are also present. Reading and writing performance generally mirrors that of auditory comprehension and oral expression. Some individuals with Broca's aphasia have agrammatism, a lack of grammatical structure in their extemporaneous or repeated output that is often associated with impaired comprehension of grammatical structures. Personality and intelligence are typically intact, and, in general, nonlinguistic cognitive functions are relatively preserved, but this is difficult to test the given role of language in cognitive functions.

Categorization

Broca's Aphasia is a type of aphasia that is characterized by speech that is effortful, sparse, and halting, with impaired repetition and relatively intact language comprehension.

Natural History, Prognostic Factors, and Outcomes

The prognosis for recovery of functional communication in individuals with Broca's aphasia depends on the underlying cause of the aphasia as well as factors such as the size of the lesion and the patient's age, premorbid language skills, and comorbid health conditions. Individuals who initially present with Broca's aphasia often evolve to a clinical profile of anomic aphasia, with relatively good auditory and reading comprehension, and deficits primarily in word-finding and the comprehension and production of complex syntax.

Neuropsychology and Psychology of Broca's Aphasia

Broca's aphasia has been traditionally associated with lesions to Brodmann's areas 44 and 45 in the frontal lobe of the dominant (typically left) hemisphere. Autopsy data and neuroimaging studies, however, have shown both the absence of Broca's aphasia in individuals with lesions in this region and also the reverse (Yang, Zhao, Wang, Chen, & Zhang, 2008). The debate about the localization of Broca's aphasia is part of a larger theoretical discussion of the modularity of language and other cognitive functions (for the treatment implications of this debate, see discussion in Basso & Marangolo, 2000).

Depression is a common psychological consequence of aphasia, and is significantly more common after anterior left-hemisphere lesions (including those associated with nonfluent aphasia) than lesions in other areas (Carson et al., 2000). The most commonly used tools for evaluation of depression rely heavily on language processes and thus have limited utility for individuals with aphasia (Turner-Stokes & Hassan, 2002). Scales designed to limit verbal demands, such as the Cornell Depression Scale (Alexopoulos, Abrams, Young, & Shamoian, 1988), have been used in studies of aphasia but have not been validated for this population (Townend, Brady, & McLaughlan, 2007). A systematic review of measures of depression in aphasia (Townend et al., 2007) indicated that adaptation of existing scales and use of other informants were common approaches to the diagnosis of depression in individuals with aphasia, and recommended collaboration between mental health and language experts in the diagnostic process.

Evaluation

Aphasia is typically evaluated using a combination of standardized language tests and careful observation of extemporaneous communication. Assessment of cognitive functions such as attention, memory, and executive functions is challenging in this group, given both the verbal demands inherent in the structure of most neuropsychological tests and the complex interplay of language and other cognitive functions. Cognitive tests considered to have relatively low language demands (e.g., the Cognitive Linguistic Quick Test, Helm-Estabrooks, 2001; or Raven's Standard Progressive Matrices, Raven 1938, included in the Western Aphasia Battery) are sometimes used to test cognitive abilities other than language, with the caveat that language impairments are likely to influence performance on these tests as well (Beeson, Bayles, Rubens, & Kaszniak, 1993).

The specific tests and measures used depend on the goals of the assessment (e.g., diagnosis vs. prediction of functional performance vs. treatment planning), the time post-onset (e.g., comprehensive test batteries are not appropriate in the context of acute stroke), and the patient's clinical presentation. As lesions typically associated with Broca's aphasia affect motor structures in the frontal lobe, many patients with Broca's aphasia also have apraxia of speech and hemiplegia or hemiparesis, which poses a particular challenge in assessment of language and other cognitive functions.

Treatment

There is a wide variety of validated treatment techniques for nonfluent aphasia, particularly for Broca's aphasia. These range from traditional stimulation-type therapies, which have been the staple of aphasia therapy since the second world war, to current treatments such as conversational script training (Manheim, Halper, & Cherney, 2009), constraint-induced aphasia therapy (Cherney, Patterson, Raymer, Frymark, & Schooling, 2008), training of communication partners (Kagan, Black, Duchan, Mackie, & Square, 2001), and direct training of underlying grammatical structures (Thompson & Shapiro, 2005). Most of the treatment literature has focused on individuals with vascular disorders, primarily stroke. For these patients, speech–language therapy interventions have been found to be effective in improving both impairments and functional communication ability even several years after the stroke.

Cross References

- ▶ Aphasia
- ▶ Nonfluent Aphasia
- ▶ Paraphasia
- ▶ Speech–Language Therapy

References and Readings

Academic of Neurologic Coaammunication Disorders and Sciences. *Evidence-based practice guidelines* (*Association*). Retrieved October 1, 2008, from www.ancds.org

Alexopoulos, G. S., Abrams, R. C., Young, R. C., & Shamoian, C. A. (1988). Cornell scale for depression in dementia. *Biological Psychiatry, 23*(3), 271–284.

