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

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Babcock Story is rarely used today, some current memory batteries incorporate its format (► Wechsler Memory Scale All Versions). 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
- Intelligence Quotient
- Wechsler Memory Scale All Versions

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.

Babinski Reflex

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Synonyms

Long tract sign; Plantar reflex; Upper motor neuron sign

Definition

The Babinski reflex is a component of the neurological exam, used to assess the adequacy of the pyramidal tract (upper motor neuron). This reflex 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 (fanning)
- Flexor (negative or normal): all toes flex and the foot everts
- Indifferent: no response

Current Knowledge

An extensor (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 for evaluation of 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 as the nervous system matures 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 normal flexor response.

Cross-References

- Developmental Delay
- Multiple Sclerosis
- Spinal Cord Injury
- ► Stroke
- ► Traumatic Brain Injury (TBI)

References and Readings

- Babinski, J. (1896). Sur le reflexe cutane plantaire dans certaines affections organiques du systeme nerveux central. Comptes Rendus des Seances de la Societe de Biologie et de Ses Filiales, 48, 207–208.
- Larner, A. J. (2016). A dictionary of neurological signs (4th ed.). Basel: Springer.
- Pearson, K., & Gordon, J. (2000). Spinal reflexes. In E. R. Kandel, J. H. Schwartz, & T. M. Jessell (Eds.), *Principles of neural science* (4th ed., pp. 713–736). New York: McGraw-Hill.

Backwards Masking

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Definition

Backward masking occurs when the perception of a stimulus is attenuated by the rapid presentation of a subsequent stimulus (the "mask"). Within the domains of neuropsychology and psychology, backward masking typically refers to visual phenomena. However, backward masking has been explored in other sensory domains such as ... (may want to list other domains here). In a typical backward masking paradigm, a visual stimulus (such as a letter) is rapidly presented and followed by a mask that encompasses the area of the visual field where the initial stimuli was presented (Breitmeyer and Ogmen 2000). The presentation of the initial stimulus, while rapid, is sufficiently long enough for a non-backward masked presentation to be perceptible. The mechanisms underlying backward masking are an active area of research; however, it is well established that central and likely cortical mechanisms are involved, given the time course of the effect as well as its ability to be produced with dichoptic presentation (stimulus and mask presented to separate eyes).

References and Readings

Breitmeyer, B. G., & Ogmen, H. (2000). Recent models and findings in visual backward masking: a comparison, review, and update. *Perception & Psychophysics*, 62(8), 1572–1595. Retrieved from http://www.ncbi. nlm.nih.gov/pubmed/11140180

Bacterial Ventriculitis

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Synonyms

Intraventricular infection; Intraventricular inflammation; Ventriculomeningitis

Definition

Bacterial ventriculitis refers to inflammation of the cerebral ventricles, typically resulting from intraventricular infection or bacterial infection of cerebral spinal fluid.

Current Knowledge

Bacterial ventriculitis is a potential life-threatening condition that can result from the rupture of a cerebral

abscess, an infection of an external ventriculostomy catheter, an infection of cerebral spinal fluid, and other infectious CNS conditions. Bacterial infection produces an immune response in the lining of the ventricles, resulting in inflammation.

Presenting symptoms can be headaches, dizziness, confusion, photophobia, and neck and upper back pain and nausea and vomiting in children. In infancy, it can cause unrecognized hydrocephalus. Ventriculitis must be confirmed by examination of the cerebrospinal fluid.

Cross-References

- Encephalitis (Viral)
- Meningitis

References and Readings

- Agrawal, A. M., Cincu, R., & Jake, R. (2008). Current concepts and approach to ventriculitis. *Infectious Dis*eases in Clinical Practice, 16(2), 100–104.
- Centers for Disease Control and Prevention (CDC). (2017). CDC/NHSN surveillance definitions for specific types of infections. Retrieved from http://www.cdc.gov/nhsn/ PDFs/pscManual/17pscNosInfDef current.pdf.
- Tabuchi, S., & Kadowaki, M. (2015). Neuroendoscopic surgery for ventriculitis and hydrocephalus after shunt infection and malfunction: Preliminary report of a new strategy. *Asian Journal Endoscopic Surgery*, 8, 180–184. https://doi.org/10.1111/ases.12162.
- Weerakkody, Y., & Gaillard, F. (n.d.). Ventriculitis. Retrieved 9 Dec 2016, from https://radiopaedia.org/ articles/ventriculitis.

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 (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 Syndrome
- 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 7 Jan 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 defi-Individuals with Balint's cits. 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, and 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.

А 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 also show impairments on visual search and counting tasks. Letter identification and reading abilities should 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, by contrast, are typically impaired at reaching for visual targets for all locations within the visual field; however, it has been noted that some patients with Balint's syndrome do demonstrate reaching difficulties in which one arm is more affected than the other. Patients with this syndrome are noted to be clumsy when grasping for 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 indeed 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 his/her 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 (see Perez et al. 1996). Case report studies, of which there are few, suggest that various rehabilitation strategies may be employed (see Rose et al. 2016; Zgaljardic et al. 2011), with minimal recovery of functional and physical abilities reported.

See Also

- Neglect Syndrome
- Simultanagnosia
- Visual Field Deficit

Further Reading

- 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.
- Rose, A., Wilson, B. A., Manolov, R., & Florschutz, G. (2016). Seeing red: Relearning to read in a case of Balint's syndrome. *NeuroRehabilitation*, 39, 111–117.
- Zgaljardic, D. J., Yancy, S., Levinson, J., Morales, G., & Masel, B. E. (2011). Balint's syndrome and post-acute brain injury rehabilitation: A case report. *Brain Injury*, 25, 909–917.

Barbiturates

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Synonyms

Central nervous system depressants; Sedativehypnotics

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 et al. 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. Reportedly, tolerance develops to the psychoactive effects of barbiturates, but less to the respiratory depressant effects, thereby increasing the 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. Some studies suggest a potential role for barbiturates in alcohol withdrawal. A recent review reported potential benefit of barbiturates, most notably for severe withdrawal and for treating seizures (Martin and Katz 2016).

See Also

Benzodiazepines

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 Associates.
- Martin, K., & Katz, A. (2016). The role of barbiturates for alcohol withdrawal syndrome. *Psychosomatics*. Epub ahead of print. Mar 2. pii: S0033–3182(16)00041–4. https://doi.org/10.1016/j.psym.2016.02.011.

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 US Supreme Court rejected Barefoot's suggestion that psychiatrists are not competent to make determinations regarding dangerousness in

future. The US 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 and 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. Barona et al. (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-IV (Wechsler 2008). Algorithms derived from the WAIS-IV with demographic variables have been developed by the Advanced Clinical Solutions (ACS; Pearson 2009). Holdnack et al. (2013) discuss the clinical utility of the Test of Premorbid Functioning (TOPF) to determine if the a patient's current performance is expected or represents a decline from a previous estimated level of ability. The TOPF can be used alone or in conjunction with demographic characteristics to estimate premorbid level of functioning. Research consistently suggests that TOPF estimates from the ACS 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-V, 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 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 and more technology-based jobs will very likely influence the occupations used for the equation. Similarly, as online education expands, an understanding of the type of education rather than amount of education may change the weighting of the model algorithms. As age expectancy increases, the role

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 = 1,18–9 = 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, Fig. 1 Barona et al. (1984) regression formulas for pre-morbid IQ

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

- Advanced Clinical Solutions for WAIS-IV and WMS-IV. (2009). Administration and scoring manual.
- 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 Clini*cal Psychology, 46, 1554–1555.

Barrow Neuropsychological Screen

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Synonyms

BNIS

В

Description

The Barrow Neurological Institute (BNI) Screen for Higher Cerebral Functions (BNIS) was developed to rapidly, but reliably and validly, assess disturbances in higher integrative brain functions (Prigatano et al. 1995). In addition to sampling speech/language, orientation, attention/concentration, visual spatial, and visual problem-solving, and memory functions, it is unique as a neuropsychological screening instrument insofar as it also assesses affect expression and perception as well as the person's awareness of memory abilities. This provides for seven subtest scores and a possible total score of 50/50 points. The latter score can be converted to an age-adjusted T score. The test has been translated into eight different languages and typically takes between 10 and 15 to administer (Prigatano et al. 2013).

Historical Background

While several screening tests of higher cerebral or integrative brain functions exist (see Lezak et al. 2004), they do not assess both cognitive and affective functions. Various brain disorders affect both dimensions, but differentially. At the BNI, a wide variety of brain dysfunctional patients are evaluated and treated. This led Prigatano and colleagues to develop a screening test that assess, in brief fashion, cognitive and affective functions that could be negatively influenced by various brain disorders. In doing so, they attempted to provide the experienced clinician with both quantitative and qualitative information useful in patient evaluations, management, and research.

Psychometric Data

The initial standardization study (Prigatano et al. 1995) reported good test-retest reliability (r = 0.94) and good sensitivity for identifying brain dysfunctional patients (i.e., 92%). The specificity of the instrument was modest (48%). Specificity was increased if performance on memory tests was taken into account (83%). That is, good

performance on memory items plus the total score on the BNIS successfully identified many normal functioning individuals.

Two doctoral dissertations have documented the reliability and validity of the BNIS. Wass (1997) demonstrated that performance on the BNIS correlated with independent and lengthier measures of neuropsychological test performance. BNIS subtest scores were also positively correlated with the Functional Independence Measure (FIM) and the adjunct of the Functional Assessment Measure (FIM + FAM of the Uniform Data Set for Medical Rehabilitation). Nearly 50% of the psychosocial-cognition score of the FIM-FAM was predicted by the seven independent subtests of the BNIS.

Denvall et al. (2002) administered a Swedish translation of the BNIS to 52 normal controls and 36 patients with well-documented brain disorders (the majority being those with traumatic brain injury and stroke). Swedish controls performed almost identically to American controls on this Swedish brain dysfunctional patients test. performed worst on the BNIS than Swedish controls. Hofgren (2009) further studied the Swedish version of the BNIS for her doctoral dissertation. The first study (Hofgren et al. 2007b) utilized 92 controls and 120 patients from a neurorehabilitation clinic. Significant differences were found between the control group and the patient group. Sensitivity was 88% and specificity was 78%. In a second study (see Hofgren 2009), the BNIS was compared to the Mini-Mental State Examination as well as to the FIM. Concordance between the BNIS total score and MMSE was good (r = 0.744). Both measures discriminated ADLdependent from nondependent patients. A third study (Hofgren et al. 2007a) used the BNIS as a predictor of return to work and level of activities of daily living (ADL) in 58 stroke victims. At 1 year follow-up, the correlation of the BNIS total score and the psychosocial-cognitive scale of the FIM was r = 0.376 (p = 0.001). BNIS total score did not predict ability to return to work, but most of the patients studied did not return to work. In a fourth study, Hofgren et al. (2008) studied ADL, housing, and return to work 2 years after cardiac arrest in 22 patients. The BNIS total score was higher in patients living in their home and who were able to

return to work (mean total score was 43/50 points, range 41–47). In contrast, the mean BNIS total score was notably lower in those living in their own home but not able to return to work (mean total score 37, range 35–42) and even lower for those living in sheltered accommodations (mean Total score was 24, range of 19–32).

A study from the Netherlands further supported the clinical utility and validity of the BNIS when measuring outcome after stroke (Boosman et al. 2013). The BNIS showed good internal consistency (alpha = 0.82) and no floor or ceiling effects in stroke patients described as having a good functional outcome using the Barthel Index. Selected subtests correlated with more time-consuming and extensive tests of different cognitive domains. For example, the Boston Naming Test scores correlated 0.538 (p = 0.000) with the speech/language subtest scores of the BNIS. Likewise, the memory subtest scores correlated with the total scores obtained from the Rey Auditory Verbal Learning Test (r = 0.548). These findings replicated the earlier observations of Wass (1997).

Recently, a normative study using the French translation of the BNIS also reported findings very similar to what was observed in the original American standardization study (see Prigatano et al. 2013).

Clinical Uses

Prigatano and Wong (1999) studied 95 heterogeneous brain dysfunctional patients treated on an inpatient neurorehabilitation unit who were classified as having achieved their rehabilitation goals or not. Patients who achieved their rehabilitation goals had higher BNIS total scores at admission compared to patients that did not achieve their rehabilitation goals. Impaired emotional functioning was equally important as impaired cognitive functioning when predicting goal attainment. Interestingly, both groups were equally impaired in their awareness of the memory functioning on admission. However, the group that eventually achieved their goals showed improved awareness after the rehabilitation experience.

Studies have also documented the potential value of the BNIS in cases of differential diagnosis.

Rosenstein et al. (1997) compared 41 patients with known cerebral dysfunction, with 22 psychiatric patients (some who were psychotic) and 22 medical inpatients. Psychiatric and medical patients scored significantly higher on the BNIS total score compared to the brain dysfunctional patients. Using the recommended cutoff score of 47/50 points, 40 of the 41 brain dysfunctional patients were correctly classified (97.5%). The specificity for medical controls was 68%. The specificity for psychiatric patients was much lower (40.9%). These findings suggest that multiple factors can influence the patient's BNIS total score, including the age, education, and psychiatric status of the patient.

In a French study, Prigatano et al. (2014a) demonstrated that patients with mild cognitive impairment of the amnestic type (MCI) had poorer scores on the BNI screen than age-matched patients who did not have MCI. MCI patients showed not only disturbance in memory functioning but impaired awareness and affect expression and perception. The study demonstrated the potential importance of assessing both cognitive and affective functions in cases of differential diagnosis.

Also, recent case studies have suggested that performance on the BNIS may help identify patients with anosognosia (e.g., Prigatano et al. 2014b).

References

- Boosman, H., Visser-Meily, J. M. A., Post, M. W. M., Duits, A., & van Heugten, C. M. (2013). Validity of the Barrow Neurological Institute (BNI) screen for higher cerebral functions in stroke patients with good functional outcome. *The Clinical Neuropsychologist*, 27(4), 667–680.
- Denvall, V., Elmstahl, S., & Prigatano, G. P. (2002). Replication and construct validation of the BNI Screen for higher cerebral function with the Swedish population. *Journal of Rehabilitative Medicine*, 34, 153–157.
- Hofgren, C. (2009). Screening of cognitive functions: Evaluation of methods and their applicability in neurological rehabilitation (Doctoral Dissertation, University of Gothenburg, Gothenburg, Sweden).
- Hofgren, C., Bjorkdahl, A., Esbjornsson, E., & Stibrant-Sunnerhagen, K. (2007a). Recovery after stroke: Cognition, ADL function and return to work. *Acta Neurologica*, 115, 73–80.
- Hofgren, C., Esbjornsson, E., Aniansson, H., & Sunnerhagen, K. S. (2007b). Application and

validation of the Barrow Neurological Institute Screen for higher cerebral functions in a control population and in patient groups commonly seen in neurorehabilitation. *Journal of Rehabilitative Medicine, 39*, 547–553.

- Hofgren, C., Lundgren-Nilsson, A., Esbjornsson, E., & Sunnerhagen, K. S. (2008). Two years after cardiac arrest; cognitive status, ADL function and living situation. *Brain Injury*, 22(12), 972–978.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
- Prigatano, G. P., & Wong, J. L. (1999). Cognitive and affective improvement in brain dysfunctional patients who achieve inpatient rehabilitation goals. *Archives of Physical Medicine and Rehabilitation*, 80, 77–84.
- Prigatano, G. P., Amin, K., & Rosenstein, L. D. (1995). Administration and scoring manual for the BNI Screen for higher cerebral functions. Phoenix: Barrow Neurological Institute.
- Prigatano, G. P., Tonini, A., Truelle, J. L., & Montreuil, M. (2013). Performance of a French sample on the French translation of the BNI Screen for higher cerebral functions. *Brain Injury*, 27(12), 1435–1440.
- Prigatano, G. P., Montreuil, M., Chapple, K., Tonini, A., Toron, J., Paquet, C., Dumurgier, J., Hugon, J., & Truelle, J. L. (2014a). Screening for cognitive and affective dysfunction in patients suspected of mild cognitive impairment. *International Journal of Geriatric Psychiatry*, 29(9), 936–942.
- Prigatano, G. P., Hendin, B. A., & Heiserman, J. E. (2014b). Denial or unawareness of cognitive deficit associated with multiple sclerosis? A case report. *Journal of Clinical and Experimental Neuropsychology*, 36(4), 335–341.
- Rosenstein, L. D., Prigatano, G. P., & Nayak, M. (1997). Differentiating patients with higher cerebral dysfunction from patients with psychiatric or acute medical illness with the BNI Screen for higher cerebral functions. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 10*(2), 113–119.
- Wass, P. J. (1997). An analysis of the construct-related and ecological validity of the Barrow Neurological Institute Screen for higher cerebral functions (Doctoral Dissertation, University of Windsor, Windsor).

Barthel Index

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Synonyms

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
- 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 and Ceiling Rules

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Synonyms

Entry and discontinue rules

Definition

A basal and ceiling rule refers to the entry point and discontinue point of a psychometric test. The purpose of basal and ceiling rules is to reduce the number of items an examinee is required to attempt, by eliminating items that are too easy and too difficult. Doing so reduces administration time and burden on the examinee.

Although specific start and stop rules and administration procedures vary across tests, basal and ceiling rules are generally used for tests in which the items are ordered from easiest to most difficult. The most common basal procedure is to first start at an early, easier item based on the examinee's age. The examinee is then required to *establish a basal* by completing a predetermined number of consecutive items correctly (e.g., three correct items in a row). In general, items are administered in reverse order, so that they are increasingly easier, until the basal is established. In doing so, the examiner can refrain from administering easier items below the basal, assuming that the examinee would be able to answer them correctly. Once the basal has been established, items are then administered in forward order, becoming increasingly more difficult. The test continues in this fashion until a ceiling rule has been met. Ceiling rules typically require that an examinee answer a predetermined number of consecutive items incorrectly (e.g., three misses in a row). Some ceiling rules require a certain number of misses out of a larger number of consecutive items (e.g., four misses across five consecutive items). Once the ceiling rule has been met, the examiner discontinues the test under the assumption that the examinee would continue to answer the remaining, more difficult items incorrectly.