Basso, A., & Marangolo, P. (2000). Cognitive neuropsychological rehabilitation: The emperor's new clothes? Special issue: Cognitive neuropsychology and language rehabilitation. *Neuropsychological Rehabilitation, 10*(3), 219–229.

Beeson, P. B., Bayles, K. A., Rubens, A. B., & Kaszniak, A. W. (1993). Memory impairment and executive control in individuals with stroke-induced Aphasia. *Brain and Language, 45*, 253–275.

Carson, A. J., MacHale, S., Allen, K., Lawrie, S. M., Dennis, M., House, A., et al. (2000). Depression after stroke and lesion location: A systematic review. *Lancet, 356*(9224), 122–126.

Cherney, L. R., Patterson, J. P., Raymer, A., Frymark, T., & Schooling, T. (2008). Evidence-based systematic review: Effects of intensity of treatment and constraint-induced language therapy for individuals with stroke-induced Aphasia. *Journal of Speech, Language, and Hearing Research, 51*(5), 1282–1299.

Goodglass, H. (1993). *Understanding aphasia*. San Diego, CA: Academic Press.

Helm-Estabrooks, N. (2001). *Cognitive linguistic quick test* (1st ed.). San Antonio, TX: Psychological Corporation.
Kagan, A., Black, S., Duchan, J., Mackie, N., & Square, P. (2001). Training volunteers as conversation partners using "supported conversation for adults with Aphasia" (SCA): A controlled trial. *Journal of Speech, Language, and Hearing Research, 44*, 624–638.
Manheim, L. M., Halper, A. S., & Cherney, L. (2009). Patient-reported changes in communication after computer-based script training for aphasia. *Archives of Physical Medicine and Rehabilitation, 90*(4), 623–627.
Raven, J. C. (1938). *Progressive matrices: A perceptual test of intelligence.* London: H.K. Lewis.
Thompson, C. K., & Shapiro, L. P. (2005). Treating agrammatic aphasia within a linguistic framework: Treatment of underlying forms. *Aphasiology, 19*(10/11), 1021–1036.
Townend, E., Brady, M., & McLaughlan, K. (2007). A systematic evaluation of the adaptation of depression diagnostic methods for stroke survivors who have aphasia. *Stroke, 38*(11), 3076–3083.
Turner-Stokes, L., & Hassan, N. (2002). Depression after stroke: A review of the evidence base to inform the development of an integrated care pathway. Part 1: Diagnosis, frequency and impact. *Clinical Rehabilitation, 16*(3), 231–247.
Yang, Z. H., Zhao, X. Q., Wang, C. X., Chen, H. Y., Zhang, Y. M. (2008). Neuroanatomic correlation of the post-stroke aphasias studied with imaging. *Neurological Research, 30*(4), 356–360.

Brodmann's Area 17

► Visual Cortex

Brodmann's Areas of the Cortex

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Definition

Brodmann's areas of the cortex refer to 52 regions of the cerebral cortex that were identified in 1909 by German Neurologist, Korbinian Brodmann, based on cytoarchitectonic (cell size, spacing or packing density, and lamination) differences. Brodmann's areas are typically shown on a map of the brain surface, but each region is continued through the depth of cerebral cortex. These regions were originally identified based on Nissl-stained sections of human brain; however, Brodmann believed that they applied to all mammals.

Current Knowledge

In some cases, the boundary identified by Brodmann is also a functional boundary. For instance, primary visual cortex is contained in Brodmann's area 17. Brodmann's area 18 is considered to be higher-order visual cortex. Somatosensory functions are associated with Brodmann's areas 3, 1, and 2, with part of area 3 being recognized as primary somatosensory cortex. Brodmann's areas 41 and 42 are associated with audition (hearing). Primary motor cortex (the output for motor commands) is associated with Brodmann's area 4, while premotor cortex (where the decision to move likely arises) is found in Brodmann's area 6.

A different interpretation of cytoarchitechtonic regions (107 areas) was published by Constantin von Economo and Georg N. Koskinas in 1925.

Cross References

► Auditory Cortex
► Cerebral Cortex
► Neocortex
► Somatosensory Cortex
► Visual Cortex

References and Readings

Brodmann, K. (2005). *Brodmann's: Localisation in the cerebral cortex.* (L. J. Carey Trans.). Berlin: Springer.
Falk, D., & Gibson, K. R. (Eds.). (2008). *Evolutionary anatomy of the primate cerebral cortex.* Cambridge: Cambridge University Press.

Bromocriptine

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Definition

Bromocriptine is one of the group of medicines classified as ergot alkaloids. Bromocriptine acts to block the release of prolactin which is produced by the pituitary gland. Bromocriptine is used to treat a variety of medical conditions including problems with menstruation, infertility,

Parkinson's disease, neuroleptic malignant syndrome, and pituitary adenomas. When used in conjunction with diet, bromocriptine can also be used to treat type 2 diabetes.

References and Readings

Ropper, A. H., & Samuels, M. A. (2009). *Adams and Victor principles of neurology* (9th ed.). New York: McGraw Hill.