After administration is complete, some tests follow a *double basal* rule. In this situation, the examinee has established an initial basal (by completing a set number of consecutive items correctly) and then subsequently completed an additional string of correct items to meet a second basal. Depending on the test, some procedures allow for the examiner to count all items below the second basal as correct, even if the examinee actually responded to some of them incorrectly. Because of the wide variability across administration protocols, it is vital for the examiner to be familiar with basal and ceiling rules prior to administration.

Current Knowledge

Although many test companies do not publish details regarding the development of their basal and ceiling rules, the general goal of the rules is to reduce the number of administered items while minimizing the effects of these rules on raw and standard scores. The Wechsler tests (e.g., WAIS-IV, WMS-IV, WISC-V) describe one such development procedure that may be commonly used among other test publishers. For these tests, national tryout data are used to determine the difficulty of all test items, to order them from easiest to hardest based on how frequently they are answered correctly in the sample. Start points are then set

low and revised upward in the standardization procedure to ensure that a minimal number of examinee raw scores (commonly 5%) change by moving the start point forward. The ideal starting item has a high pass rate in the normative sample, to minimize the occurrence of reversal procedures. Discontinue rules in the standardization procedure are also initially conservative and then adjusted down to minimize changes in examinee raw and age-adjusted standard scores. Additionally, discontinue rules are developed to maintain a high rank-order correlation (e.g., 0.98) of total raw scores before and after the adjustment. This ensures that the standardization sample subjects maintain the same position in their rank order relative to others in their age group.

See Also

- Ceiling Effect
- Floor Effect
- ► Item Difficulty
- ► Standard Scores
- Test Construction
- Testing the Limits

References and Readings

- Wechsler, D. (2008). *Wechsler adult intelligence scale* (4th ed.). Bloominton: Pearson.
- Wechsler, D. (2009). *Wechsler memory scale* (4th ed.). Bloominton: Pearson.
- Wechsler, D. (2014). Wechsler intelligence scale for children (5th ed.). Bloomington: Pearson.

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

Current Knowledge

Role in behavior and cognition. Rosvold demonstrated a topographical coupling between the prefrontal cortex and basal ganglia (Rosvold 1972; Johnson et al. 1968; Middleton and Strick 2000; Averbeck et al. 2014). Recent neuroimaging findings using functional magnetic resonance imaging (Arsalidou et al. 2013) and diffusion tensor imaging tools (Kotz et al. 2013) confirmed such topographical organization between the basal ganglia and the prefrontal cortex. Considering the major outflow of the basal ganglia to the thalamus (Haber and Calzavara 2009), it is not surprising that a substantial amount of research strongly supports the role of the basal ganglia in higherorder behavioral and cognitive tasks (Haber and Knutson 2010). Much of this research relies on striatal dopaminergic deficit and cortical lesion models in both human patients and animal models.

Basal Ganglia

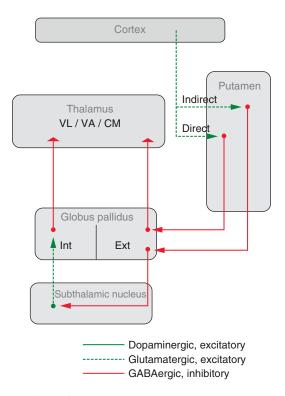
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Synonyms

Basal nuclei

Definition

The basal ganglia refer specifically to a group of subcortical structures considered as extrapyramidal motor components. These components include caudate and putamen, substantia nigra, subthalamic nucleus, and globus pallidus (GP). Figure 1 depicts major circuitry within the basal ganglia.



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

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 et l. 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, self-monitoring (Brown and Marsden 1991; Brown et al. 1998; Taylor et al. 1986), and certain types of learning (Shohamy 2011). For example, PD patients demonstrate significant impairment in Petrides' selfordered pointing task (Petrides and Milner 1982; West et al. 1998).

The basal ganglia have been shown to regulate temporal coupling and ordering of both motor and nonmotor sequences (Malapani et al. 1998; Kotz et al. 2009). Interestingly, a role in auditory rhythm detection and generation has been supported (Grahn and 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 et al. 2009). For example, Smits-Bandstra et al. have described the basal ganglia in the setting of persons who stutter (Smits-Bandstra and 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, automaticity, and category learning (Ashby et al. 2010; Seger 2008; Tricomi et al. 2009; Tricomi and Fiez 2008). Further, different subregions of the basal ganglia have been shown to process learning stimuli under different time scales and fulfill different roles during learning. For example, it has been shown that the anterior part of the basal ganglia (head of the caudate nucleus) is involved in learning through immediate feedback, while posterior regions of the basal

ganglia (the putamen and GP) are involved in learning through feedback presented with a delay between action and outcome (Dobryakova and Tricomi 2013). The basal ganglia are also involved in a number of other cognitive functions including working memory (e.g., Baier et al. 2010), attentional systems (Sarter et al. 2006), and executive decision-making and control (Ino et al. 2010; Kim and Hikosaka 2013). 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 behaviors and as such are part of the complex "executive" system of the brain. Thus, the basal ganglia are critical in performing everyday practical tasks in an effortless and efficient manner (Koziol and Budding 2009).

Organization. The striatal complex is composed of the caudate nucleus and the putamen (Graybiel 2000). The caudate nucleus can be further subdivided into the head, body, and tail that play separable roles in cognition (Seger 2008). 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 et al. 2007). This difference in acetylcholinesterase content provides a convenient histochemical differentiation between neostriatal regions (DiFiglia et al. 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 et al. 1999). The striatum tonically inhibits its pallidal and nigral targets.

The striatum itself receives inhibitory GABAergic projections from substantia nigra pars reticulata (Boyes and 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 et al. 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. Intra-nigral connections serve as modulatory loops: GABAergic input to SNpc decreases dopaminergic activity within the pars compacta; dopaminergic input to SNpr decreases GABAergic activity (Boyes and Bolam 2007; DeLong and 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 biconvexshaped 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 et al. 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 and 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, and pathological CAG trinucleotide repeats (Shao and Diamond 2007). Mechanism of disease may include enhanced corticostriate activity and enhanced thalamic disinhibition (Centonze et al. 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 et al. 2004). Speech and gait therapy are often employed. Depression is common among Huntington's disease patients, and antidepressant treatment has been described (Korenyi and Whittier 1967).

PD, late idiopathic onset, and early-onset signs include resting tremor, oculomotor disturbance, and loss of postural reflexes, among other dyskinesias. Pathology includes loss of nigral dopaminergic neurons, although the cause is multivariable (Nagatsu and 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 and 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 Degeneration
- Deep Brain Stimulator (Parkinson's)
- ▶ 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
- Parkinson's Dementia
- Physiologic Tremor
- ▶ Putamen
- Pyramidal System

- ► Rigidity
- Striatum
- Substantia Nigra
- Supplementary Motor Area (SMA)
- ► Tardive Dyskinesia
- Thalamus
- ► Tremor

References and Readings

- Arsalidou, M., Duerden, E. G., & Taylor, M. J. (2013). The centre of the brain: Topographical model of motor, cognitive, affective, and somatosensory functions of the basal ganglia. *Human Brain Mapping*, 34(11), 3031–3054. https://doi.org/10.1002/hbm.22124.
- Ashby, F. G., Turner, B. O., & Horvitz, J. C. (2010). Cortical and basal ganglia contributions to habit learning and automaticity. *Trends in Cognitive Sciences*, 14(5), 208–215. https://doi.org/10.1016/j.tics.2010. 02.001.
- Averbeck, B. B., Lehman, J., Jacobson, M., & Haber, S. N. (2014). Estimates of projection overlap and zones of convergence within frontal-striatal circuits. *Journal of Neuroscience*, 34(29), 9497–9505. https://doi.org/ 10.1523/JNEUROSCI.5806-12.2014.
- Baier, B., Karnath, H. O., & Dieterich, M. (2010). Keeping memory clear and stable-the contribution of human basal ganglia and prefrontal cortex to working memory. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 30(29), 9788–9792.
- 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 Neurosciences*, 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.
- Dobryakova, E., & Tricomi, E. (2013). Basal ganglia engagement during feedback processing after a substantial delay. *Cognitive, Affective, & Behavioral Neuroscience, 13*(4), 725–736. https://doi.org/10.3758/ s13415-013-0182-6.
- 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.
- Graybiel, A. M. (2000). The basal ganglia. Current Biology, 10(14), R509–R511. https://doi.org/10.1016/ S0960-9822(00)00593-5.
- Haber, S. N., & Calzavara, R. (2009). The cortico-basal ganglia integrative network: The role of the thalamus. *Brain Research Bulletin*, 78(2–3), 69–74. https://doi. org/10.1016/j.brainresbull.2008.09.013.
- Haber, S. N., & Knutson, B. (2010). The reward circuit: Linking primate anatomy and human imaging. *Neuropsychopharmacology*, 35(1), 4–26. https://doi. org/10.1038/npp.2009.129.
- Haines, D. (2002). Fundamental neuroscience. New York: Churchill Livingstone.
- 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.
- Ino, T., Nakai, R., Azuma, T., Kimura, T., & Fukuyama, H. (2010). Differential activation of the striatum for decision making and outcomes in a monetary task with gain and loss. *Cortex*, 46(1), 2–14. https://doi.org/10.1016/ j.cortex.2009.02.022.
- 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 Neurol*ogy, 21, 20–34.
- Kern, D., & Kumar, R. (2007). Deep brain stimulation. *The Neurologist*, 13(5), 237–252.
- Kim, H. F., & Hikosaka, O. (2013). Distinct basal ganglia circuits controlling behaviors guided by flexible and stable values. *Neuron*, 79(5), 1001–1010. https://doi. org/10.1016/j.neuron.2013.06.044.
- 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.
- Kotz, S. A., Anwander, A., Axer, H., & Knösche, T. R. (2013). Beyond cytoarchitectonics: The internal and external connectivity structure of the caudate nucleus. *PloS One*, 8(7), e70141. https://doi.org/10.1371/journal.pone.0070141.
- 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.
- Middleton, F. A., & Strick, P. L. (2000). Basal ganglia and cerebellar loops: Motor and cognitive circuits. *Brain Research Reviews*, 31(2–3), 236–250.
- 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 subjectordered tasks after frontal and temporal lobe lesions in man. *Neuropsychologia*, 20, 601–604.
- Rosvold, H. (1972). The frontal lobe system: Corticalsubcortical interrelationships. *Acta Neurobiologica Experimentalis (Warsaw)*, 32, 439–460.
- Sarter, M., Gehring, W. J., & Kozak, R. (2006). More attention must be paid: The neurobiology of attentional effort. *Brain Research Reviews*, 51(2), 145–160. https://doi.org/10.1016/j.brainresrev.2005.11.002.
- Seger, C. a. (2008). How do the basal ganglia contribute to categorization? Their roles in generalization, response selection, and learning via feedback. *Neuroscience and Biobehavioral Reviews*, 32(2), 265–278. https://doi. org/10.1016/j.neubiorev.2007.07.010.
- Shao, J., & Diamond, M. I. (2007). Polyglutamine diseases: Emerging concepts in pathogenesis and therapy. *Human Molecular Genetics*, 15(16), R115–R123. Spec No 2.
- Shohamy, D. (2011). Learning and motivation in the human striatum. *Current Opinion in Neurobiology*, 21(3), 408–414. https://doi.org/10.1016/j. conb.2011.05.009.
- 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.
- Tricomi, E., & Fiez, J. a. (2008). Feedback signals in the caudate reflect goal achievement on a declarative memory task. *NeuroImage*, 41(3), 1154–1167. https://doi. org/10.1016/j.neuroimage.2008.02.066.
- Tricomi, E., Balleine, B. W., & O'Doherty, J. P. (2009). A specific role for posterior dorsolateral striatum in human habit learning. *The European Journal of Neuroscience*, 29(11), 2225–2232. https://doi.org/10.1111/ j.1460-9568.2009.06796.x.
- 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.

BASC-3

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Description

The Behavior Assessment System for Children, Third Edition (BASC-3; Reynolds and Kamphaus 2015) is a multimethod, multidimensional system of related instruments that can be used to conduct a comprehensive assessment of behavioral and emotional functioning of children, adolescents, and young adults aged 2–25 years. The BASC-3 is a multimethod in that it has the following components, which may be used individually or in any combination:

- 1. A Teacher Rating Scale (TRS) and a Parent Rating Scale (PRS) that gather multiple perspectives of observable behavior across settings and raters, using age-appropriate forms
- A Self-Report of Personality (SRP) that a child, adolescent, or young adult can use to describe his or her behaviors, emotions, and self-perceptions
- 3. A Structured Developmental History (SDH) form that provides information about the course of development and family history that are important to make accurate diagnosis
- A Student Observation System (SOS) form that can be used for recording and classifying directly observed classroom behavior, using a smartphone, laptop computer, or paper form

The BASC-3 is multidimensional in that it measures numerous aspects of behavior and personality, including positive (adaptive) as well as negative (clinical) dimensions. Like its previous editions (BASC-2, Reynolds and Kamphaus 2004; BASC, Reynolds and Kamphaus 1992), the BASC-3 forms remain psychometrically strong instruments that are easy to complete and relevant to both school-based and clinically based settings. The norm samples are new and reflect the latest US Census estimates available at the time of the standardization project. In addition to new test items, the BASC-3 TRS, PRS, and SRP now offer Clinical Indexes and new Executive Functioning Indexes. The Clinical Indexes were developed based on items that discriminated between clinical and nonclinical samples and may be particularly useful for helping to rule in or rule out certain clinical diagnoses or educational classifications and for assessing the amount of functional impairment being experienced by the child or adolescent. The Executive Functioning Indexes found on the TRS and PRS provide insight into specific executive functioning domains that are important when working with deficits such as attention deficit hyperactivity disorder (ADHD), without the need for additional rating scales. The Self-Report of Personality Interview form, for ages 6-7, has been redesigned. Rather than simply reading a series of items to the child, a total of 14 items are read to the child. Responses are obtained using

a semi-structured format that helps to elicit more natural responses by the child that can be used to supplement findings from the BASC-3 TRS and PRS and aid in treatment planning. The SDH form now offers an option for paired administration with the PRS. When both are administered digitally, additional items will be automatically included during the SDH administration based on PRS results, providing additional context that can be helpful for making accurate classification or diagnostic decisions.

While paper administration and hand scoring options are available for the TRS, PRS, SRP, SOS, and SDH forms, the primary way to administer and score BASC-3 forms is digitally using the Qglobal web-based scoring and reporting platform. Forms can be administered locally on a laptop or web-enabled digital handheld device, or a web link can be emailed to a respondent to complete a form via a secured testing portal. Upon completion, forms can be immediately scored, and reports generated. Report options have been consolidated from previous editions. The basic report offered is the Interpretive Summary Report, which provides extensive score profiles, along with basic interpretive and clinical interpretive information, critical items, and item-by-scale listings. The most comprehensive report offered is the Interpretive Summary Report with Intervention Recommendations. This report provides intervention recommendations based on the obtained score profiles and is based on the BASC-3 Behavior Intervention Guide (Vannest et al. 2015a; see below for more detailed information).

Like its previous editions, the BASC-3 remains committed to a triangulated view of the child's behavioral and emotional functioning by examining behavior in multiple settings (at home and school) and evaluating the child's emotions, personality, and self-perceptions.

Key Features of the BASC-3

The BASC-3 has numerous features that make it one of the most sophisticated and reliable systems of behavior assessment available today. A hallmark of the BASC tools has been the comprehensiveness and breadth of behavioral and emotional problems covered. The number of problem areas included on the BASC-3 tools is useful for helping to rule in (and out) behavioral and emotional functioning deficits that can look similar in nature, providing a distinct advantage over more narrowband classification instruments. The information contained on the BASC-3 tools are directly relevant to behavioral disorders found in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (American Psychiatric Association 2013), as well as general categories of problems addressed in legislation such as the Individuals with Disabilities Education Act (e.g., the diagnosis of severe emotional disturbance). In addition, the inclusion of adaptive behaviors on the BASC-3 TRS, PRS, and SRP forms provides clinicians with information that can be used to help leverage a child's or adolescent's existing strengths when developing individualized intervention or treatment plans. Another key BASC-3 feature is a grounded development approach that emphasizes a balance of both theory and statistics, resulting in tools with strong psychometric properties and clinical utility. Finally, the BASC-3 tools promote ease of administration and scoring, and the inclusion of scales that can help detect threats to the validity and usefulness of obtained responses makes the BASC-3 applicable to clinical, forensic numerous school, and applications.

Historical Background

The original BASC (Reynolds and Kamphaus 1992) was published by American Guidance Service, following 7 years of development work. It was standardized for use with children and adolescents ages 4–18 years and was rapidly adopted as the most frequently administered behavior scales in the schools in the United States. Spanish versions were subsequently developed for international applications, as well as smaller research-only adaptations in several additional languages. The release of the second edition (BASC-2, Reynolds and Kamphaus 2004) continued to be well

received by users in both the United States and a number of other countries. Several other tools in the BASC-2 family were released in subsequent years, including the Parenting Relationship Questionnaire (PRQ, Kamphaus and Reynolds 2006), the Behavioral and Emotional Screening System (BESS; Kamphaus and Reynolds 2007), the BASC-2 Intervention Guide (Vannest et al. 2008), and the BASC-2 Progress Monitor (Reynolds and Kamphaus 2010). These tools were consistent with the Response to Intervention movement in the US educational system that emphasized a screening, intervention, and monitoring approach to addressing a student's functional deficits. The BASC-3 continues the tradition of innovation with improvements to existing instruments and the development of the new components described below.

Psychometric Data

The scales included on the TRS, PRS, and SRP are 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. The ease of scale interpretation is partly attributable to the items which comprise them. The approach used to develop the original BASC items involved surveying teachers, parents, and students about behaviors that were the most difficult to manage or behaviors that were the most disruptive; in addition, respondents were asked to provide examples of positive behaviors that were observed. This survey process was repeated during the BASC-3 standardization project, helping to ensure that the items written during the development of the original BASC remain relevant and that new behaviors deemed important were also included on the BASC-3 edition. Factor-analytic evidence presented in the BASC-3 Manual provides support for the overall scale and composite structure used for reporting results.

The BASC-3 scales and composites have high internal consistency and test-retest reliability. Most alpha coefficients for the BASC-3 subscales and composites exceed 0.80 and are sufficiently reliable for application to diagnostic and treatment issues. Additionally, the BASC-3 offers various types of validity checks to help the clinician detect careless or untruthful responding, misunderstanding, or other threats to validity. The BASC-3 Manual demonstrates validity evidence for the proposed applications of the BASC-3 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.

BASC-3 Uses

Clinical Diagnosis

The BASC-3 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-5. Because the components of the BASC-3 can be used separately or in combination, the BASC-3 may be easily used 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-3. 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.

Educational Classification

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-3 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 ADHD and depression have known academic consequences; learning disabilities and intellectual disability are often associated with adjustment problems such as low self-concept or anxiety. It is strongly suggested that every child experiencing academic difficulties receives 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.

Program Evaluation

Repeated use of the BASC-3 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).

Forensic Evaluation

The BASC-3 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) provide examples of uses of the original BASC in forensic situations such as child custody evaluations, personal injury litigation, and juvenile certification.

The BASC-3 Manual contains considerable information on the reliability of scale 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 are additional crucial data on the ability of the BASC-3 scale scores 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-3 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 and Kamphaus 2002). In court proceedings, individuals may have much to gain by appearing to have more or fewer problems than what 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-3 has scales designed and tested for the detection of dissimulation in responding by parents, teachers, and children. In particular, the BASC-3 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.

Additional BASC-3 Components

The BASC-3 includes a variety of other instruments that can be used to help identify and improve behavioral and emotional functioning. Each of these instruments is described briefly below.

BASC-3 Behavioral and Emotional Screening System (BESS)

The BASC-3 BESS (Kamphaus and Reynolds 2015b) is designed to quickly and efficiently identify risk for behavioral or emotional problems and predict mental health and educational outcomes. The BASC-3 BESS consists of two teacher forms (ages 3-5 and ages 6-18), two parent forms (ages 3-5 and ages 6-18), and one self-report form (ages 8-18). Each form of the BASC-3 BESS is brief and requires no prior training and coaching of the informant.

Each BASC-3 BESS form provides a Behavioral and Emotional Risk Index (BERI), which indicates the amount of risk a child or adolescent has of having or developing a behavioral or emotional problem. The teacher and parent forms offer additional subindex scores, including an Externalizing Risk Index, Internalizing Risk Index, and Adaptive Skills Risk Index. In addition to the BERI, the self-report form also provides subindex scores for an Internalizing Risk Index, a Self-Regulation Risk Index, and a Personal Adjustment Risk Index.

Using the same item response formats as the BASC-3 TRS and PRS, each BASC-3 BESS form produces a single score indicating "normal risk" (T = 20-60), "elevated risk" (61-70), or "extremely elevated risk" (T = 71 or higher). Validity indexes are provided for each form, and Spanish adaptations and translations are available for parent and student forms. Administration, scoring, and reporting (both individual- and group-level reports) are available on the Q-global testing platform.

The BASC-3 BESS manual includes a detailed discussion of development procedures and a separate chapter devoted to validity and reliability evidence collected to date. All BERI reliability coefficients exceed 0.90, and test-retest correlations are in the upper .80s or higher. A variety of correlational studies are presented, including the BASC-3 TRS, PRS, and SRP, along with other behavioral/emotional functioning tests. The BASC-3 BESS manual also provides a detailed discussion of promising screening practices, including the use of multiple "gates" (i.e., assessment stages that range from broad-based screening on all students to more detailed assessment/ evaluation on students identified at previous gates). In addition, the Manual discusses linking screening results with early intervention strategies aimed at preventing the onset of mental health disorders or unsuccessful educational outcomes.

BASC-3 Parenting Relationship Questionnaire (PRQ)

The BASC-3 Parenting Relationship Questionnaire (PRQ; Kamphaus and Reynolds 2015a) is designed to capture a parent's perspective of the parent-child relationship (or the perspective of a person serving a similar role). It assesses traditional parent-child dimensions such as attachment and involvement and also provides information on parenting style, parenting confidence, stress, and satisfaction with the child's school. The BASC-3 PRQ is used in clinical, pediatric, counseling, school, and other settings where there is a need to understand the nature of the parent-child relationship. It is particularly important when implementing home-based intervention strategies and/or treatment monitoring. The BASC-3 PRQ can be completed in approximately 15 minutes and is available in English and Spanish. It should be administered to mothers and fathers (or caregivers) of children ages 2-18 years. Administration, scoring, and reporting are available on the Qglobal platform. The BASC-3 PRQ Manual provides information about the reliability and validity of evidence collected during the standardization stage of development. Internal consistency reliability coefficients for each scale were typically in the mid-.80s or higher. A variety of correlational studies are also presented.

BASC-3 Flex Monitor

The BASC-3 Flex Monitor (Reynolds and Kamphaus 2016) is used to monitor and track the effect of behavioral interventions implemented by a psychologist or other professionals in a school or clinical environment. Available via Q-global, the BASC-3 Flex Monitor provides a bank of over 700 behaviorally or emotionally based items that can be selected to create a customized monitoring form for teachers, parents, or students (via a self-report form) that enable score comparisons to a nationally representative population sample. While creating forms, users can automatically calculate reliability estimates that are based on a normative sample. In addition, the BASC-3 Flex Monitor offers a variety of standard forms that are available for immediate use, including inattention/hyperactivity, internalizing problems, disruptive behaviors, developmental social disorders, and school problems. Parent and self-report forms are available in both English and Spanish. Individual reports tracking progress on up to ten administrations of a form or aggregated group-based reports can be easily generated on the Q-global platform.

BASC-3 Behavior Intervention Guide

The BASC-3 Behavior Intervention Guide (Vannest et al. 2015a) provides evidence-based intervention strategies for 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. These interventions represent a compendium of the most effective strategies that have been published in empirically based research studies.

In addition to providing background characteristics and conditions of each behavioral or emotional problem area, each chapter provides a number of intervention strategies that are presented in a preparation, implementation, and evaluation format. The intervention steps that are provided can be easily used by behavior experts (e.g., psychologists, counselors, behavioral specialists) to promote more desirable behavior and reduce problem behaviors. The BASC-3 Behavior Intervention Guide also offers accompanying tools that are designed to promote intervention fidelity and positive outcomes, including parent tip sheets that promote and provide structured involvement by parents or caregivers, supplemental forms that accompany the intervention strategies (e.g., daily log journals, activities, sample forms, etc.), and a fidelity documentation checklist.

For small group or classroom-based solutions, the BASC-3 Behavioral and Emotional Skill Building Guide (Vannest et al. 2015b) can be used in general or special education settings to help promote positive behavioral and emotional functioning. Based on the foundations established in the BASC-3 Behavior Intervention Guide, this guide provides tier one and tier two intervention strategies in a classroom curriculum format, as well as additional strategies that are targeted for small group settings. Teachers can follow the lesson plans provided in the guide to teach schoolwide expectations using activities that are fast-paced and brief, lasting around 5 minutes each. Additional instructional strategies for classrooms and small groups are also available for the behavioral and emotional problems included in the BASC-3 Behavior Intervention Guide.

References and Readings

- American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). Washington, DC: American Psychiatric Association.
- Kamphaus, R. W., & Reynolds, C. R. (2006). Parenting relationship questionnaire. Minneapolis: Pearson.
- Kamphaus, R. W., & Reynolds, C. R. (2007). Behavioral and emotional screening system. Minneapolis: Pearson.
- Kamphaus, R. W., & Reynolds, C. R. (2015a). BASC-3 parenting relationship questionnaire. Bloomington: NCS Pearson Inc.
- Kamphaus, R. W., & Reynolds, C. R. (2015b). BASC-3 behavioral and emotional screening system. Bloomington: NCS Pearson Inc.
- Reynolds, C. R., & Kamphaus, R. W. (1992). Behavior assessment system for children. Bloomington: 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: Pearson Assessments.
- Reynolds, C. R., & Kamphaus, R. W. (2010). BASC–2 progress monitor. Bloomington: NCS Pearson Inc.
- Reynolds, C. R., & Kamphaus, R. W. (2015). *Behavior* assessment system for children (3rd ed.). Bloomington: NCS Pearson, Inc. (BASC–3).
- Reynolds, C. R., & Kamphaus, R. W. (2016). BASC–3 Flex Monitor. Bloomington: NCS Pearson, Inc.
- 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: Pearson.
- Vannest, K. J., Reynolds, C. R., & Kamphaus, R. W. (2015a). BASC-3 behavior intervention guide. Bloomington: Pearson.
- Vannest, K. J., Reynolds, C. R., & Kamphaus, R. W. (2015b). BASC-3 behavioral and emotional skill building guide. Bloomington: Pearson.

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):

Base rate

= #cases with condition of interest/ #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 et al. 2012). 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 et al. 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., Bigler, E. D., & Tranel, D. (2012). *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.

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. It is published by Pearson and was first made available in 2004. 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

demonstration, training modules, 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 subtests can be administered independently to measure specific skills or in any combination. There are four grade levels (I-IV), 3rd-4th grade, 5th-6th grade, 7th-8th, and 9th-12th. The survey form is a screening tool comprising two subtests: verbal skills and math skills. Verbal skills are assessed using vocabulary, language mechanics, and reading comprehension questions, and math skills are assessed using math computation and math application questions. 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 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-global web-based administration, Q local software, or mail-in scoring. The student summary report is a one-page report that summarizes the student's performance by skills composite, subtest, and learning objectives. The student summary report includes standard scores, percentile scores, age equivalents, and grade equivalents as well as a performance classification (low average, average to above average) by achievement area. A parent's report is included with each student report. The parent's report graphs percentile scores and includes a space for written comments. There is also an optional report available for level 4 of the BASI comprehensive version, titled the BASI college report. The college report shows how a student's BASI scores compared to the scores of a census-matched national sample at the student's grade level.

The adult summary report is a one-page report that summarizes an adult's performance by skills composite, subtest, and learning objectives. It includes percent correct, grade equivalent, and classification by achievement area.

Historical Background

Achilles N. Bardos, PhD, is the author of the test (http://www.unco.edu/cebs/SchoolPsych/faculty/ BASI/index.html). The BASI was published in 2004. 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.

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).

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 in reading, writing and math; (3) place for college students; (4) make placement decisions for ESL, GED, and program placement; (5) track academic progress, (6) efficiently complete triennial evaluations for students with an IEPs or 504 plans; and (7) practice for or predict performance on high-stakes tests." Specific applications in four settings are proposed in the BASI Flash demonstration (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. 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.

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 17 Feb 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 17 Feb 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 anti-thrombotic 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 et al. 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 et al. 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 et al. 2012). 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 survey from Sweet et al. (2011) 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 78% in 2010. 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., Bigler, E. D., & Tranel, D. (2012). *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., Meyer, D. G., Nelson, N. W., & Moberg, P. J. (2011). The TCN/AACN 2010 "salary survey": Professional practices, beliefs, and incomes of U.-S. neuropsychologists. *The Clinical Neuropsychologist*, 25, 12–61.

Battle's Sign

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Synonyms

Periauricular or mastoid ecchymosis

Definition

Named after English surgeon, Dr. William Henry Battle, this is a clinical symptom suggestive of basilar skull/middle cranial fossa fracture. After blunt force head trauma, leaking of blood from the blood vessels in the skull, typically the posterior auricular artery, leads to a crescent-shaped bruise wrapping behind the base of the earlobe and extending posteriorly toward the point of the neck where the base of the skull meets the neck. A patient with this symptom may present with acute bloody discharge of the ear and/or nose. Battle's 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.

Battlefield Assessment

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Description

Researchers report that "Due to enemy tactics and the frequency of operational missions, many service members are at risk of sustaining more than one concussion during deployment" (Barth et al. 2010). It is further noted that as numerous service members go on multiple deployments, the opportunity for injury is increased. According to the Defense and Veterans Brain Injury Center (DVBIC), 40% of all blast injuries in the Operation Enduring Freedom/Operation Iraqi Freedom (OEF/OIF) conflicts involve traumatic brain injury (TBI). Furthermore, many mild and moderate head injuries are reportedly overlooked due to more imminent medical treatments focused on polytrauma injuries including amputation and burns (Martin et al. 2008).

DoD instructional policy (DoDi 6490.11), entitled "Guidance for Management of Mild Traumatic Brain Injury/Concussion in the Deployed Setting," is intended to protect service members involved in potentially concussive events, direct leaders on mandated screening and reporting requirements, guide medical evaluation and treatments, and outline minimum mandatory rest periods. Specifically, the policy mandates that a service member be medically evaluated if exposed to a potentially concussive event such as (1) involvement in a vehicle blast, collision, or rollover, (2) a direct blow to the head or witnessed loss of consciousness, (3) presence within 50 m of a blast (inside or outside), and/or (4) exposure to more than one blast event (the service member's commander shall direct a medical evaluation). Beginning with recognition, the policy outlines additional steps including, refer, report, rest, and return to duty.

Command Guidance

Recognize

Military leadership is required to recognize all personnel involved in any potentially concussive event, including those without apparent injuries, as soon as safely possible using the IED/HEADS checklist (Table 1).

Refer

Leaders refer any service member involved in a potentially concussive event to be evaluated by a medic or health care provider and if applicable, be re-evaluated and medically cleared before returning to duty.

Report

Military leaders are required to report all service members involved in a potentially concussive event by completing the significant activities (SIGACT) report within 24 h of the injury. Depending on the command, reports may be required using the Blast Exposure and Concussion Incident Report (BECIR) module, located

Battlefield Assessment, Table 1: IED/HEADS checklist

I-Injury	Was the service member injured during the event?	Yes/ no
E- evaluation	Are any of the "HEADS" symptoms present? H-headaches and/or vomiting? (yes/no) E-ears ringing? (yes/no) A-amnesia, altered/loss of consciousness? (yes/no) D-double vision and/or dizziness? (yes/no) S-something feels wrong or is not right? (yes/no)	Yes/ no
D-distance/ proximity	Was the service member within 50 m of the blast? Record distance from blast	Yes/ no

within the Combined Information Data Network Exchange (CIDNE).

Medical Guidance

Medical requirements include (1) utilization of the Military Acute Concussion Evaluation (MACE) for screening of a potentially concussive event, (2) documentation and appropriate International Classification of Diseases (ICD) coding of the medical encounter within the electronic health record, (3) and utilization of the "Concussion Management in Deployed Settings" algorithm (DCoE 2011; Fig. 1). The algorithm outlines three levels of intervention: (1) Level I: Combat Medic/Corpsman Concussion Triage, (2) Level II: Initial Management of Concussion in Deployed Setting, and (3) Level III: Comprehensive Concussion Evaluation.

Medical personnel are instructed to use section one of the MACE (Fig. 2), which is used to assess the service member's potential concussion/mild traumatic brain injury (mTBI). Questions on the MACE guide the provider to obtain a description of the incident, as well as assess of any alterations or losses of consciousness (AOC/LOC), and/or post-traumatic amnesia (PTA). Assessments are to be completed as close to the time of injury as feasible. The MACE is currently the primary instrument, used mostly by corpsmen/medics, to assess in-theater concussive events and determine the need for additional levels of care.

Rest

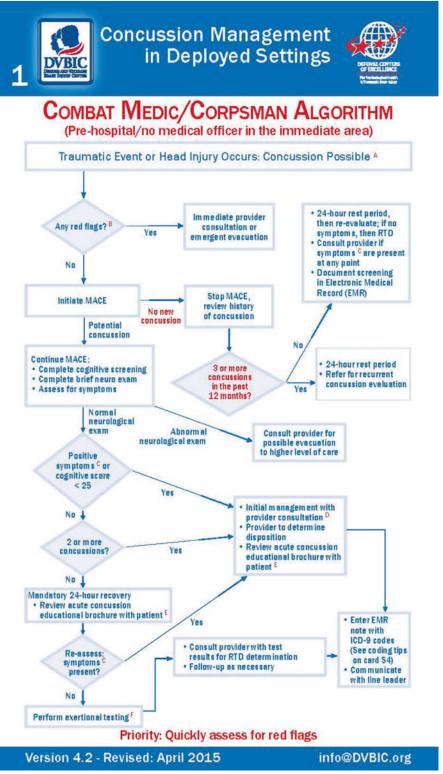
Service members must receive a minimum of 24 h of rest/downtime after a potentially concussive event. The 24-h clock starts at the time of the event, not at the time of the evaluation. Recovery care is to include sleep and pain management. As current research supports the return to neurocognitive baseline as soon as a few days post-injury, and many times without formal intervention (Mittenberg and Strauman 2000), the service member should remain in place for several days to allow for this recovery (Barth et al. 2010).

If the service member has been diagnosed with two concussions within the prior 12 months, the service member should receive seven additional days of rest after symptoms resolve. If three or more concussions have been diagnosed within the previous 12 months, the service member is mandated to receive a recurrent concussion evaluation before returning to duty. Of note, commanders may determine that mission requirements supersede an individual's welfare in certain circumstances and can waive or postpone the mandatory rest period.