Brown–Séquard Syndrome

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Synonyms

Hemisection of spinal cord

Definition

Brown–Sequard syndrome is a neurological condition in which, as a result of a lesion affecting one half of the spinal cord, there is paralysis and loss of proprioception, vibration, and fine tactile discrimination on one side of the body and loss of pain and temperature on the other.

Current Knowledge

To fully appreciate this syndrome, it is helpful to understand some basic anatomy of the spinal cord. Recall that the *lateral corticospinal* tract, which carries voluntary motor impulses originating in the cortex, descends in the lateral portion of the cord after having crossed the midline (decussated) in the medulla. On the sensory side, fibers that mediate position sense (*proprioception*), fine tactual discrimination (*stereognosis*), and vibration enter the cord through the dorsal nerve roots and, without synapsing, travel up the same side of the cord from which they enter (via the posterior columns or *lemniscal system*) until they synapse in the medulla. By contrast, those dorsal root (sensory) fibers that carry information regarding pain and temperature synapse in the dorsal horn of the cord on the same side in which they entered. From there, their second-order neurons cross the midline of the cord in the ventral while commissure and then ascend contralaterally as the ventral and lateral spinothalamic tracts (*anterolateral system*).

Thus, a lesion that transects one-half of the spinal cord will cause motor symptoms on the same side of the body as a result of severing the lateral corticospinal tract on that side. This will result in residual upper motor neuron type deficits below the level of the lesion (e.g., spastic paralysis, hyperreflexia, clonus, loss of superficial reflexes, and a positive Babinski). As the lesion also affects the ventral horns, lower motor neuron signs (flaccid paralysis, severe atrophy, hyporeflexia, and fasciculations) are potentially discernable at the level of the lesion. Because the posterior columns are affected, the individual will also demonstrate a loss of proprioception, fine tactual discrimination, and vibratory sense below the level of the lesion on that same side. Finally, as a result of a disruption of the ascending spinothalamic tracts, there will be a loss of pain and temperature. However, because these latter tracts represent sensory information that has crossed over from the opposite side of the cord, the loss of pain and temperature will be contralateral to the lesion (and one of the sides opposite to the motor and posterior column symptoms). Because the fibers carrying signals for pain and temperature ascend and descend a couple of spinal segments before decussating, the level of loss of pain and temperature will be slightly below that of proprioception and stereognosis.

A Brown–Séquard syndrome usually results from a penetrating injury as might be found in a knife or bullet wound. Because such wounds lack anatomical precision, exactly one half of the cord is rarely severed, but the term is applied if the clinical picture generally matches that which was described above.

Cross References

▶ Anterolateral System
▶ Lateral Corticospinal Tracts
▶ Posterior Columns

Bruise

▶ Cortical Contusion
▶ Hematoma

Brunel Balance Assessment

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Synonyms

BBA

Definition

The Brunel Balance Assessment (BBA) clinical assessment tool is designed as an outcome measure to assess balance before and after stroke physiotherapy interventions. It consists of 12 items that progress from easy to difficult in a hierarchical manner to form an ordinal scale. The easiest item is "static sitting balance with upper limb support"; a mid-range item is "dynamic standing balance"; the hardest item is "advanced change of the base of support." Each item is assessed by evaluating performance on a specific task with task-specific criteria for succeeding; for example, dynamic standing balance is assessed by evaluating the distance that the individual can reach beyond arm's length. Success at this task is set at a minimum reach of 7 cm. The BBA items are arranged into three subscales which can be used individually: sitting; standing; and stepping balance.

Current Knowledge

Homogeneity of the scale: All items have item-total correlations of more than 0.20. Cronbach's alpha, a measure of internal consistency, was 0.93 (Tyson & DeSouza, 2004). Therefore, the scale was deemed homogeneous and internally consistent.

Reliability: Test-retest reliability was assessed using observations on consecutive days, while inter-rater reliability was assessed using two independent raters. There was 100% agreement for both forms of reliability (Tyson & DeSouza, 2004).

Validity: Criterion validity was assessed by comparison with the sitting section of the Motor Assessment Scale (sitting balance), the Berg Balance Test (standing balance), and the Rivermead Mobility Index (stepping balance/functional mobility). The correlation coefficients were 0.83 (Motor Assessment Scale), 0.97 (Berg Balance Test), and 0.95 (Rivermead Mobility Index). Predictive validity was assessed by comparing BBA scores during in-hospital admission within 2–4 weeks after stroke to scores on the Barthel Index and Rivermead Mobility Index at 3 months post-stroke in 102 individuals (Tyson, Hanley, Chillala, Selley, & Tallis, 2007). Individuals who had limited sitting balance during hospitalization showed little recovery of functional mobility independence. Individuals who were able to walk (with or without assistive equipment) during hospitalization were mostly independent at 3 months post-stroke in transfers, walking, and stairs. For those individuals with limited standing balance during hospitalization, the majority regained the ability to walk, conduct transfers, and navigate stairs, although more of these individuals required assistance.