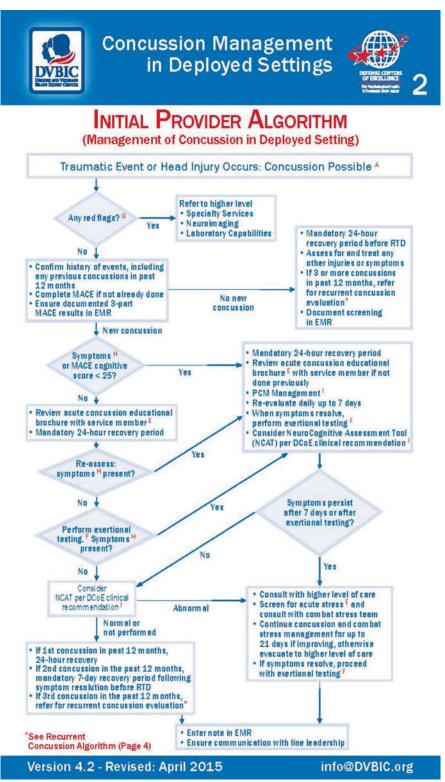
The recurrent concussion evaluation can be initiated at any time that it is clinically warranted and should be used to inform treatment and returnto-duty (RTD) decisions. Recurrent concussion evaluations are to include (1) neurological examination, including completion of the Neurobehavioral Symptom Inventory (NSI), a validated acute stress reaction assessment, and a vestibular assessment; (2) neuroimaging; (3) neuropsychological assessment; (4) functional assessment, including evaluation of cognitive, sensorimotor, and physical endurance; (5) and a duty status determination by a neurologist or other qualified licensed independent practitioner trained according to service policies in the care of mTBI/ concussion.

Regarding neuropsychological assessment, the Clinical Recommendation (DCoE 2011) indicates that a neurocognitive assessment tool (NCAT) should be considered if symptoms persist. In accordance with the National Defense Authorization Act (NDAA HR 4986), the DoD selected the Automated Neuropsychological Assessment Metrics (ANAM) as the NCAT to be utilized for predeployment baseline assessments and for postconcussion testing in theater.

In theater, clinical guidelines indicate that the ANAM test battery be administered if symptoms of concussion are present 24-h post-injury. The first administration of the test should be within 24–72 h from injury, and the test can be readministered on a regular basis to assess for cognitive deficits and symptom resolution (Kelly et al. 2012). To this end, the service member's post-injury test scores can be compared to his/her own baseline data, if possible, or to the normative database to determine recovery (DCoE 2011).

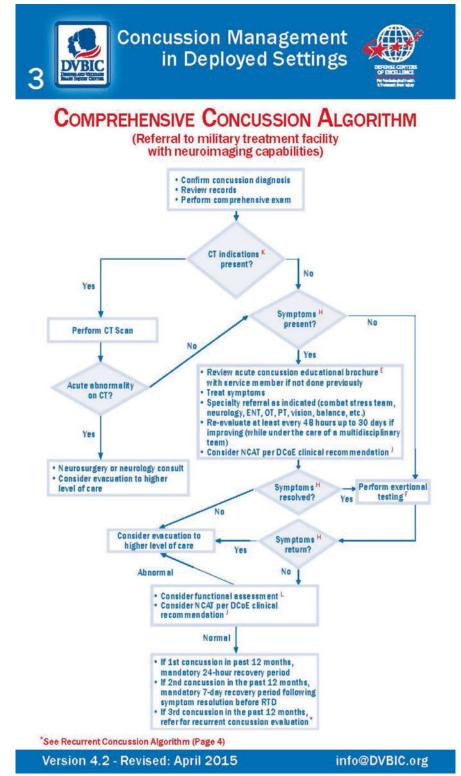


Battlefield Assessment, Fig. 1 (continued)

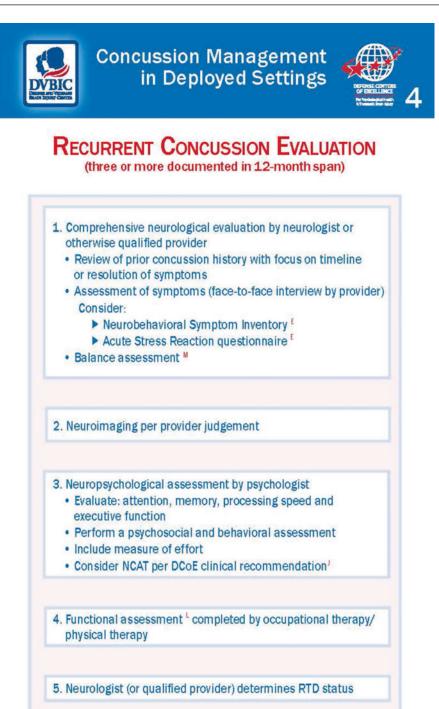


Battlefield Assessment, Fig. 1 (continued)

В



Battlefield Assessment, Fig. 1 (continued)



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Battlefield Assessment, Fig. 1 (continued)

В



Concussion Management in Deployed Settings



Traumatic Event or Head Injury Occurs: Concussion Possible

- ^A Mandatory Events Requiring Concussion Evaluation:
- 1. Any service member in a vehicle associated with a blast event, collision or rollover
- 2. Any service member within 50 meters of a blast (inside or outside)
- 3. Anyone who sustains a direct blow to the head
- 4. Command directed such as, but not limited to, repeated exposures

B Medic/Corpsman Algorithm Red Flags:

- 1. Witnessed loss of consciousness (LOC)
- 2. Two or more blast exposures within 72 hrs
- 3. Unusual behavior/combative
- 4. Unequal pupils
- 5. Seizures
- 6. Repeated vomiting

- 7. Double vision/loss of vision
- 8. Worsening headache
- 9. Weakness on one side of the body
- 10. Cannot recognize people or disoriented to place
- 11. Abnormal speech

^c Medic/Corpsman Algorithm Symptoms:

(Persisting beyond initial traumatic event)

- 1. Headache
- 2. Dizziness
- 3. Memory problems
- 4. Balance problems
- 5. Nausea/vomiting

- 6. Difficulty concentrating
- 7. Irritability
- 8. Visual disturbances
- 9. Ringing in the ears
- 10. Other___

^D Medic/Corpsman Initial Management of Concussion:

- 1. Give acute concussion educational
 - brochure to all concussion patients, available at: dvbic.dcoe.mil
- 2. Reduce environmental stimuli
- 3. Mandatory 24-hour recovery period
- E Available Resources (dvbic.dcoe.mil):
- Acute Stress Reaction Questionnaire
- Acute Concussion Educational Brochure
- Neurobehavioral Symptom Inventory

- 4. Aggressive headache management - Use acetaminophen q 6 hrs x 48 hrs
 - After 48 hours may use naproxen pm
- 5. Avoid tramadol, Fioricet, excessive triptans and narcotics
- Line Leader Fact Sheet
- Coding Guidance
- DCoE NeuroCognitive Assessment Tool (NCAT) Recommendation

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Battlefield Assessment, Fig. 1 (continued)



F Exertional Testing:

- Exert to 65-85% of target heart rate (THR=220-age) using push-ups, sit-ups, running in place, step aerobic, stationary bike, treadmill and/or hand crank
- 2. Maintain this level of exertion for approximately 2 minutes
- Assess for symptoms (headache, vertigo, photophobia, balance, dizziness, nausea, visual changes, etc.)
- 4. If symptoms/red flags exist with exertional testing, stop testing, and consult with provider

^G Provider Algorithm Red Flags:

- 1. Progressively declining level of consciousness
- 2. Progressively declining neurological exam
- 3. Pupillary asymmetry
- 4. Seizures
- 5. Repeated vomiting
- 6. Clinically verified GCS < 15
- 7. Neurological deficit: motor or sensory
- 8. LOC > 5 minutes
- 9. Double vision
- 10. Worsening headache
- 11. Cannot recognize people or
- disoriented to place
- 12. Slurred speech
- 13. Unusual behavior

^H Provider Algorithm Symptoms:

1. Confusion (24 hours)	4. Vertigo/dizziness	7. Phonophobia	
2. Irritability	5. Headache	8. Sleep issues	
3 linsteady on feet	6 Photophobia		

Primary Care Management (PCM):

- 1. Give acute concussion educational brochure to all concussion patients, available at: dvbic.dcoe.mil
- 2. Reduce environmental stimuli
- 3. Mandatory 24-hour recovery period
- 4. Aggressive headache management - Use acetaminophen q 6 hrs x 48 hrs
- After 48 hours may use naproxen prn 5. Avoid tramadol, Fioricet, excessive triptans
- and narcotics
- Consider nortriptyline q HS or amitriptyline q HS for persistent headache (> 7 days). Prescribe no more than 10 pills.

- 7. Implement duty restrictions
- Review current medications and sleep hygiene (Healthy Sleep fact sheet available at dvbic.dcoe.mil) and consider short-term low dose non-benzodiazepine hypnotic (e.g., zolpidem 5mg)
- 9. Pain management if applicable
- 10. Send consult to med.consult.army@mail.mil for further guidance if needed
- 11. Consider evacuation to higher level of care if clinically indicated
- 12. Document concussion diagnosis in EMR

med.consult.army@mail.mil is a Department of Defense email consultation service provided by the Army OTSG Telemedicine Teleconsultation Programs to assist deployed clinicians with the treatment of TBI and RTD decisions.

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Battlefield Assessment, Fig. 1 (continued)

В



Concussion Management in Deployed Settings



³ DCoE NeuroCognitive Assessment Tool (NCAT) Recommendation: Current DoD policy is that all service members must be tested with a neurocognitive assessment tool (NCAT) prior to deployment. Among several tests that are available, the DoD has selected the Automated Neuropsychological Assessment Metrics (ANAM) as the NCAT to use for both pre-deployment baseline testing and for post-concussion assessment in theater. Detailed instructions for administering a post-injury ANAM are provided at dvbic.dcoe.mil.

For ANAM baseline results send requests to: usarmy.jbsa.med.com.mbx.otsg--anam-baselines@mail.mil

K CT Indications:*

- 1. Physical evidence of trauma above the clavicles
- 2. Seizures
- 3. Vomiting
- 4. Headache

- 5. Age > 60
- 6. Drug or alcohol intoxication
- 7. Coagulopathy
- 8. Focal neurologic deficits

* Haydel MJ, Preston CA, Mills TJ, Luber S, Blaudeau E, DeBlieux PM. Indications for computed tom ography in patients with minor head injury. N Engl J Med. 2000 Jul 13;343(2):100-5.

^L Functional Assessment:

Assess the service member's performance of military-relevant activities that simulate the multi-system demands of duty in a functional context. Selected assessment activities should concurrently challenge specific vulnerabilities associated with mTBI including cognitive (such as executive function), sensorimotor (such as balance and gaze stability), and physical endurance. Rehabilitation providers should not only evaluate the service member's performance but also monitor symptoms before, during and after functional assessment.

M The Balance Error Scoring System (BESS - Modified):**

Stand on flat surface, eyes closed, hands on hips in 3 positions:

- 1. On both feet (20 seconds)
- 2. On one foot (20 seconds)
- 3. Heel-to-toe stance (20 seconds)

1. Stepping, stumbling or falling

For each position, score 1 point for any of the following errors:

4. Forefoot or heel lifted

- 2. Opening eyes
- 3. Hands lifted above the iliac crests
- Score 10 points if unable to complete

Total Balance Score

- 5. Hip moved > 30 degrees flexion or abduction
- 5. htp moveu > 50 degrees nexton or abduction
- 6. Out of test position > 5 seconds

Guskiewicz KM, Ross SE, Marshall SW. Postural Stability and Neuropsychological Deficits After Concussion in Collegiate Athletes. J Athl Train. 2001 Sep;36(3):263-273.

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Battlefield Assessment, Fig. 1 (continued)





2015 DoD Definition of Traumatic Brain Injury:

A traumatically induced structural injury or physiological disruption of brain function, as a result of an external force, that is indicated by new onset or worsening of at least one of the following clinical signs immediately following the event:

- Any alteration in mental status (e.g., confusion, disorientation, slowed thinking, etc.).
- · Any loss of memory for events immediately before or after the injury.
- · Any period of loss of or a decreased level of consciousness, observed or self-reported.

Coding Tips:

- Primary code (corpsman/medics require co-sign)
 - 850.0 Concussion without LOC
 - 850.11 Concussion with LOC ≤ 30 min.
- 2. Personal history of TBI in Global War on Terror (GWOT)
 - V15.52_2 Injury related to GWOT, mild TBI
- 3. Symptom codes
- As appropriate
- 4. Deployment status code
- V70.5_5 During deployment encounter
- 5. Screening code for TBI
 - V80.01
- 6. External cause of injury code (E-code)
 - E979.2 (if applicable) Terrorism involving explosions and fragments

Key Algorithm Directives:

· Personnel are required to use the algorithms to treat concussion in the deployed setting

- Mandatory event-driven protocols for exposure to potentially concussive events
 Requires a medical evaluation and minimum 24-hour rest period
- · All sports and activities with risk of concussion are prohibited until after a 24-hour rest period
- · Military Acute Concussion Evaluation (MACE) documentation will address all 3 MACE parts
- Service members diagnosed with concussion will be given the acute concussion educational brochure available at: dvbic.dcoe.mil
- Specific protocols for anyone sustaining ≥ 2 concussions within 12 months

MACE Documentation

Document using the mnemonic "CNS"

- (1) C Cognitive score
- (2) N Neurological exam reported as normal or abnormal
- (3) S Symptoms reported as present or absent

If a head injury event or AOC/LOC/PTA is not reported, then a concussion has not occurred. The MACE is stopped because the cognitive portion is not valid in non-concussed patients. Evaluate and treat any other symptoms or injuries, and document the event in the EMR. The MACE score should be reported as N/A.

Repeat MACE Tips:

Repeating the MACE's Cognitive Exam with a different version (A-F) may be used to evaluate acute concussion recovery; however, a physical exam and symptom assessment must accompany any repeated cognitive exam. Providers should be mindful of other factors affecting the MACE cognitive score such as sleep deprivation, medications or pain.

For additional copies or information call 1.866.966.1020 or email info@DVBIC.org

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Battlefield Assessment, Fig. 1 DVBIC concussion-management-algorithm pocket-cards version-4.2

	MI A Military Acut	C E le Concussion E	valuation	DVBIC DVBIC
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Patient Name:		
Service Member ID#:	Unit:	
Date of Injury:	Time of Injury:	_
Examiner:		_
Date of Evaluation:	Time of Evaluation:	_

CONCUSSION SCREENING

Complete this section to determine if there was both an injury event AND an alteration of consciousness.

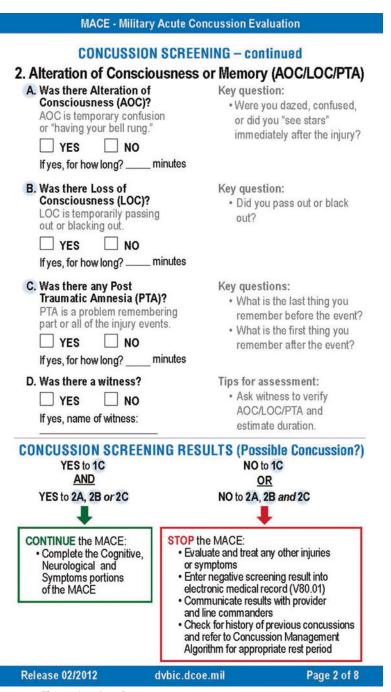
1. Description of Incident

A. Record the event as described by the service member or witness.

Jse	open-ended	questions	to get	as	much	detail	as	possible
-----	------------	-----------	--------	----	------	--------	----	----------

	Key questions: • Can you tell me what you remember? • What happened?
B. Record the type of event. Check all that apply: Explosion/Blast Fragment Blunt Object Sports Inju Fall Other	Motor Vehicle Crash
C. Was there a head injury event?	Key questions: • Did your head hit any objects? • Did any objects strike your head? • Did you feel a blast wave? (A blast wave that is felt striking the body/head is considered a blow to the head.)
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Battlefield Assessment, Fig. 2 (continued)



Battlefield Assessment, Fig. 2 (continued)

MACE - Military Acute Concussion Evaluation

COGNITIVE EXAM^a

3. Orientation

Score 1 point for each correct response.

Ask This Question	Incorrect	Correct
"What month is this?"	0	1
"What is the date or day of the month	?" 0	1
"What day of the week is it?"	0	1
"What year is it?"	0	1
"What time do you think it is?"	0	1
Correct response must be within 1	hour of actua	l time.

ORIENTATION TOTAL SCORE

4. Immediate Memory

Choose one list (A-F below) and use that list for the remainder of the MACE.

Read the script for each trial and then read all 5 words. Circle the response for each word for each trial. Repeat the trial 3 times, even if the service member scores perfectly on any of the trials.

Trial 1 Script:

 "I am going to test your memory. I will read you a list of words and when I am done, repeat back to me as many words as you can remember, in any order."

Trials 2 and 3 Script:

 "I am going to repeat that list again. Repeat back to me as many words as you can remember, in any order, even if you said them before."

	Tria	al 1	Tria	al 2	Tria	al 3
List F	Incorrect	Correct	Incorrect	Correct	Incorrect	Correct
Dollar	0	1	0	1	0	1
Honey	0	1	0	1	0	1
Mirror	0	1	0	1	0	1
Saddle	0	1	0	1	0	1
Anchor	0	1	0	1	0	1
Immediate I	Memory /	Alternat	e Word L	ists		/15
		_			tR	_
List E	List D	L	ist C	Lis	t B	List
List E Jacket	List D Finger	B	ist C laby	Lis Ca	ndle	List /
List E Jacket Arrow	List D Finger Penny	B N	ist C aby Ionkey	Lis Ca Pa	ndle per	List A Elbox Apple
List E Jacket Arrow Pepper	List D Finger Penny Blanket	B N P	ist C Jaby Nonkey Perfume	Lis Ca Pa Su	ndle per gar	List A Elbox Apple Carp
List E Jacket Arrow Pepper Cotton	List D Finger Penny	B M P S	ist C aby Ionkey	Lis Ca Pa Su Sa	ndle per	List A Elbox Apple

Battlefield Assessment, Fig. 2 (continued)

NEUF	ROLOGICAL EXAM
5. Eyes Test pupil response to light, tracking Normal Abnormal	 Tips for assessment: Pupils should be round, equal in size and briskly constrict to a direct, bright light. Both eyes should smoothly track your finger side-to-side and up and down.
6. Speech Test speech fluency and word finding Normal Abnormal	 Tips for assessment: Speech should be fluid and effortless – no pauses or unnatural breaks. Assess difficulties with word finding: Does service member have trouble coming up with the name of a common object?
7. Motor Test grip strength and pronator drift Normal Abnormal	 Tips for assessment: Assess grip strength. Assess for pronator drift for 5-10 seconds by directing patient to close eyes and extend arms forward, parallel to the ground with palms up: Does either palm turn inward? Does either arm drift down?
8. Balance Tandem Romberg Test Normal Abnormal	Tips for assessment: • Have patient stand with eyes closed, one foot in front of the other heel-to-toe, arms extended forward, palms up. Observe for 5-10 seconds: - Does the service member stumble or shift feet?
NEUROLOGICAL Exam results	All Normal Any Abnormal Green Red

Battlefield Assessment, Fig. 2 (continued)

MACE - Military Acute Concussion Evaluation

COGNITIVE EXAM^a - Continued

9. Concentration

A. Reverse Digits

Read the script and begin the trial by reading the first string of numbers in Trial 1.

Script:

 "I am going to read you a string of numbers. When I am finished, repeat them back to me backward. That is, in reverse order of how I read them to you. For example, if I said 7 - 1 - 9, then you would say 9 - 1 - 7."

Circle the response for each string.

- If correct on string length of Trial 1, proceed to the next longer string length in the same column.
- If incorrect on string length of Trial 1, move to the same string length of Trial 2.
- If incorrect on both string lengths in Trials 1 and 2, STOP and record score as zero for that string length. Record total score as sum of previous correct trials.

List F		
Trial 2 (if Trial 1 is incorrect)	Incorrect	Correct
4-7-9	0	1
3-9-2-4	0	1
8-3-9-6-4	0	1
3-1-7-8-2-6	0	1
	Trial 2 (if Trial 1 is incorrect) 4-7-9 3-9-2-4 8-3-9-6-4	Trial 2 (if Trial 1 is incorrect) Incorrect 4-7-9 0 3-9-2-4 0 8-3-9-6-4 0

Concentration Alternate Number Lists

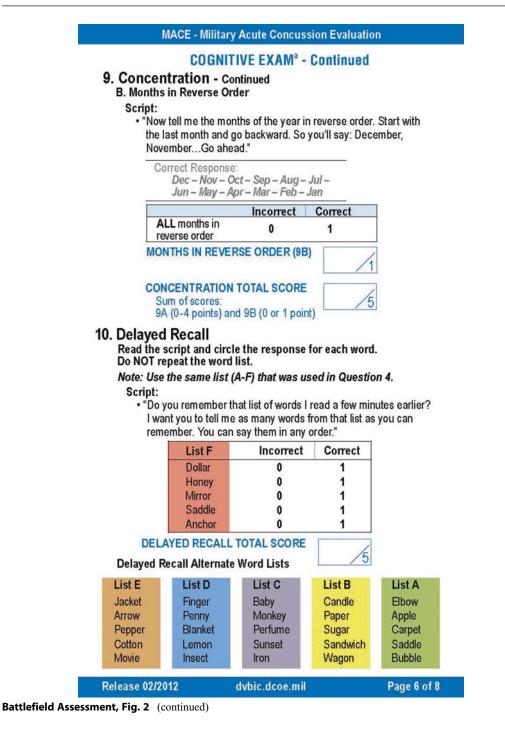
Note:	Use the	same list	t (A-F)	that was	used in	Question 4.
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4

List E		List	D				
	Trial 1	Trial 2	Trial 1	Trial 2			
	3-8-2	5-1-8	7-8-2	9-2-6			
	2-7-9-3	2-1-6-9	4-1-8-3	9-7-2-3			
	4-1-8-6-9	9-4-1-7-5	1-7-9-2-6	4-1-7-5-2			
	6-9-7-3-8-2	4-2-7-9-3-8	2-6-4-8-1-7	8-4-1-9-3-5			
	List	С	List	В	List A		
	Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2	
	1-4-2	6-5-8	5-2-6	4-1-5	4-9-3	6-2-9	
	6-8-3-1	3-4-8-1	1-7-9-5	4-9-6-8	3-8-1-4	3-2-7-9	
	4-9-1-5-3	6-8-2-5-1	4-8-5-2-7	6-1-8-4-3	6-2-9-7-1	1-5-2-8-5	
	3-7-6-5-1-9	9-2-6-5-1-4	8-3-1-9-6-4	7-2-7-8-5-6	7-1-8-4-6-3	5-3-9-1-4-8	
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Battlefield Assessment, Fig. 2 (continued)

1



MACE - N	Ailitary Acute Concussio	n Evaluation		
	SYMPTOM SCREENI			
_· ·	Check all that apply:			
Headache	Balance Problems	Irritability		
Dizziness	Nausea/Vomiting	Visual Disturbances		
Memory Problems	Difficulty Concentrating	Ringing in the Ears Other		
SUMMARY Record the data for corr	rect MACE documentation			
Cognitive Summary				
Orientation Total S	core - Q3	/5		
Immediate Memory	/ Total Score (all 3 trials) -	04 45		
-	•			
Concentration lota	al Score (Sections A and B	3) - Q9 <u>5</u>		
Delayed Recall Tot	al Score - Q10	5		
COGNITIVE RESULTS		60		
NEUROLOGICAL RES				
(Page 4)				
	Normal (Green)	Abnormal (Red)		
SYMPTOM RESULTS	(Green)			
	No sympt (A)	toms 1 or more symptoms (B)		
MACE RESULTS (Report all 3 parts.) Example: 24/Red/B Abnormality in any area should be discussed with provider. C C				
12. During the past 1 a concussion, no YES N If yes, how many?	RY IN PAST 12 MONTHS 2 months have you been t counting this event? IO			
A ST	Management Algorithm fo	-		
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Battlefield Assessment, Fig. 2 (continued)

MACE - Military Acute Concussion Evaluation

ADDITIONAL INFORMATION ABOUT MACE COGNITIVE SCORES

Although cognitive is listed first in the summary of MACE results, this should not suggest that any one of the three screening categories is more or less important than the others. Each area (Cognitive, Neurological, Symptoms) must be evaluated carefully. The results of all three evaluations must be included in any MACE report for it to be considered complete.

Regarding cognitive scores, in studies of non-concussed subjects, the mean total cognitive score was 28. Therefore, a score of < 30 does not imply that a concussion has occurred. Definitive normative data for a cut-off score are not available. The Concussion Management Algorithm stipulates that a cognitive score of < 25 or the presence of symptoms requires consultation with a provider.

Repeating the MACE cognitive exam with a different version (A-F) may be used to evaluate acute concussion recovery; however, a physical exam and symptom assessment must accompany any repeated cognitive exam. Providers should be mindful of other factors affecting the MACE cognitive score such as sleep deprivation, medications or pain.

Coding Tips for Concussion:
1. Primary code (corpsmen/medics require co-sign)
850.0 – Concussion without LOC
 850.11 – Concussion with LOC ≤ 30 min.
2. Personal history of TBI in Global War on Terror (GWOT)
 V15.52_2 – Injury related to GWOT, mild TBI
3. Symptom codes
As appropriate
4. Deployment status code
 V70.5_5 – During deployment encounter
5. Screening code
 V80.01 – Special screening for TBI code
6. E-code (external cause of injury)
 E979.2 (if applicable) – Terrorism involving
explosions and fragments
ferences
McCrea M Standardized Mental Status Testing on the Sideline After

 McCrea, M. Standardized Mental Status Testing on the Sideline After Sport-Related Concussion. J Athl Train. 2001 Sep;36(3):274-279.

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Battlefield Assessment, Fig. 2 DVBIC military-acute-concussion-evaluation pocket-card

Re

When the concussed service member is no longer reporting symptoms, physical exertion testing is recommended. Exertional testing is typically conducted by having the service member exert 65-85% of their target heart rate by engaging in aerobic activities (i.e., treadmill running, push-ups, or stationary biking), which is theorized to increase intracranial pressure and activate dormant symptoms (Barth et al. 2010). Immediately after exertion (after approximately 2 min), the medical provider assesses for the presence of any symptoms (i.e., headache, dizziness, vision changes, vertigo, etc.). If symptoms are present, then continued rest and observation is recommended, while those who remain asymptomatic may be considered for RTD.

Service members exhibiting symptoms of concussion for extended periods of time postinjury may require referral to a higher-level facility, where neuropsychological assessment in conjunction with a clinical examination and input from various disciplines (e.g., physical and occupational therapies) is strongly encouraged to determine return-to-duty (RTD) recommendations. After treatment in theater and/or via combat support hospital, the wounded service member requiring greater intensity of care is transported to Landstuhl Regional Medical Center (LRMC) in Germany, and then later to Walter Military Medical Reed National Center (WRNMMC) in Washington, D.C. From there, service members are assessed, treated, and transferred to other military and Veterans facilities Administration (VA) as needed (French et al. 2012).

Return to Duty

While recovering from TBI, service members should not return to duty or engage in other activities that place them at risk for concussion (i.e., sports, combatives, etc.). Military leaders are instructed to consult with medical personnel for RTD recommendations.

While service members may need to be removed from combat to ensure safety of the individual as well as of others, Barth et al. (2010) caution that clinicians must also take into consideration mission goals and iatrogenic effects of imposed rest:

Unit cohesion is an important component of military life, lengthy separations can reduce expectations of return to duty, and service members may become less likely to recover and return to full duty. Keeping people out of action or evacuating them so they can recover must be balanced with the negative expectations about not returning to duty that may arise when they are removed from their units. (Barth et al. 2010, pp. 135)

Historical Background

The assessment of mTBI can be challenging to diagnose as symptoms can vary from person to person and may not seem sufficiently "bad enough" for a service member or their command to discern that an injury has occurred. The Army Research Laboratory's report on the use of the ANAM for TBI assessment highlighted the importance of assessment:

Some service members who are determined to accomplish their mission and feel a strong desire to remain in-country and support their unit, may mask their symptoms (e.g., cognitive deficiency, chronic daily headaches) by simply not seeking care. The motivation not to leave one's fellow service members behind is strong, even when an individual has been injured, and perhaps even more so when the injured service member is uncertain whether their injury is real or an imagined set of symptoms related to stress. Undiagnosed mTBI can endanger not only the individual, but also the entire unit. A soldier's cognitive as well as physical and emotional deficits may not be evident until a mistake is made that could put both the service member and his or her team in jeopardy. (Rice et al. 2011, pp. 1)

In 2006, the Defense and Veterans Brain Injury Center and Brain Trauma Foundation in collaboration with academic experts developed Guidelines for the Field Management of Combat-Related Head Trauma. This guideline included the development of the Military Acute Concussion Evaluation (MACE) tool (French et al. 2008) and a decision tree for determining additional evaluation and treatment.

In 2007, mandatory concussion screening protocols (i.e., MACE) were implemented

throughout the DoD. Consequently, this led to abrupt increases in the reporting of TBI in the deployed setting as well as across military installations, both before and after deployment (Helmick et al. 2015). Research has also consistently shown the majority of TBI occurs in nondeployed settings secondary to military training accidents, auto accident (private and military vehicles), falls, sports, and recreational activities. However, many of those injuries diagnosed postdeployment have been found to be deploymentrelated events that were diagnosed weeks or months after return from deployment (Scholten et al. 2012). Prior to DoD instruction on concussion management in the deployed setting, the identification of concussion was somewhat challenging as the military culture traditionally discouraged admitting to being injured, so that the service member could "get back to the fight."

In 2010, the DoD responded to this trend by implementing an incident-based system of reporting (DoDi 6490.11) in which command leaders were required to remove service members from combat and report all service members who had been exposed to a potentially concussive event (DoD 2012). This policy, as described in the description in the above section, has reportedly increased awareness of mTBI and consequently decreased the stigma, which will optimistically mitigate any potential long-term effects of second-ary to mTBI (Helmick et al. 2015).

See Also

- Automated Neuropsychological Assessment Metrics (ANAM)
- ► Blast Effects
- Concussion
- Defense and Veterans Brain Injury Center
- Mild Traumatic Brain Injury
- Military Acute Concussion Evaluation

References and Readings

Barth, J., Isler, W., Helmick, K., Wingler, I., & Jaffee, M. (2010). Acute battlefield assessment of concussion/ mild TBI and return-to-duty evaluations. In C. H. Kennedy & J. Moore (Eds.), *Military neuropsychology* (pp. 127–174). New York: Springer.

- Bryan, C., & Hernandez, A. M. (2012). Magnitudes of decline on automated neuropsychological assessment metrics subtest scores relative to predeployment baseline performance among service members evaluated for traumatic brain injury in Iraq. *The Journal of Head Trauma Rehabilitation*, 27(1), 45–54. https://doi.org/ 10.1097/HTR.0b013e318238f146.
- Defense and Veterans Brain Injury Center. In *DoD worldwide numbers for TBI*. Retrieved from http://Dvbic. Dcoe.Mil/Dod-Worldwide-Numbers-TBi
- Defense and Veterans Brain Injury Center. (2012, November). Policy guidance for management of mild traumatic brain injury/concussion in the deployed setting (line leader fact sheet). Retrieved from http://dvbic.Dcoe. Mil/files/line-leader-fact-sheet-2012 V3 Final.Pdf
- Defense Centers of Excellence (DCoE) for Psychological Health and Traumatic Brain Injury. (2011, May). *Indications and conditions for in-theater post-injury neurocognitive assessment tool (NCAT) testing* (DCoE clinical recommendation). Retrieved from http://dvbic. Dcoe.Mil/files/resources/DCoE_clinical_Recommen dations post injury NCAT 05-31-2011 f.Pdf
- Department of Defense. (2008). National defense authorization act for fiscal year 2008 (HR 4986, section 1673). Retrieved from https://www.Congress.Gov/Bill/110thcongress/house-bill/4986
- Department of Defense. (2012, September). *DoD policy* guidance for management of mild traumatic brain injury/concussion in the deployed setting (DoDi 6490.11). Retrieved from http://www.Dtic.Mil/whs/ directives/corres/pdf/649011p.Pdf
- Eckner, J. T., Kutcher, J. S., Broglio, S. P., & Richardson, J. K. (2014). Effect of sport-related concussion on clinically measured simple reaction time. *British Journal of Sports Medicine*, 48(2), 112–118. https://doi.org/ 10.1136/bjsports-2012-091579.
- French, L., McCrea, M., & Baggett, M. (2008). The military acute concussion evaluation (MACE). *Journal of Special Operations Medicine*, 8(1), 68–77.
- French, L. M., Anderson-Barnes, V., Ryan, L. M., Zazeckis, T. M., & Harvey, S. (2012). Neuropsychological practice in the military. In C. H. Kennedy & E. A. Zillmer (Eds.), *Military psychology: Clinical and operational applications* (2nd ed., pp. 185–210). New York: Guilford Press.
- Helmick, K. M., Spells, C. A., Malik, S. Z., Davies, C. A., Marion, D. W., & Hinds, S. R. (2015). Traumatic brain injury in the US military: Epidemiology and key clinical and research programs. *Brain Imaging and Behavior*, 9, 358–366. https://doi.org/10.1007/ s11682-015-9399-z.
- Joint Mental Health Advisory Team 7 (J-MHAT 7). (2011). Report of the advisory team on *Operation Enduring Freedom 2010 Afghanistan*. Retrieved from http:// armymedicine.Mil/documents/J_MHAT_7.pdf
- Kelly, M. P., Coldren, R. L., Parish, R. V., Dretsch, M. N., & Russell, M. L. (2012). Assessment of acute

concussion in the combat environment. *Archives of Clinical Neuropsychology*, 27(4), 375–388. https://doi.org/10.1093/arclin/acs036.

- Martin, E. M., Lu, W. C., Helmick, K., French, L., & Warden, D. (2008). Traumatic brain injuries sustained in the Afghanistan and Iraq wars. *American Journal of Nursing*, 108(4), 40–47. https://doi.org/10.1097/01. NAJ.0000315260.92070.3f.
- Mittenberg, W., & Strauman, S. (2000). Diagnosis of mild head injury and the postconcussion syndrome. *The Journal of Head Trauma Rehabilitation*, 15(2), 783–791. Retrieved from http://journals.Lww.Com/ headtraumarehab/abstract/2000/04000/Diagnosis_of_ mild Head injury and the.3.Aspx.
- Norris, J. N., Carr, W., Herzig, T., Labrie, D. W., & Sams, R. (2013). ANAM4 TBI reaction time-based tests have prognostic utility for acute concussion. *Military Medicine*, 178(7), 767–774. https://doi.org/10.7205/ MILMED-D-12-00493.
- Rice, V. J., Lindsay, G., Overby, C., Jeter, A., Alfred, P. E., Boykin, G. L., & Bateman. R. (2011, July). Automated neuropsychological assessment metrics (ANAM) traumatic brain injury (TBI): Human factors assessment (Army Research lab report ARL-TN-0440). Retrieved from http://www.Dtic.Mil/Cgi-bin/GetTRDoc?AD= ADA549141
- Scholten, J. D., Sayer, N. A., Vanderploeg, R. D., Bidelspach, D. E., & Cifu, D. X. (2012). Analysis of US veterans health administration comprehensive evaluations for traumatic brain injury in operation enduring freedom and operation Iraqi freedom veterans. *Brain Injury*, 26(10), 1177–1184. https://doi.org/10.3109/ 02699052.2012.661914.

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 and 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 and 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 Pharmaceuticals (1993)
- 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 attorneyneuropsychologists 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 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 considered to be the reference standard for developmental assessment. It is an individually administered test, applicable from 1 to 42 months of age. The 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 1700 children (divided into 17 age groups) and development is 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 can be 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 first eight items yield a Sensory Processing Score. The Adaptive Behavior scale is composed of items from the Parent/Primary Caregiver Form of the Adaptive Behavior Assessment System-Second Edition (Harrison and 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 such as the California Scales 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, administered to infants over the first $2\frac{1}{2}$ 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 (100), the SD was now 15. When compared to the original BSID, the BSID-II MDI scores were 12 points lower and the PDI was10 points lower. 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 Part C requirements for estimates of 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, 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 0.86 to 0.93; the intercorrelation between Cognitive and Language composites is 0.52, for Cognitive and Motor composites, 0.50, and the intercorrelation between Language and Motor composites is 0.49. Growth scores are provided 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 0.71, the Motor Composite and the BSID-II PDI = 0.60, and the Cognitive Composite and the BSID-II MDI = 0.60. However, in contrast to the expected Flynn effect, the Bayley-III Mental and Motor composite scores, on average, are approximately seven points *higher* than the corresponding BSID-II MDI and PDI. This phenomenon has also been reported with other developmental tests and has been termed the "reverse Flynn effect."