Cross References

- ▶ Barthel Index
- ▶ Berg Balance Scale
- ▶ Motor Assessment Scale
- ▶ Rivermead Mobility Index

References and Readings

Tyson, S. F., & Connell, L. A. (2009). How to measure balance in clinical practice. A systematic review of the psychometrics and clinical utility of measures of balance activity for neurological conditions. *Clinical Rehabilitation, 23*, 824–840.

Tyson, S. F., & DeSouza, L. H. (2004). Development of the Brunel Balance Assessment: A new measure of balance disability post stroke. *Clinical Rehabilitation, 18*, 801–810.

Tyson, S. F., Hanley, M., Chillala, J., Selley, A. B., & Tallis, R. C. (2007). The relationship between balance, disability, and recovery after stroke: Predictive validity of the Brunel Balance Assessment. *Neurorehabilitation and Neural Repair, 21*, 341–346.

BSI

- ▶ Brief Symptom Inventory

BSI-18

- ▶ Brief Symptom Inventory

BSID-III

- ▶ Bayley Scales of Infant and Toddler Development

Bucco-facial Apraxia

► Apraxia

Building-related Illness

► Sick Building Syndrome

Bupropion

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Metarie, LA, USA

Generic Name

Bupropion

Brand Name

Wellbutrin, Wellbutrin SR, Wellbutrin XL, Zyban

Class

Antidepressant

Proposed Mechanism(s) of Action

Increases norepinephrine/noradrenaline and dopamine, blocks norepinephrine reuptake pump, may increase dopamine neurotransmission in the frontal cortex, blocks dopamine reuptake pump.

Indication

Major depressive disorder, nicotine addiction

Off Label Use

Bipolar-disorder, ADHD

Side Effects

Serious

Seizures, hypomania, induction of mania, and activation of suicidal ideation (controversial)

Common

Dry mouth, constipation, nausea, weight loss, insomnia, dizziness, headache, agitation, tremor, abdominal pain, tinnitus, and sweating

References and Readings

Physicians' Desk Reference (62nd ed.). (2007). Montvale, NJ: Thomson PDR.
Stahl, S. M. (2007). *Essential psychopharmacology: the prescriber's guide* (2nd ed.). New York, NY: Cambridge University Press.

Additional Information

Drug Interaction Effects: http://www.drugs.com/drug_interactions.html
Drug Molecule Images: http://www.worldofmolecules.com/drugs/
Free Drug Online and PDA Software: www.epocrates.com
Gene-Based Estimate of Drug interactions: http://mhc.daytondcs.com:8080/cgi bin/ddiD4?ver=4&task=getDrugList
Pill Identification: http://www.drugs.com/pill_identification.html

Burden of Proof

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Baltimore, MD, USA

Synonyms

Standards of proof

Definition

This refers to the duty to provide evidence for allegations raised in the context of legal action. The Standard of Proof is the degree of proof needed in a legal action to persuade the court (e.g., judge or jury) that a given allegation is

indeed founded or true. There are three main types of standards of proof: beyond a reasonable doubt, clear and convincing evidence, and a preponderance of the evidence. Artificial percentages have been associated with each of these standards of proof with beyond reasonable doubt coinciding with 90–95% certainty, clear and convincing evidence 75%, and a preponderance of the evidence associated with just over 50%. Each of these standards is used during different inquiries in criminal procedure (e.g., insanity defense, competency to stand trial, and competency to be executed) and there are other standards used by appellate courts when reviewing trial court records. Under the current *Mental Penal Code* Standard for Insanity, "The defendant has the burden of providing the defense of insanity by clear and convincing evidence."

Cross References

- ▶ Beyond a Reasonable Doubt
- ▶ Clear and Convincing Evidence
- ▶ Preponderance of the Evidence

References and Readings

Denney, R. L. (2005). Criminal forensic neuropsychology and assessment of competency. In G. Larrabee (Ed.), *Forensic neuropsychology: A scientific approach.* New York: Oxford University Press.

Melton, G. B., Petrila, J., Poythress, N. G., & Slobogin, C. (2007). *Psychological evaluations for the courts: A handbook for mental health professionals and lawyers* (3rd ed.). New York: Guilford Press.

Buschke Selective Reminding Test

- ▶ Selective Reminding Test

Buspirone

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Children's Hospital of New Orleans
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Generic Name

Buspirone

Brand Name

Buspar

Class

Anxiolytic

Proposed Mechanism(s) of Action

Serotonin 1A partial agonist, thus diminishing the overall serotonergic activity at those receptor sites

Indication

Anxiety disorders

Off Label Use

Mixed anxiety and depression, treatment resistant depression

Side Effects

Serious

Very rare cardiac symptoms

Common

Dizziness, sedation, nervousness, and nausea

References and Readings

Physicians' Desk Reference (62nd ed.). (2007). Montvale, NJ: Thomson PDR.

Stahl, S. M. (2007). *Essential psychopharmacology: the prescriber's guide* (2nd ed.). New York, NY: Cambridge University Press.