The increase in scores may be due to inclusion of infants at risk for developmental delay in the standardization sample (10%). These "mixed norms" inflate scores and decrease diagnostic accuracy. In addition, there are concerns regarding a weak test floor. However, despite at-risk infants receiving "normal" scores, these still are below comparison group scores, which typically are higher than the standardization mean (as seen in comparison of preterm infants and term controls). This conundrum becomes further complicated by the possibility that the Bayley-II was too conservative and underestimated development.

Clinical Uses

Administration of the BSID-III yields quantitative and qualitative data that provide 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. There also are concerns that the Bayley-III under-identifies children who could qualify for intervention services. 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 (e.g., >90 min at 13 months) and this is problematic when testing infants and toddlers. 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

- Aylward, G. P. (2013). Continuing issues with the Bayley-III: Where to go from here. *Journal of Developmental* and Behavioral Pediatrics, 9, 697–701.
- Bayley, N. (1969). The Bayley scales of infant development. San Antonio: The Psychological Corporation.
- Bayley, N. (1993). The Bayley scales of infant development (2nd ed.). San Antonio: The Psychological Corporation.
- Bayley, N. (2006). Bayley scales of infant and toddler development (3rd ed.). San Antonio: The Psychological Corporation.

- Greenspan, S. I. (2004). Greenspan social-emotional growth chart. A screening questionnaire for infants and young children. San Antonio: Harcourt Assessment.
- Harrison, P. L., & Oakland, T. (2003). Adaptive behavior assessment system (2nd ed.). San Antonio: The Psychological Corporation.
- Weiss, L. G., Oakland, T., & Aylward, G. P. (Eds.). (2010). Bayley III: Clinical use and interpretation. Boston: Elsevier.

Beck Anxiety Inventory

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Description

The Beck Anxiety Inventory (BAI) (Beck et al. 1988; Beck and Steer 1993) is a 21-item inventory which identifies anxiety symptoms and quantifies their intensity. Respondents are asked to rate how much they have been bothered by each item over the past week, including today, on a four-point scale ranging from 0 ("not at all") to 3 ("severely – I could barely stand it"). Items are summed, resulting in a total score ranging from 0 to 63, with higher scores representing greater levels of anxiousness (Table 1).

This assessment generally takes adults 5–10 min to complete. It can be self-report or interviewer administered. Traditionally, this assessment is administered in a paper-and-pencil or interview formats, but it is also increasingly given via the computer.

Beck Anxiety Inventory, Table 1 Anxiety categorization by score (Beck and Steer 1993)

Anxiety level	Minimal	Mild	Moderate	Severe
BAI score	0–7	8-15	16–25	26-63

Historical Background

The BAI was designed to assess anxiety symptoms independent of depressive symptoms. Authors compiled a pool of 86 items from three preexisting anxiety symptom checklists (the Anxiety Checklist (Beck et al. 1985), the Physician's Desk Reference Checklist (Beck 1978), and the Situational Anxiety Checklist (Beck 1982)). They eliminated repetitive items, conducted successive iterative principal factor analyses, and completed a series of validity analyses to whittle the item list down to 21. This final 21-item BAI was found to have high internal consistency. It also demonstrated both convergent validity with non-symptom constructs theoretically associated with anxiety and discriminant validity with those constructs associated with depression among a psychiatric outpatient population.

Psychometric Data

In the years since its original publication, the BAI has consistently shown good reliability in a variety of both clinical and nonclinical populations. A meta-analysis by de Ayala et al. (2005) found the average coefficient alpha to be 0.91. Test-retest values showed significantly more heterogeneity, ranging from 0.35 to 0.83, with the greatest variability nonpsychiatric among noncollege populations. Given increasing use of computerbased assessment administration, it is critical to consider the impact of the mode of administration on the psychometrics of questionnaires. Preliminary studies evaluating effect of administration mode found comparable internal consistencies but lower mean scores when the BAI was administered via the internet compared to paper-andpencil versions (Carlbring et al. 2007). This suggests that it may be necessary to use "internet norms" when administering the BAI in a computer format.

The BAI shows strong convergent validity with anxiety symptom self-report instruments, clinical ratings, and diaries. It has also shown moderate discriminant validity with measures of other types of psychopathology in both clinical and nonclinical samples (Steer 2009). Discriminant validity with depression symptoms is more variable based on populations, with lower discriminant validity among older adults (Morin et al. 1999; Wetherell and Gatz 2005) and nonclinical Spanish speakers (Magán et al. 2008). Scores on the BAI are linearly related to depression scales; however, individual items from these assessments have a strong tendency to load onto different factors (Beck et al. 1988).

Among clinical populations, factor analytic studies generally support a two-factor structure, with one factor representing cognitive symptoms of anxiety and the other representing somatic symptoms (Wilson et al. 1999). Among nonclinical populations, the factor structure is more varied, with evidence to support four (subjective, neurophysiological, autonomic, and panic (Osman et al. 1993)), five (subjective fear, somatic neurophysiological, nervousness, muscular/ motoric, respiration (Borden et al. 1991)), and six (somatic, fear, autonomic hyperactivity, panic, nervousness, and motor tension (Morin et al. 1999)) factor models. The broad categories of subjective and physiological symptoms still apply, but findings suggest that at nonclinical levels of anxiety, respondents may experience more nuanced physiological symptoms of anxiety. Given the lack of consensus in the literature regarding factor structure, the use of the total score remains the recommended approach for measuring anxiety symptoms with this scale (Steer 2009).

Clinical Uses

Overall, the BAI's strongest qualities are its ability to assess panic symptomology and distinguish between panic disorder and non-panic disorder symptom profiles (Leyfer et al. 2006). It is sensitive to changes in anxiety symptoms both in psychiatric (Brown et al. 1997) and medical populations (Lee et al. 2010). Because of its brevity and ease of administration, the BAI is commonly used as an anxiety screening instrument. However, the BAI is not a diagnostic measure, and research suggests it has limited utility when used in isolation as a measure of anxiety (Manne et al. 2001; Hoyer et al. 2002).

In addition to its use with general clinical populations, the BAI has demonstrated utility in neuropsychological populations as well. 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 (Zhou et al. 2013; Cantor et al. 2005). The BAI has been used to assess anxiety among Veterans with a history of TBI and was found to be associated with increased neuropsychiatric symptoms (King et al. 2012). It has also been used to assess anxiety poststroke (Baker-Collo 2007).

While the BAI has been used often with medical and neuropsychiatric populations, research suggests that there may be some overlap with somatic symptoms, which would be potentially problematic in a medical setting. As the BAI was developed to assess anxiety independent of depression, it excludes many anxiety symptoms which overlap with depression. It has been criticized for placing too heavy emphasis on somatic symptoms of anxiety, which may be more characteristic of panic as opposed to the overall construct of anxiety. Of the 21 items, 14 assess somatic symptoms, and patients with panic disorder have been shown to score higher on the BAI (Leyfer et al. 2006). Because of its emphasis on somatic symptoms, the BAI has less utility in populations with greater medical illnesses (such as older adults Wetherell and Gatz 2005). These populations endorse more physical complaints, which results in inflated scores on the BAI. Providers should use caution when using the BAI as a broad anxiety screening tool, particularly with populations with increased medical complications.

Diversity Considerations

Internal reliability is comparable between the genders; however, women consistently score higher on the BAI than men (Beck and Steer 1993; Hewitt and Norton 1993; Osman et al. 1993; Morin et al. 1999; Vázquez-Morejón et al. 2014). This gender difference remains even after differential item analysis identified and removed potentially biased items (Magán et al. 2008). These findings are consistent with lifetime prevalence data, which suggests that women have higher rates of anxiety (Kessler et al. 2005).

While the BAI is one of the most widely used tool for measuring anxiety symptomology, there is little research regarding its use in ethnic minority populations. An initial study examining the factor structure of the BAI for African Americans (Chapman et al. 2009) found that the originally proposed two factor structure did not hold for an African American nonclinical sample. Rather, they proposed an alternative two factor model with more items loading onto the somatic factor. Examination of the psychometric properties of the BAI in Latino populations (Contreras et al. 2004) supported the original factor structure and found that the BAI had strong internal reliability. Of note, nonclinical Latino participants' average scores were within normal ranges but significantly higher than Caucasian American populations.

The BAI has been translated and validated in several other languages, including Spanish (Fernández and Navarro 2003), French (Freeston et al. 1994), Turkish (Ulusoy et al. 1998), Norwe-(Nordhagen 2001), gian and Icelandic (Sæmundsson et al. 2015). While there are some reported differences in factor structures, the overall findings suggest comparable psychometric properties to the English version of the BAI. Findings from studies examining the utility of the BAI in the international community have varied, with some finding comparable normative values and others showing significant variability between cultures (Pillay et al. 2001; Hoge et al. 2006). The BAI has demonstrated some cross-cultural utility, but it should continue to be used with caution in diverse settings.

See Also

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► Anxiety
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References

- Ashman, T. A., Cantor, J. B., Gordon, W. A., Spielman, L., Flanagan, S., Ginsberg, A., Engmann, C., Egan, M., Ambrose, F., & Greenwald, B. (2009). A randomized controlled trial of sertraline for the treatment of depression in persons with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 90(5), 733–740.
- Barker-Collo, S. L. (2007). Depression and anxiety 3 months post stroke: Prevalence and correlates. *Archives* of Clinical Neuropsychology, 22(4), 519–531.
- 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., & Steer, R. A. (1993). Beck anxiety inventory manual. San Antonio: Psychological Corporation.
- Beck, A. T., Steer, R. A., & Brown, G. (1985). Beck anxiety checklist. Unpublished manuscript, University of Pennsylvania.
- 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(6), 893.
- 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(4), 345–356.
- Brown, G. K., Beck, A. T., Newman, C. F., Beck, J. S., & Tran, G. Q. (1997). A comparison of focused and standard cognitive therapy for panic disorder. *Journal* of Anxiety Disorders, 11(3), 329–345.
- Cantor, J. B., Ashman, T. A., Schwartz, M. E., Gordon, W. A., Hibbard, M. R., Brown, M., Spielman, L., Charatz, H. J., & Cheng, Z. (2005). The role of self-discrepancy theory in understanding post-traumatic brain injury affective disorders: A pilot study. *The Journal of Head Trauma Rehabilitation*, 20(6), 527–543.
- Carlbring, P., Brunt, S., Bohman, S., Austin, D., Richards, J., Öst, L. G., & Andersson, G. (2007). Internet vs. paper and pencil administration of questionnaires commonly used in panic/agoraphobia research. *Computers in Human Behavior*, 23(3), 1421–1434.
- Chapman, L. K., Williams, S. R., Mast, B. T., & Woodruff-Borden, J. (2009). A confirmatory factor analysis of the Beck anxiety inventory in African American and European American young adults. *Journal of Anxiety Disorders*, 23(3), 387–392.
- Contreras, S., Fernandez, S., Malcarne, V. L., Ingram, R. E., & Vaccarino, V. R. (2004). Reliability and validity of the Beck depression and anxiety inventories in Caucasian Americans and Latinos. *Hispanic Journal of Behavioral Sciences*, 26(4), 446–462.
- de Ayala, R. J., Vonderharr-Carlson, D. J., & Kim, D. (2005). Assessing the reliability of the Beck anxiety inventory scores. *Educational and Psychological Measurement*, 65(5), 742–756.

- Fernández, J. S., & Navarro, M. E. (2003). Propiedades psicométricas de una versión española del Inventario de Ansiedad de Beck (BAI) en estudiantes universitarios. *Ansiedad y Estrés*, 9(1), 59–84.
- Freeston, M. H., Ladouceur, R., Thibodeau, N., & Gagnon, F. (1994). L'inventaire d'anxiété de Beck. Propriétés psychométriques d'une traduction française. L'Encéphale: Revue de psychiatrie clinique biologique et thérapeutique.
- Hewitt, P. L., & Norton, G. R. (1993). The Beck anxiety inventory: A psychometric analysis. *Psychological Assessment*, 5(4), 408.
- Hoge, E. A., Tamrakar, S. M., Christian, K. M., Mahara, N., Nepal, M. K., Pollack, M. H., & Simon, N. M. (2006). Cross-cultural differences in somatic presentation in patients with generalized anxiety disorder. *The Journal of Nervous and Mental Disease*, 194(12), 962–966.
- Hoyer, J., Becker, E. S., Neumer, S., Soeder, U., & Margraf, J. (2002). Screening for anxiety in an epidemiological sample: Predictive accuracy of questionnaires. *Journal of Anxiety Disorders*, 16(2), 113–134.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62 (6), 593–602.
- King, P. R., Donnelly, K. T., Donnelly, J. P., Dunnam, M., Warner, G., Kittleson, C. J., Bradshaw, C. B., Alt, M., & Meier, S. T. (2012). Psychometric study of the neurobehavioral symptom inventory. *Journal of Rehabilitation Research and Development*, 49(6), 879.
- Lee, Y. W., Park, E. J., Kwon, I. H., Kim, K. H., & Kim, K. J. (2010). Impact of psoriasis on quality of life: Relationship between clinical response to therapy and change in health-related quality of life. *Annals of Dermatology*, 22(4), 389–396.
- Leyfer, O. T., Ruberg, J. L., & Woodruff-Borden, J. (2006). Examination of the utility of the Beck anxiety inventory and its factors as a screener for anxiety disorders. *Journal of Anxiety Disorders*, 20(4), 444–458.
- Magán, I., Sanz, J., & García-Vera, M. P. (2008). Psychometric properties of a Spanish version of the Beck Anxiety Inventory (BAI) in general population. *The Spanish Journal of Psychology*, 11(02), 626–640.
- Manne, S., Nereo, N., DuHamel, K., Ostroff, J., Parsons, S., Martini, R., Williams, S., Mee, L., Sexson, S., Lewis, J., Vickberg, S. J., & Redd, W. H. (2001). Anxiety and depression in mothers of children undergoing bone marrow transplant: Symptom prevalence and use of the Beck Depression and Beck anxiety inventories as screening instruments. *Journal of Consulting and Clinical Psychology*, 69(6), 1037.
- 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(1), 19–29.

- Nordhagen, T. (2001). Beck anxiety inventory: Translation and validation of a Norwegian version (Master's thesis, The University of Bergen).
- Osman, A., Barrios, F. X., Aukes, D., Osman, J. R., & Markway, K. (1993). The Beck anxiety inventory: Psychometric properties in a community population. *Journal of Psychopathology and Behavioral Assessment*, 15 (4), 287–297.
- Pillay, A. L., Edwards, S. D., Sargent, C., & Dhlomo, R. M. (2001). Anxiety among university students in South Africa. *Psychological Reports*, 88(3c), 1182–1186.
- Sæmundsson, B. R., Þórsdóttir, F., Kristjánsdóttir, H., Ólason, D. Þ., Smári, J., & Sigurðsson, J. F. (2015). Psychometric properties of the Icelandic version of the Beck anxiety inventory in a clinical and a student population. *European Journal of Psychological Assessment*, 27, 133–141.
- Steer, R. A. (2009). Amount of general factor saturation in the Beck anxiety inventory responses of outpatients with anxiety disorders. *Journal of Psychopathology* and Behavioral Assessment, 31(2), 112–118.
- Ulusoy, M., Sahin, N. H., & Erkmen, H. (1998). Turkish version of the Beck anxiety inventory: Psychometric properties. *Journal of Cognitive Psychotherapy*, 12(2), 163–172.
- Vázquez Morejón, A. J., Vázquez-Morejón Jiménez, R., & Zanin, G. B. (2014). Beck anxiety inventory: Psychometric characteristics in a sample from the clinical Spanish population. *The Spanish Journal of Psychol*ogy, 17, E76.
- Wetherell, J. L., & Gatz, M. (2005). The Beck anxiety inventory in older adults with generalized anxiety disorder. *Journal of Psychopathology and Behavioral Assessment*, 27(1), 17–24.
- Wilson, K. A., De Beurs, E., Palmer, C., & Chambless, D. (1999). Beck anxiety inventory. *The use of psychological testing for treatment planning and outcomes assessment*, 2, 971–992.
- Zhou, Y., Kierans, A., Kenul, D., Ge, Y., Rath, J., Reaume, J., Grossman, R. I., & Lui, Y. W. (2013). Mild traumatic brain injury: Longitudinal regional brain volume changes. *Radiology*, 267(3), 880–890.

Beck Depression Inventory

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Synonyms

BDI; BDI-II

Description

The Beck Depression Inventory (BDI; Beck et al. 1961) is one of the most widely used self-report measures to assess depressive symptom severity in adolescents and adults. It was amended in 1979 to allow for simpler administration and scoring (BDI-IA; Beck et al. 1979). In 1996, a more substantial revision was made so it would correspond to the Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition (DSM-IV; American Psychiatric Association 1994) criteria for clinical depression (BDI-II; Beck et al. 1996b).

The BDI-II is a self-report measure comprised of 21 items reflecting specific cognitive, affective, and physical symptoms of depression. Scores range from 0 to 3, with higher numbers indicating greater symptom severity. If more than one statement from a given item is chosen by the patient, the statement of greatest severity is scored. The maximum total score is 63.

The BDI-II takes approximately 5–10 min to complete and can be administered to individuals 13–80 years old. It is typically self-administered, although if clinically indicated, the examiner may read the items to the individual.