Additional Information

Drug Interaction Effects: http://www.drugs.com/drug_interactions.html
Drug Molecule Images: http://www.worldofmolecules.com/drugs/

Free Drug Online and PDA Software: www.epocrates.com
Gene-Based Estimate of Drug interactions: http://mhc.daytondcs.com:8080/cgi bin/ddiD4?ver=4&task=getDrugList
Pill Identification: http://www.drugs.com/pill_identification.html

Butters, Nelson (1937–1995)

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University of Pittsburgh School of Medicine
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Major Appointments

- NIMH Postdoctoral Research Fellow at the Neuropsychology Section of NIMH, Bethesda, MD (1964–1966)
- Instructor, College of General Studies, George Washington University (1965–1966)
- Assistant Professor, Ohio State University (1966–1967)
- Lecturer in Psychology, Antioch College (1966–1967)
- Lecturer in Psychology, Wellesley College (1967–1968)
- Lecturer in Psychology, University of Massachusetts (Boston) (1967–1983)
- Research Career Scientist, Boston Veterans Administration Medical center (1967–1983)
- Professor of Neurology (Neuropsychology), Boston University School of Medicine (1967–1983)
- Senior Lecturer, Northeastern University, University College (1967–1983)
- Affiliate Professor of Psychology, Clark University (1973–1983)
- Chief, Psychology Service, San Diego Department of Veteran Affairs Medical Center (1983–1995)
- Professor of Psychiatry, University of California School of Medicine (San Diego) (1983–1995)

Major Honors and Awards

- Phi Beta Kappa, Summa Cum Laude, A.B. with Honors
- NIH Predoctoral Research Fellowship (1961–1964)
- NIMH Postdoctoral Research Fellowship (1964–1966)
- Member of the Collegium of the Distinguished Alumni of the College of Liberal Arts of Boston University (elected 1974)
- Fellow, American Association for the Advancement of Science
- American Psychological Association (Fellow Divisions 3, 6, 40; President of Division 40 1982–1983; Representative to APA Council 1990–1992)
- International Neuropsychological Society (Secretary-Treasurer 1974–1977; Board of Governors 1978–1981; Treasurer 1980–1983; President, 1984–1985)
- National Academy of Neuropsychology (Fellow; President 1992–1993)
- American Psychological Society (Fellow, 1988)
- American Board of Clinical Neuropsychology (Founding Fellow, Vice-President 1991–1993)
- Distinguished Clinical Neuropsychologist Award from the National Academy of Neuropsychology (1991)
- Meritorious Service Award from the Department of Veteran Affairs (1993)
- Distinguished Service Award from the American Board of Professional Psychology (1993)

Landmark Clinical, Scientific, and Professional Contributions

- Nelson Butters authored or coauthored over 200 peer-reviewed scientific articles, 60 invited monographs and book chapters and reams of abstracts. He coedited or coauthored six books and delivered a multitude of invited lectures and presentations. Butters expended much professional energy demonstrating the existence of distinct dissociations among cognitive functions, especially memory, within and between patients with various forms of cerebral dysfunction. He was unusually successful (perhaps uniquely so) at integrating neuroanatomy and cognitive theory with applied neuropsychology. As a result, one of the most distinguishing features of his career was that his work was very highly regarded by cognitive and clinical researchers as well as practicing clinicians. He was especially proud of this "cross-professional" appeal.
- Butters' earliest work was conducted in primates. Two tests in particular could reveal memory loss in monkeys – the delayed response (DR) and delayed alternation (DA) tasks. For both DR and DA, the animals had to hold information in memory for a short time before making a response to obtain a reward. Lesions in the prefrontal cortex, in particular along the sulcus principalis, severely disrupted the monkeys' ability to either perform or learn the tasks. In 1969, Butters, in collaboration with Deepak Pandya, demonstrated that the middle third of the sulcus principalis was the critical region associated with impairments of DR and DA. In 1971, they described functional differentiation along the axis of the sulcus principalis; the middle third differed from the anterior and posterior portions with regard to their efferent (outgoing)

projections of the prefrontal cortex. In 1972, with their student Gary Van Hoesen, they reported on the afferent (incoming) projections to the entorhinal cortex around the hippocampus – another brain region critical for memory functions.