Historical Background

The original BDI (Beck et al. 1961) was developed with the use of descriptors provided by clinicians and patients with depression. These descriptors were then consolidated into 21 items. Directions for the assessment instructed the individual to choose the descriptor that best described how they felt at the moment. Originally, it was a measure administered by the examiner reading the statements to the examinee. Revisions on the BDI-IA (1979) turned it into a self-report, pencil-and-paper measure, and made the instrument easier to understand, such as by removing double negatives. In total, 15 of the original 21 items were modified. Additionally, directions were revised to instruct the individual to choose the descriptor that best described how they have felt in the past week, including the current day. A criticism of the BDI-IA, however, was that it only addressed six

Therefore, the measure was again revised. The BDI-II reworded select statements and introduced items to assess agitation, worthlessness, concentration difficulties, and loss of energy. It also included questions to reflect *increases* in sleep and appetite. Furthermore, items assessing body image, work difficulty, weight loss, and somatic preoccupations were removed, and the directions were modified so that the individual was instructed to choose the descriptor that best described how they have felt *in the past 2 weeks*. These changes improved correspondence to DSM-III-R (1987) and-DSM-IV (1994) criteria for clinical depression.

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). This is consistent with later independent study samples, which have found alphas ranging from 0.86 to 0.93 when assessing nonclinical populations, including high school-aged students (ages 14–18 years; Osman et al. 2008), undergraduates (ages 17–39 years; Storch et al. 2004), and older adults (ages 59–90 years; Segal et al. 2008), as well as clinical samples, including adolescents (ages 13–17 years; Osman et al. 2004) and adults (mean age = 37.6 years; Beck et al. 1996a).

Test-retest reliability of the BDI-II was assessed by Beck and colleagues (1996b), yielding correlation coefficients of 0.92 in a sample of 26 psychiatric outpatients, and 0.93 for their sample of college students.

Convergent and discriminant validity of the BDI-II was demonstrated by Beck and colleagues (1996b), who assessed 87 psychiatric outpatients with the BDI-II, Hamilton Rating Scale for

Depression (HRSD; Hamilton 1960), and Hamilton Rating Scale for Anxiety (HAM-A; Hamilton 1959). The BDI-II was more positively correlated with the HRSD (r = 0.71) than the HAM-A (r = 0.47).

Factor validity of the BDI-II was also assessed by Beck and colleagues (1996b), who found a two-factor solution using their outpatient standardization sample. Dozois et al. (1998) suggested that, overall, "somatic-affective" and "cognitive" are the two factors that tend to emerge in clinical samples, whereas "cognitive-affective" and "somatic" are the two factors that tend to emerge in nonclinical samples.

Overall, the BDI-II has demonstrated good internal consistency, test-retest reliability, convergent and discriminant validity, and factor validity.

Clinical Uses

The purpose of the BDI-II is to measure depression symptom severity. It can be used as part of a diagnostic battery or as a repeated measure to track treatment efficacy. Additionally, clinicians are advised to be aware that a score of "2" or "3" on item 2 (pessimism) or 9 (suicidal thoughts or wishes) is associated with greater risk for suicide (Beck et al. 1996b). Overall, the manual designates the following total raw score classifications for depression severity: 0-13 = minimal, 14-19 = mild, 20-28 = moderate, and>29 = severe. However, the instrument's developers suggest that different cutoff scores may be required depending on the characteristics of the sample and the purpose for using the instrument (e.g., lower thresholds for greater sensitivity in identifying depression, greater thresholds for greater specificity, such as in research). Other factors to consider when interpreting the BDI-II include individual characteristics, such as ethnic and cultural background, gender, age, and presence of additional medical conditions.

Given the ethnic and cultural diversity of psychiatric and neurological patient populations, there is a need for linguistically diverse and culturally sensitive psychiatric inventories. The BDI-II has been translated into numerous languages including Arabic (e.g., Hamdi et al. 1988), Chinese (e.g., Wu and Chang 2008), Dutch (e.g., Roelofs et al. 2013), Japanese (e.g., Kojima et al. 2002), Korean (e.g., Hong and Wong 2005), Portuguese (e.g., Gomes-Oliveira et al. 2012), Spanish (e.g., Gonzalez et al. 2015; Wiebe and Penley 2005), Turkish (e.g., Canel-Çınarbaş et al. 2011), and Xhosa (e.g., Edwards and Steele 2008). Some studies have suggested that BDI-II responding in different languages and cultures may have different psychometric properties. For example, studies have found that a three-factor model (instead of a two-factor model) may emerge among some ethnic groups (e.g., Mexicans: Gonzalez et al. 2015) or more ethnically diverse groups (e.g., ethnically diverse group of college students: Carmody 2005; Whisman et al. 2013); other studies, however, have confirmed the two-factor model (e.g., Chinese-heritage and European-heritage college students in Canada: Dere et al. 2015; Japanese: Kojima et al. 2002). Nevertheless, overall, studies have shown that the psychometric properties of the BDI-II in other languages and cultures are often comparable to that of the English version. Clinicians utilizing a translated measure should be aware of the different ways diverse groups may describe and experience symptoms of depression. For example, although Canel-Çınarbaş et al. (2011) found many of the psychometric properties of the Turkish version of the BDI-II to be comparable to its English counterpart, they noted that the Turkish word for depression connotes somatic symptoms such as "bodily tightness"; consistent with this, somatic symptoms were more likely be endorsed in this population.

Some studies have found gender differences in reporting on the BDI-II. Beck and colleagues (1996b) found that, among their sample of 500 outpatients, there was a significant mean difference among sexes, with women having higher overall scores than men (23.61 (SD = 12.31) for females, 20.44 (SD = 13.28) for males). The same pattern was found among their sample of 120 college students. Other studies have also found higher overall scores for women (e.g., Kojima et al. 2002; Roelofs et al. 2013). Additionally, Wagener et al. (2016) found that, in terms of specific symptom endorsements, women are

more likely to score higher on sadness and selfcriticalness, while men are more likely to endorse past failure and loss of pleasure. However, studies with other populations have not found similar gender differences (e.g., chronic pain; Harris and D'Eon 2008). Likewise, among US college students, the BDI-II provided an assessment of the severity of depression symptoms that was equivalent across gender, race, and ethnicity (Whisman et al. 2013). Consistent with this finding, in terms of BDI-II reporting by patients of different ages, studies have found it to also have strong psychometric support among geriatric patients (Segal et al. 2008; Steer et al. 2000).

It is also important to consider the possible differences in items endorsed among individuals with specific medical conditions. Physical sequelae of certain conditions may mimic somatic symptoms of depression, resulting in false classifications of depressive symptomatology. Despite this possibility, overall, the BDI-II has been found to be useful across medical populations, including individuals with chronic pain (Harris and D'Eon 2008), multiple sclerosis (Sacco et al. 2016), myocardial infarction (Huffman et al. 2010), and traumatic brain injury (Rowland et al. 2005). Some studies have suggested, however, that there may be different optimal cutoff scores for different populations (e.g., Huffman et al. (2010) found >=16 to be the optimal cutoff for patients with a history of myocardial infarction). Patterson et al. (2011) found that, among patients with hepatitis C, questions targeting cognitive and affective symptoms (rather than somatic symptoms) may be a more valid measurement of depression.

While the reading level requirement for the BDI-II is reported at a fifth to sixth grade level, examination of the cognitive complexity of this measure may require more scrutiny with certain clinical populations. The presence of multiple response options increases the complexity of this self-administered measure, which may impact its appropriateness for specific populations or settings in which motivation to respond may be low, even when the individual possesses the literacy skills necessary for response (Shumway et al. 2004).

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 generally appears to extend to ethnic and culturally diverse populations as well, although research is ongoing. Of note, the BDI-II is not intended to be used for the sole purpose of "specifying a clinical diagnosis," but as an indicator of the severity of depressive symptoms (Beck et al. 1996b). Therefore, it is important for clinicians to use clinical judgment, consider the demographic characteristics and medical condition of their patients, and consult current research when evaluating a patient for depression and interpreting BDI-II results.

Cross-References

- Center for Epidemiological Studies: Depression
- ► Geriatric Depression Scale
- ▶ Hamilton Depression Rating Scale
- Self-Report Measures
- Zung Self-Rating Depression Scale

References and Readings

- American Psychiatric Association. (1987). Diagnostic and statistical manual of mental disorders (3rd ed.). 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., & Steer, R. A. (1987). Manual for the Beck Depression Inventory. San Antonio: 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.
- 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., Ball, R., & Ranieri, W. (1996a). 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. (1996b). Beck Depression Inventory manual (2nd ed.). San Antonio: The Psychological Corporation.
- 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.

- Canel-Çınarbaş, D., Cui, Y., & Lauridsen, E. (2011). Cross-cultural validation of the Beck Depression Inventory–II across U.S. and Turkish samples. *Mea*surement and Evaluation in Counseling and Development, 44(2), 77–91.
- Carmody, D. P. (2005). Psychometric characteristics of the Beck Depression Inventory-II with college students of diverse ethnicity. *International Journal of Psychiatry in Clinical Practice*, 9(1), 22–28.
- Dere, J., Watters, C. A., Yu, S. C., Bagby, R. M., Ryder, A. G., & Harkness, K. L. (2015). Cross-cultural examination of measurement invariance of the Beck Depression Inventory–II. *Psychological Assessment*, 27(1), 68–81.
- Dozois, D. J. A., Dobson, K. S., & Ahnberg, J. L. (1998). A psychometric evaluation of the Beck Depression Inventory–II. *Psychological Assessment*, 10, 83–89.
- Edwards, D. A., & Steele, G. I. (2008). Development and validation of the Xhosa translations of the Beck inventories: 3. Concurrent and convergent validity. *Journal of Psychology in Africa*, *18*(2), 227–236.
- Gomes-Oliveira, M. H., Gorenstein, C., Neto, F. L., Andrade, L. H., & Wang, Y. P. (2012). Validation of the Brazilian Portuguese version of the Beck Depression Inventory-II in a community sample. *Revista Brasileira de Psiquiatria*, 34(4), 389–394.
- González, D. A., Rodríguez, A. R., & Reyes-Lagunes, I. (2015). Adaptation of the BDI–II in Mexico. Salud Mental, 38(4), 237–244.
- Hamdi, N., Abu-Hajlah, N., & Abu-Talib, S. (1988). The factorial structure, reliability, and validity of an Arabic version of the Beck Depression Inventory. *Dirasat*, 15(1), 30–40.
- 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.
- Harris, C. A., & D'Eon, J. L. (2008). Psychometric properties of the Beck Depression Inventory-Second Edition (BDI-II) in individuals with chronic pain. *Pain*, 137(3), 609–622.
- Hong, S., & Wong, E. C. (2005). Rasch rating scale modeling of the Korean version of the Beck Depression Inventory. *Educational and Psychological Measurement*, 65(1), 124–139.
- Huffman, J. C., Doughty, C. T., Januzzi, J. L., Pirl, W. F., Smith, F. A., & Fricchione, G. L. (2010). Screening for major depression in post-myocardial infarction patients: Operating characteristics of the Beck-Depression Inventory-II. *International Journal of Psychiatry in Medicine*, 40(2), 187–197.
- Kojima, M., Furukawa, T. A., Takahashi, H., Kawai, M., Nagaya, T., & Tokudome, S. (2002). Cross-cultural validation of the Beck Depression Inventory-II in Japan. *Psychiatry Research*, 110(3), 291–299.
- 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., 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.
- 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.
- Patterson, A. L., Morasco, B. J., Fuller, B. E., Indest, D. W., Loftis, J. M., & Hauser, P. (2011). Screening for depression in patients with hepatitis C using the Beck Depression Inventory-II: Do somatic symptoms compromise validity? *General Hospital Psychiatry*, 33(4), 354–362.
- Roelofs, J., van Breukelen, G., de Graaf, L. E., Beck, A. T., Arntz, A., & Huibers, M. H. (2013). Norms for the Beck Depression Inventory (BDI-II) in a large Dutch community sample. *Journal of Psychopathology and Behavioral Assessment*, 35(1), 93–98.
- Rowland, S. M., Lam, C. S., & Leahy, B. (2005). Use of the Beck Depression Inventory-II (BDI-II) with persons with traumatic brain injury: Analysis of factorial structure. *Brain Injury*, 19(2), 77–83.
- Sacco, R., Santangelo, G., Stamenova, S., Bisecco, A., Bonavita, S., Lavorgna, L., ..., & Gallo, A. (2016). Psychometric properties and validity of Beck Depression Inventory II in multiple sclerosis. *European Journal of Neurology*, 23(4), 744–750.
- 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(2–3), 191–198.
- Steer, R. A., Rissmiller, D. J., & Beck, A. T. (2000). Use of Beck Depression Inventory-II with depressed geriatric inpatients. *Behaviour Research and Therapy*, 38(3), 311–331.
- 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 and Anxiety*, 19 (3), 187–189.
- Wagener, A., Baeyens, C., & Blairy, S. (2016). Depressive symptomatology and the influence of the behavioral avoidance and activation: A gender-specific investigation. *Journal of Affective Disorders*, 193, 123–129.
- Whisman, M. A., Judd, C. M., Whiteford, N. T., & Gelhorn, H. L. (2013). Measurement invariance of the Beck Depression Inventory–Second Edition (BDI-II) across gender, race, and ethnicity in college students. *Assessment*, 20(4), 419–428.
- 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.
- Wu, P., & Chang, L. (2008). Psychometric properties of the Chinese version of the Beck Depression Inventory-II using the Rasch model. *Measurement and Evaluation* in Counseling and Development, 41(1), 13–31.

Beery Developmental Test of Visual-Motor Integration (VMI), Sixth Edition

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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 et al. 2004), typically referred to as the Beery VMI, is designed to assess the integration of visual and motor abilities. The current version includes two forms for the Beery VMI and two supplemental subsections, motor coordination and visual perception. The supplemental tests may be administered after results from the VMI test indicate the need for further assessment in order to separate an individual's pure motor and visual abilities. For the VMI, examinees are administered either a 21-item short form or a 30-item long form; each requires copying geometric forms that become increasingly complex. The short form is designed for use with children aged 2–7 years, and the long form for individuals up to age 100. The long form takes approximately 10-15 min to administer, and the short form takes approximately 10 min. The test may also be administered to a group; however, the authors recommend individual testing for children who have developmental delays or neurological impairments. For children at or over the functional age of 5, administration begins with item 7. If the examinee is unable to complete item 7 correctly, the manual provides instructions for how to proceed. The examinees use a pen or pencil to complete the geometric forms. One point is given for each correctly drawn figure, and

testing is discontinued after three consecutive failures. A composite standard score is obtained.

Background

The first edition of the Beery VMI was published in 1967 (Beery, Buktenica, and Beery). While other measures of visual-motor integration were available at the time, none involved a sequence of increasingly complex geometric forms. Although the Beery VMI is currently in its sixth edition, the test items found in the current edition are almost identical to the original stimuli. The 1997 edition added the two supplemental forms so that visual-motor integration could be compared to pure visual or pure motor performance. The sixth and most recent edition includes suggestions for teaching and improving visualmotor integration.

Psychometric Data

The Beery VMI manual reports internal consistency reliabilities averaging 0.92 for visualmotor integration, 0.91 for visual perception, and 0.90 for motor coordination. Analyses of convergent validity found the Beery VMI correlates 0.52 with the drawing subtest of the Wide Range Assessment of Visual Motor Abilities (WRAVMA) and 0.75 with the copying subtest of the Developmental Test of Visual Perception (DTVP-2).

Normative data for the Beery VMI were updated in the sixth edition (2010) with a sample of 1,700 individuals with demographic characteristics closely matching those from the 2010 US Census. From ages 2–13, standard scores are provided in 2-month intervals for the Beery VMI, and in 3-month intervals for the two supplemental tests. For ages 13–19, Beery VMI norms are by year, and the supplemental tests are by 2-year periods. For adults, norms are by decade. Standard scores have a mean of 100 and standard deviation of 15, and scores may be converted to other scales (e.g., scaled scores, percentiles).

Clinical Use

The Beery VMI is a useful early screening tool for psychologists, learning disability specialists, school counselors, teachers, and other professionals to identify children with visual-motor impairments. Test results assist in making appropriate referrals for services, or to test the effectiveness of educational and other intervention programs. Researchers use the test to examine deficits in visual-motor integration in specific neurodevelopmental disorders. In one study, the Beery VMI was used to compare visual-motor integration in children diagnosed with ADHD and those with comorbid ADHD and reading disability, and/or oppositional defiant disorder (Kooistra et al. 2005). This study found increases in motor impairments among children with ADHD as a function of comorbid diagnoses, particularly reading disability. Another study examined differences in VMI performance between children with traumatic brain injury and ADHD in order to examine the instrument's validity, and found support for the use of the VMI in children with both developmental and acquired brain dysfunction (Sutton et al. 2011). The Beery VMI has also been used in research studies of the neuropsychological outcomes for children born preterm (Baron et al. 2009), and individuals with Autism Spectrum Disorder (Green et al. 2015). Performance on the Beery VMI can inform diagnostic decisions across a wide spectrum of disorders.

Cross-References

Rey Complex Figure Test

References and Further Readings

- Baron, I. S., Erickson, K., Ahronovich, M., Coulehan, K., Baker, R., & Litman, F. (2009). Visuospatial and verbal fluency relative deficits in 'complicated' late-preterm preschool children. *Early Human Development*, 85, 751–754.
- Beery, K. E., Buktenica, N. A., & Beery, N. A. (2004). The Beery-Buktenica developmental test of visual-motor

integration: Administration, scoring, and teaching manual (5th ed.). Minneapolis: NCS Pearson, Inc..

- Beery, K. E., Buktenica, N. A., & Beery, N. A. (2010). The Beery-Buktenica developmental test of visual-motor integration: Administration, scoring, and teaching manual (6th ed.). Minneapolis: NCS Pearson, Inc..
- Green, R. R., Bigler, E. D., Froehlich, A., Prigge, M. B., Travers, B. G., Cariello, A. N., & Lainhart, J. E. (2015). Beery VMI performance in autism spectrum disorder. *Child Neuropsychology*. https://doi.org/10.1080/ 09297049.2015.1056131.
- Kooistra, L., Crawford, S., Dewey, D., Cantell, M., & Kaplan, B. J. (2005). Motor correlates of ADHD contribution of reading disability and oppositional defiant disorder. *Journal of Learning Disabilities*, 38, 195–206.
- Sutton, G. P., Barchard, K. A., Bello, D. T., Thaler, N. S., Ringdahl, E., Mayfield, J., & Allen, D. N. (2011). Beery-Buktenica developmental test of visual-motor integration performance in children with traumatic brain injury and attention-deficit/hyperactivity disorder. *Psychological Assessment*, 23, 805.