- At the same time, Butters was working closely with Donald Stein and Jeffrey Rosen on recovery of function following frontal lobe lesions in nonhuman primates. They showed that if one removed sections of the frontal lobes in monkeys in serial fashion, that is, over a series of operations, the resulting deficit was less than that exhibited by monkeys who experienced the same lesion, in one step. They concluded that these results demonstrated at least partial recovery of function. This finding in adult monkeys was important because at the time, it was believed that if a brain lesion occurred after 12 months of age (in the monkey), recovery was not possible. Patricia Goldman was showing that infant macaques with brain lesions could fully recover some functions, but not others – the data from the Rosen, Stein, and Butters series shed light on the question of whether, and by how much, older animals (and by extension, humans) could recover function after a brain lesion.
- One of his earliest human studies (published with Melvin Barton in 1970) on the role of the frontal and parietal lobes in concrete operations, shaped much of Butters' philosophy of neuropsychology and laid the groundwork for his career examining the brain structures that mediate abnormal cognition. They took concepts and methods that were popular in human cognition at that time and used them to examine the role of various brain structures in these cognitive processes. For example, humans with damage to the parietal lobes have difficulty reversing – or changing – their behavior. That is, once taught a rule, they have difficulty deviating from that rule. What was unclear at the time was whether this deficit was a fundamental deficit in reversal or a consequence of a deficit in retention – the ability to remember the old rule or learn the new one. The results of these experiments not only informed investigators on the nature of the behavioral and cognitive changes that follow focal brain damage, but lent insight into the cognitive processes themselves.
- Butters made exemplary use of what Hans-Lukas Teuber referred to as the "natural fracture lines of behavior." That is, when a patient suffers a brain injury, behavior and cognition fail neither completely nor in a random manner. Rather, the breakdown occurs at points where different cognitive processes intersect. By comparing and contrasting the nature and extent of changes in cognition across patients with different forms of brain damage, we advance our understanding of the neuroanatomical basis of the cognitive process in question, the organization of the process itself, and how these affect the individual patient. This concept became a theme – sometimes explicit, sometimes implicit – that was woven into the fabric of Butters' research career.
- An early series of studies in the 1970's with Ina Samuels exemplified this approach. Together they systematically examined visual and auditory short-term memory in individuals with a variety of lesions. In the early 1970s cognitive psychologists were debating the relative merits of serial versus parallel processing in normal human memory. Butters' work with Laird Cermak on patients with amnesic disorders was among the first to examine this and other central topics of cognitive psychological research. The Butters and Cermak collaboration was particularly fruitful because it directly influenced the development of models of normal human memory – particularly the notion that memory is neither a serial nor parallel process, but rather that the *type* or *intensity* of the processing was what was critical (i.e., "levels of processing" argument espoused by Fergus Craik).
- While in Boston, Butters and his colleagues made great strides in elucidating the neuropsychological effects of alcoholism. The papers that emanated from studies of chronic, non-amnestic alcoholics (in collaboration with Christopher Ryan, James Becker, Kathleen Montgomery, and Barbara Jones) had a lasting influence on the way that the neuropsychology of alcoholism was viewed and studied. Butters' work also focused on the amnesic syndrome associated with chronic, severe alcoholism. With Laird Cermak, his systematic series of studies focused on the role of interference and encoding in the short-term memory defects of patients with alcoholic Korsakoff's syndrome. It is noteworthy that Butters developed personal relationships with many of these patients, venturing out to their homes (sometimes state hospitals or nursing homes) and repeatedly reevaluating them for different projects. This work was important not only because of what it revealed about both normal and pathological memory, but also because it clearly demonstrated that a long-term consequence of alcoholism can be an amnestic disorder (a matter that was hotly debated at the time). Perhaps the most enduring legacy of Butters and Cermak's work from this period was the 1980 publication of *Alcoholic Korsakoff's Syndrome: An Information Processing Approach to Amnesia*. The title was not an accident – they

wanted to emphasize the role of neuropsychology in analyzing information processing, and how this research informed our models of normal memory and memory systems. The publication of this text marked the beginning of the modern era of "cognitive neuroscience."

- Another trend in Butters' research during the middle-to-late 1970s was prompted by his desire to better understand retrograde amnesia, which had not yet been studied in any systematic way, due in part, to the lack of suitable test instruments. He collaborated with Marilyn Albert, who took on the task of developing the Boston Retrograde Amnesia Battery, which became the first carefully validated and well-normed retrograde memory battery applicable to individuals of various ages. This productive collaboration, which later included Jason Brandt and Donald Stuss, firmly established that the retrograde memory impairment exhibited by Korsakoff's patients had a clear temporal gradient, suggesting that the neural substrates of memory change with the passage of time. That is, over time, episodic memories mediated by the hippocampal circuitry are actively recalled, thereby losing their spatial and temporal markers, becoming part of semantic memory, stored elsewhere (likely the temporal–parietal association cortices).
- During his last years in Boston, Butters devoted considerable time to studying the cognitive effects of Huntington's disease (HD), demonstrating a functional dissociation between the locus of the lesions in HD (the basal ganglia) and those found in Korsakoff's syndrome (the limbic system). This research predated the identification of the HD gene and development of a test to determine whether the offspring of HD patients were destined to develop the disease. As part of the "Center Without Walls," Butters worked with people "at risk" for HD (i.e., who had a parent with the disease) to determine whether measures of cognition administered earlier in life could predict those who would ultimately develop the disease.
- In 1983 Butters accepted an offer to join the Department of Psychiatry at the University of California-San Diego (UCSD), where he relished the opportunity to develop a clinical service with neuropsychologists who conducted research as well as provided clinical care. In addition to developing a first-rate psychology service, Butters maintained his prolific research publication rate. Soon after arriving in San Diego, UCSD was designated an Alzheimer's Disease Research Center, which provided Butters with the opportunity to expand his research on memory. His studies now included Alzheimer's disease, which he considered the prototypical "cortical" dementia. In San Diego his work with David Salmon, William Heindel, William Beatty, Munro Cullum, Eric Granholm, Alexander Troster, Agnes Chan, Andreas Monsch, and a host of other students and colleagues focused on three main areas, yielding a significant scientific contribution in each.
- Butters' work with David Salmon and their colleagues demonstrated that the memory disorders of cortical and subcortical dementias are dissociable, with Alzheimer's disease characterized by poor consolidation and rapid forgetting due to limbic damage and Huntington's disease by poor retrieval associated with fronto-striatal dysfunction. Butters' work with trainee Mark Bondi and others in his research group was among the first to show that the neuropsychological characteristics of very early Alzheimer's disease differed from the benign cognitive changes of normal aging and could therefore be used for the early and even pre-clinical detection of the disease, presaging similar findings in HD. Finally, researchers were beginning to investigate the phenomenon of implicit memory, and Butters' work with William Heindel, David Salmon, Jane Paulsen, and others showed that various forms of implicit memory could be dissociated in Alzheimer's disease, Huntington's disease, and Parkinson's disease. These studies were the first to dissociate priming and procedural learning in the brain, showing that procedural learning deficits were associated with basal ganglia dysfunction while priming deficits were linked with neocortical damage that occurs in Alzheimer's disease. Any one of these contributions would have been impressive; together they represent a truly remarkable accomplishment.
- In 1986 the American Psychological Association (APA) acquired the journal *Neuropsychology* and Butters was appointed Editor. APA tradition allows new editors to choose the colors of the journal's binding – and Butters chose a white background with (Boston) Celtic green for the text (he was a lifelong fan of his hometown basketball team). Butters' goal for the journal was that it should become one of the journals of record for basic, applied, *and* clinical research in neuropsychology. This will be another of his lasting legacies.
- Most important to Butters were his students – his academic children (and in some cases grandchildren) were almost as important to him as his biologic children, Meryl, Paul, and Lisa. His attraction was international (e.g., John Hodges, Narinder Kapur, Matti

Laine), and his mentees continue to lead the field (e. g., Kathie Welsh-Bohmer, Terry Jernigan).

- A narrative of Butters' contributions to neuropsychology would be woefully incomplete without a personal description. He possessed tremendous internal drive, and held both his students and colleagues to the high standards he applied to himself. Every Butters' student carries distinct memories of the red ink and blunt reviews accompanying their manuscript drafts. His academic rigor notwithstanding, Butters was universally known as an involved, supportive mentor and colleague. His role extended to that of job-broker and he devoted an inordinate amount of time and energy to assisting and advising a vast array of students and colleagues about both professional and personal matters. He was described as being biologically incapable of tolerating an unhappy "student" (a term that he applied quite broadly); he had to help make things right (even if the student did not immediately appreciate his wisdom). In his later years he took tremendous pleasure in referring to himself as the "Godfather of Neuropsychology."
- Butters was blunt, brash, and famously irreverent. While his lifestyle was decidedly mainstream, he reveled in characterizing himself as something of a rebel. He was unendingly curious about those who walked to the beat of a different drummer. His personal role models included Marlon Brando, Lenny Bruce, and Woody Allen. He had a keen sense for and appreciation of the absurd and ironic and found humor in the darker side of life, though he never took himself too seriously. On more than one occasion he was heard saying: "If I hadn't become a neuropsychologist, I would've been a comedian." At every APA and INS meeting social hour, Butters could be found, beer in hand, listening to and recounting hilarious stories about friends, colleagues, and family. Most of the time, he played a leading role in these comedic tales. In sum, Butters' rare combination of intellectual creativity, drive, irreverence, and appreciation of satire allowed him to make his mark both in the field of neuropsychology and in the hearts of his contemporaries.

Short Biography

Nelson Butters was born on May 7, 1937, in Boston, MA. He initially attended public school in Brookline, MA, and then Worcester Academy, a private boarding school, graduating first in his class. He was an accomplished athlete and scholar. A concussion sustained playing football left him with lifelong anosmia, an irony not lost on him when he later published articles on the importance of assessing olfaction in brain-damaged patients. In addition to his obvious academic talent, he took great pride in his carefully crafted image as a motor scooter-riding "bad boy," emulating James Dean and Marlon Brando. Later in life, he took great pleasure in recounting tales of his friendship with his classmate, the late social and political activist Abbie Hoffman. Butters particularly enjoyed retelling stories of "drag racing" against Hoffman from Worcester to Boston, MA, and attending wild parties at Hoffman's home. Somehow surviving high school, he went on to attend the University of Chicago and Boston University.

Butters was at first unsure of his professional goals. His parents had pushed for him to become a physician or lawyer, but neither profession held much appeal. Public speaking came rather easily to him, and he was excited by the idea of being a positive influence in the lives of college students. Therefore, during the second or third year of college he decided to become a psychology teacher.

Butters entered the doctoral program in psychology at Clark University in the fall of 1960. At the time Clark had one of the leading psychology programs in the world. He was awarded a Woodrow Wilson Fellowship for his first year of graduate school because of his commitment to college teaching. During his second or third year in graduate school, he ran across a research article by Mortimer Mishkin and H. Enger Rosvold about delayed alternation, reversal learning, and the frontal lobes in monkeys. Butters became fascinated with the concept that one could examine the neurological structures that underlie cognitive and psychological processes. At the time he was particularly interested in what went on in the brain and what brain structures played a part in the development of concrete or formal operations in the thinking processes of children.

At the end of his third year of graduate school, Butters wrote to Hal Rosvold, a researcher at the National Institutes of Health (NIH), asking whether he would consider taking him as a postdoctoral fellow. A visit to Rosvold's lab led to a postdoctoral fellowship with Rosvold and Mishkin from 1964 to 1966. His work at the NIH focused on the roles of the septal nuclei, basal forebrain, and caudate nucleus in reversal learning and delayed alternation performance. Butters was fulfilling his plan to spend "a couple of years" studying physiological psychology and perhaps do "a few studies" on the neurological bases of cognition before resuming the path toward his teaching career.

Butters resumed his teaching path in 1966 by taking teaching positions at Wright State University and Antioch

College in Ohio. However, the "few studies" he had conducted while at NIH caught the attention of Harold Goodglass and Norman Geschwind of the Boston Veteran's Administration Hospital and Department of Neurology of the Boston University School of Medicine. Moreover, Butters and Goodglass had become acquainted during the former's years at Clark. The personal relationship, combined with Butters' cutting-edge animal work, led to an invitation from Geschwind and Goodglass to join their burgeoning Aphasia Research Center. Butters eagerly accepted the opportunity to join this exciting group and to return home to Boston. While he initially studied nonhuman primates, he took the opportunity to learn about human neuropsychology from the early pioneers, many of whom resided in Boston. In addition to Geschwind and Goodglass, he reveled in learning from and exchanging ideas with the likes of Edith Kaplan, Marlene Oscar Berman, Howard Gardner, Edgar Zurif, and many others. The unparalleled atmosphere at the Boston VA Hospital led to Butters' first studies in human neuropsychology. During 1967–1970 he conducted both animal and human research and after 1970 his work focused exclusively on human cognition.

In 1983 Butters moved to San Diego, CA, to join the Department of Psychiatry at UCSD, where he continued to flourish. Continuing his research program, Butters also built a psychology service with a particular emphasis on training. He surrounded himself with superb colleagues including, among others, Robert Heaton and Dean Delis.

In early 1993 Butters developed oral motor weakness and by March 1993 it was clear that he had amyotrophic lateral sclerosis. He was 55 years old and at the height of his career. He handled his nearly 3-year battle with ALS in typical Butters fashion, continuing to work and socialize until the week before his death. He took the opportunity to tell his students, colleagues, and family how grateful he was to have had them in his life. When he lost the ability to speak, he used adaptive computer equipment to communicate. The week before he died, he was still telling off-color and self-deprecating jokes by laboriously typing with his big toe, irreverent and irrepressible until the end.

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Cross References

▶ Alzheimer's Disease
▶ Amnesia
▶ Amnestic Disorder
▶ Amnestic Syndrome
▶ Anterograde Amnesia
▶ Dementia
▶ Episodic Memory
▶ Goodglass, Harold (1920–2002)
▶ Implicit Memory
▶ Kaplan, Edith
▶ Korsakoff's Syndrome
▶ Mammillary Bodies
▶ Medial Temporal Lobe
▶ Memory
▶ Memory Impairment
▶ Mishkin, Mortimer (1926–)
▶ Parkinson's Dementia
▶ Parkinson's Disease
▶ Procedural Memory
▶ Retrograde Amnesia
▶ Semantic Memory
▶ Subcortical Dementia
▶ Temporal Lobes
▶ Thalamus

References and Readings

Butters, N. (1996). Memoirs, in Nelson Butters remembered. *APA Division 40 Newsletter, 14*(3), 1, 3–7.

Butters, M., Becker, J. T., & Brandt, J. (1996). A legend in his own time: tribute to Nelson Butters, in Nelson Butters remembered. *APA Division 40 Newsletter, 14*(3), 2, 7–8.

Butters, M. A., & Butters, N. M. (2002). Nelson M. Butters: One step ahead. In A. Y. Stringer, E. L. Cooley, & A. L. Christensen (Eds.), *Pathways to prominence: Reflections of 20th century neuropsychologists*. New York: Psychology Press.

Cermak, L. S. (Ed.). (1994). *Neuropsychological explorations of memory and cognition: Essays in Honor of Nelson Butters*. New York: Plenum.

BVRT

▶ Benton Visual Retention Test