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 prosocial. Prosocial behaviors are typically seen as personally fulfilling, productive, and socially acceptable. The process typically includes identifying negative behaviors, raising awareness about alternative behaviors, and changing the environment by modifying antecedents to behaviors or the consequences. 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 (antecedent) cues) than operant conditioning (e.g., recalling the contingency between behaviors and the resulting consequences).

Cross-References

- Applied Behavior Analysis
- Behavior Modification
- Behavioral Therapy

References and Readings

- Jacobs, H. E. (1993). Behavior analysis guidelines and brain injury rehabilitation: People, principles and programs. Gaithersburg: Aspen.
- Karol, R. L. (2003). Neuropsychological intervention: The practical treatment of severe behavioural dyscontrol after acquired brain injury. Boca Raton: CRC Press.
- Novack, T., PhD. (n.d.). Management of behavioral problems during acute rehabilitation of individuals with TBI. Retrieved 30 Nov 2016, from http://www.brainline.org/ content/2008/07/management-behavioral-problems-dur ing-acute-rehabilitation-individuals-tbi pageall.html

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 and improve daily functioning. These techniques are typically based on functional assessment and used to reinforce adaptive behaviors while diminishing or extinguishing maladaptive behaviors. Behavioral modification techniques can be used to address learning issues as well as social, emotional, behavioral, or psychiatric problems. Seven characteristics of behavior modification, identified by Martin and Pear (2015), 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
- Behavioral Assessment
- Behavior Management
- 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 and Clinic*, 39(5), 305.
- Hicinbothem, J., Gonsalves, S., & Lester, D. (2006). Body modification and suicidal behavior. *Death Studies*, 30(4), 351.
- Kazdi, A. E. (2012). Behavior modification in applied settings. Long Grove: Waveland Press.
- 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.J. (2015). *Behavior modification: What it is and how to do it* (10th ed.). New York, NY: Routledge Psychology Press.
- Ntinas, K. (2007). Behavior modification and the principle of normalization: Clash or synthesis? *Behavioral Interventions*, 22(2), 165–177.

Behavior Rating Inventory for Executive Function

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Synonyms

BRIEF; BRIEF2; BRIEF-A; BRIEF-P; BRIEF-SR

Description

The Behavior Rating Inventory of Executive Function (BRIEF) family of measures are rating scales designed to facilitate assessment of the behavioral manifestations of executive dysfunction in everyday environments such as home, school, and work. First published in 2000 as parent and teacher rating scales of executive function in children and adolescents, the family of measures has grown to include versions for assessing preschool children, adolescents' self-report, and adults.

The original BRIEF consists of two forms, a parent questionnaire and a teacher questionnaire, designed to assess executive function behaviors in children and adolescents aged 5–18 years in home and school environments. It includes 86 items with 8 theoretically and empirically derived clinical scales measuring Inhibit, Shift, Emotional Control, Initiate, Working Memory, Plan/Organize, Monitor, and Organization of Materials. 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).

In 2015, the first revision of the BRIEF, the BRIEF2, was published, reducing the length of the measure by approximately one quarter while adding numerous enhancements. These were informed by the hundreds of peer-reviewed papers that have employed the measure in a wide range of clinical and normative groups in multiple languages on six continents; tested the factor structure; explored relationships with academic, behavioral, emotional, social, and adaptive functioning; documented associations with biological factors including brain structure and function; and demonstrated sensitivity to change with recovery or treatment. The BRIEF2 includes parallel Parent and Teacher forms and incorporates the previously separate adolescent self-report form. Parent and Teacher forms are composed of 63 items within 9 theoretically and empirically derived clinical scales that are largely consistent with those of the BRIEF, with the exception that the Monitor scale was separated into Self-Monitor and Task Monitor scales. The BRIEF2 includes three validity scales, Inconsistency, Negativity, and a new Infrequency scale. Finally, the nine scales form three broader indexes based on the factor structure, Behavior Regulation, Emotion Regulation, and Cognitive Regulation (similar to the metacognition index on the BRIEF), as well as a Global Executive Composite score, or the GEC.

The BRIEF2 also includes a revised and co-normed version of the BRIEF Adolescent Self-Report. This is a 55-item Self-Report form designed to complement the BRIEF2 Parent and Teacher forms. It is appropriate for older children and adolescents ages 11–18 years with a fifthgrade or higher reading ability. The items yield information for seven clinical scales: Inhibit, Self-Monitor, Shift, Emotional Control, Task Completion, Working Memory, and Plan/ Organize. The clinical scales form three indexes, the Behavior Regulation Index (BRI), Emotion Regulation Index (ERI), and Cognitive Regulation Index (CRI), and an overall summary score, the Global Executive Composite (GEC). The BRIEF2 Self-Report also includes three validity scales – Infrequency, Inconsistency, and Negativity.

An important enhancement in the BRIEF2 is the inclusion of three 12-item screening forms for parents, teachers, and adolescents. These concise forms were created to meet the needs of large-scale assessment in education, health, and research settings. Each correlates strongly with the BRIEF2 Global Executive Composite score (r > 90) and discriminates between typically developing children and those with executive function deficits with large effect sizes. Reliabilities are strong, there are multiple lines of evidence for validity, and the standardization sample was large and stratified by gender, race/ ethnicity, parent education, and geographic region. Classification statistics including sensitivity/specificity and likelihood ratios are used to identify children at risk for executive function problems who should be more fully assessed.

The BRIEF-Preschool Version (BRIEF-P) measures the behavioral manifestations of executive function in preschool-aged children, ages 2-5. It 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 5 theoretically and empirically derived clinical scales measuring Inhibit, Shift, Emotional Control, Working Memory, and Plan/Organize. These scales form three factorderived indexes, Inhibitory Self-Control, Flexibility, and Emergent Metacognition, and one composite score, the GEC. The BRIEF-P also includes two validity scale, Inconsistency and Negativity.

The BRIEF-A measures an adult's executive functions in his or her everyday home and work environment. Two forms are available, 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 9 clinical scales measuring: Inhibit, Self-Monitor, Shift, Emotional Control, Initiate, Working Memory, Plan/Organize, Task-Monitor, and Organization of Materials. The clinical scales form two broader factor-based indexes: Behavioral Regulation (BRI) and Metacognition (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 ability to predict functioning in the everyday environment. Fundamentally, executive functions are necessary for organization of goal-directed behavior in the everyday, "realworld" 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 functions through the assessment of an 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 with respect to which cognitive and behavioral processes are viewed as part of the executive function domain, 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 novel and/or complex problems, flexible shifting of behavior and problem-solving strategies when necessary, monitoring and evaluation of problemsolving behavior and task performance more generally, as well as monitoring the effects of one's own behavior on others. In support of these behaviors, working memory capacity plays a fundamental role in holding information actively "online" 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, as well as studies using advanced brain imaging techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI). 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. Damage to any given component of this circuitry may result in executive dysfunction.

The BRIEF was originally developed beginning in 1994 following a commonly accepted developmental model of executive function (Welsh and Pennington 1988; Holmes-Bernstein and Waber 1990). The impetus arose from the authors' 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. The measure found acceptance initially within the field of pediatric neuropsychology and was published first in 2000. 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 (Roth et al. 2014; Strauss et al. 2006).

Psychometric Data

BRIEF2 (Ages 5–18 Years: Parent and Teacher Forms)

Standardization: Normative data are based on 1,400 parents and 1,400 teachers from rural, suburban, and urban areas. The samples were diverse to match proportions of race/ethnicity, parental education level, geographic region, and gender across all 50 states, based on the US population data from the Current Population Survey, March 2013 by the US Census Bureau, 2012, Washington, DC. 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 2,892 parents and 1,889 teachers rating children with developmental disorders or acquired neurological disorders (e.g., learning disabilities, ADHD, TBI, Tourette's syndrome, mental retardation, epilepsy, and language disorders).

Reliability: High internal consistency (Cronbach's alpha >0.90 for Parent and Teacher Index Scores). Test-retest reliability for composites was r = 0.82-0.89 for parent normative ratings and r = 0.83-0.90 for teacher normative ratings. Interobserver reliability reflected moderate to high correlations for parent-parent ratings (mean r = 0.77 for normative sample, 0.59 for clinical sample), moderate correlations for parent-teacher (mean r = 0.30-0.50), teacher-teacher

ratings (mean r = 0.39 for normative sample, mean r = 0.56 for clinical sample).

Validity: Evidence of validity is demonstrated by several lines of evidence including high interrater 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, BASC, CBCL, and CRS. Exploratory and confirmatory factor analysis of the BRIEF2 Parent, Teacher, and Self-Report forms yielded a consistent three-factor solution (i.e., Behavior Regulation, Emotion Regulation, Cognitive Regulation) for normative and clinical samples. Two of the scales, Working Memory and Inhibit, are clinically useful in detecting and predicting the diagnosis of attention-deficit/hyperactivity disorder (ADHD).

BRIEF2 Self-Report (BRIEF2-SR)

Standardization: The BRIEF2-SR was standardized and validated for use with children and adolescents aged 11–18 years. The normative sample includes 803 participants using the same comprehensive sampling as the Parent and Teacher forms matched to the US Census data. Clinical data are based on 473 children and adolescents with a variety of developmental disorders or acquired neurological disorders (e.g., learning disabilities, ADHD, TBI, Tourette's syndrome, autism spectrum disorders, epilepsy, and brain tumor disorders).

Reliability: The BRIEF2-SR scales demonstrate appropriate reliability. Internal consistency is high for the GEC ($\alpha = 0.97$) and moderate to high for the clinical scales ($\alpha = 0.81-0.88$). Temporal stability is strong ($\underline{r} = 0.85$ for the GEC over a period of 3.7 weeks), and there is strong interrater agreement for the GEC with parent ratings on the BRIEF (r = 0.71). Teacher ratings on the BRIEF2-SR correlated moderately with adolescent ratings on the BRIEF-SR (GEC r = 0.57).

Validity: Principal factor analysis of the BRIEF2-SR yielded a three-factor solution (i.e., Behavior Regulation, Emotion Regulation, Cognitive Regulation) for normative and clinical samples. Correlational analyses with other selfreport 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 BRIEF2-SR profiles in a variety of clinical groups provides further evidence of validity based on clinical utility. BRIEF2-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 low to moderate (r = 0.25 - 0.35), suggesting agreement yet different perspectives as well.

BRIEF-Preschool (BRIEF-P: Ages 2–5 Years, Parent and 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 ($\alpha = 0.80-0.95$ for parent sample and $\alpha = 0.90-0.97$ 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-Adult (BRIEF-A: Self-Report and Informant Report)

Standardization: The BRIEF-A was standardized and validated for use with men and women from ages 18–90 years. The normative sample includes 1,050 adult self-reports and 1,200 informant reports 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 ($\alpha = 0.73-0.90$ for clinical scales; 0.93-0.96 for indexes and GEC) and high for the Informant Report normative sample $(\alpha = 0.80-0.93$ 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 ($\alpha = 0.80-0.94$ for clinical scales; 0.96-0.98 for indexes and GEC) and the Informant Report form ($\alpha = 0.85 - 0.95$ 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 = 0.91-0.94 for the Informant Report Form (n = 0.44). Correlations between Self-Report ratings and Informant Report ratings were moderate, ranging from r = 0.44-0.68 for the clinical scales and from 0.61–0.63 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-Report and Informant Report on the Frontal Systems Behavior Scale, Dysexecutive Questionnaire, and Cognitive Failures Questionnaire. Validity has been further demonstrated via studies of clinical populations. Factor analysis of Self-Report form data yielded a two-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 two-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 family of instruments 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 life span. Executive function deficits measured via the BRIEF have been demonstrated in a variety of populations such as ADHD, traumatic brain injury, 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 prenatal alcohol exposure. The BRIEF-A has been examined in clinical populations such as mild cognitive impairment, ADHD, epilepsy, traumatic brain injury, schizophrenia, and cancer survivors.

The measures also show promise for veridicality, that is, predicting behavior in the natural environment. For example, correlational analyses suggest strong, logical relationships between the Inhibit scale and aggression and the Working Memory scale with attention problems. Correlations have also been reported between BRIEF and aspects of real-world functioning such as adaptive functioning in individuals with developmental disabilities, scholastic achievement and performance on high-stakes testing in children, as well as college adjustment and academic procrastination in young adults. While there are modest correlations between the BRIEF and performance tests that tap aspects of executive functions, the BRIEF shows significant associations with biological markers such as lead levels, structural and functional neuroimaging (e.g., frontal lobe volume, white matter integrity), and genetic markers (e.g., polymorphisms of the monoamine oxidase A gene). Finally, certain BRIEF profiles of executive function in the everyday environment can help predict diagnoses such as ADHD and autism spectrum disorder.

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. 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. 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, assessment of executive function with the BRIEF can inform clinical treatment design, monitoring, and outcome evaluation. Given the inherent difficulty in administering performance measures of executive function in a repeated fashion, behaviorally anchored measures may be well suited to such within-subject 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 (Ransom et al. 2016). The BRIEF2 monitoring form may be especially useful for such situations where frequent reassessment is needed in a timeand resource-sensitive manner.

See Also

- Attention Deficit Hyperactivity Disorder
- ► Concussion
- ► Executive Functioning
- ► Traumatic Brain Injury (TBI)

Further Reading

- 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., Kenworthy, L., & Barton, R. M. (2002). Profiles of everyday executive function in acquired and developmental disorders. *Child Neuropsychology*, 8, 121–137.
- 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: Psychology Press.

- Gioia, G. A., Kenworthy, L., & Isquith, P. K. (2010). Executive function in the real world: BRIEF lessons from mark Ylvisaker. *The Journal of Head Trauma Rehabilitation*, 25(6), 433–439.
- Holmes-Bernstein, J., & Waber, D. (1990). Developmental neuropsychological assessment: A systemic approach. *NeuroMethods*, 17, 311–372.
- 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.
- Isquith, P. K., Roth, R. M., & Gioia, G. (2013). Contribution of rating scales to the assessment of executive functions. *Applied Neuropsychology: Child*, 2(2), 125–132.
- Isquith, P. K., Roth, R. M., Kenworthy, L., & Gioia, G. (2014). Contribution of rating scales to intervention for executive dysfunction. *Applied Neuropsychology: Child*, 3(3), 197–204.
- 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.
- Ransom, D. M., Burns, A., Youngstrom, E. A., Sady, M. D., Vaughan, C. G., & Gioia, G. A. (2016). Applying an evidence-based assessment model to identify students at risk for perceived academic problems following concussion. *Journal of the International Neuropsychological Society*, 22(10), 1038–1049.
- Roth, R. M., Isquith, P. K., & Gioia, G. A. (2014). Assessment of executive functioning using the behavior rating inventory of executive function (BRIEF). In S. Goldstein & J. A. Naglieri (Eds.), *Handbook of executive functioning* (2nd ed., pp. 301–331). New York: Springer.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). Behavior rating inventory of executive function (BRIEF). In A compendium of neuropsychological tests, administration, norms, and commentary (3rd ed., pp. 1090–1099). Oxford: Oxford University Press.

Behavioral Assessment

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Synonyms

Behavior/behavioral analysis; Behavior/behavioral observation

Definition

Behavioral assessment is systematic а 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

- Applied Behavior Analysis
- ► Behavior Management
- Behavior Modification
- ► Behavior Rating Inventory for Executive Function
- Behavioral Assessment of the Dysexecutive Syndrome
- Behavioral Therapy
- ► Conners Comprehensive Behavior Rating ScalesTM
- Functional Assessment
- Functional Assessment Measure

References and Readings

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

Behavioral Assessment of the Dysexecutive Syndrome

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Synonyms

BADS

Description

The BADS (Wilson et al. 1996) is a test battery aimed at predicting everyday difficulties that arise as a result of the Dysexecutive Syndrome (DES). It consists of six subtests and a 20-item questionnaire that tap executive functioning in an ecologically valid way. The subtests are as follows: The Rule Shift Cards Test is a measure of cognitive flexibility that consists of 21 spiral-bound cards that are used to assess the individual's ability to respond correctly to a rule and to shift from one rule to another. It is scored based on the time taken and number of errors made. In the first part, the individual is asked to respond "Yes" to a red card and "No" to a black card. This component establishes a pattern of behavior that is geared to increase the probability of perseverative errors in the second part, when the rules are changed. In the second part, the individual is asked to respond "Yes" if the card just turned over is 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 the individual 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. The individual is asked to get the cork out of the tube using any of the objects around without lifting the stand, the, tube or the beaker and without touching the lid with their fingers.

In the Key Search Test, the individual is 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. The individual is 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 individual is scored based on how efficient the search process is.

The Temporal Judgment Test comprises four questions concerning everyday events which range from requiring a few seconds to several years. The individual is 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, subjects 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, the individual will incur a high number of errors by simply visiting the locations in the order given in the instructions. In the low demand component, the individual is simply required to follow the instructions to produce an error-free performance. The goal of the test is to assess the individual's 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. The individual is required to attempt at least a part from each of the six subtests within a 10-minute period and is instructed not to do the two parts of the same task consecutively. The goal of this component is to determine the person's ability to plan, organize, and monitor their 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 individual and the other by an informant.

Historical Background

The BADS is designed to evaluate the pattern 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 likely to present as impulsive, distractible, and unable to use feedback to modify their responses and behave inappropriately in social situations.

The BADS was developed due to the fact that patients with impaired executive functioning often performed adequately on tests such as the Wisconsin Card Sorting Test or the Stroop Test. These same individuals, however, exhibited obvious impairment in their day-to-day functioning. To this end, Shallice and Burgess (1991) developed the Six Elements Test, which required the individual to carry out six tasks in a limited time frame without violating certain rules. It was tailored to a difficulty level that was in line with the high level of functioning of Shallice and Burgess' patients. Wilson et al. (1996) modified the Six Elements Test, simplifying it for more severely impaired and less intellectually able patients that are often seen by neuropsychologists. This evolved into the BADS.

Psychometric Data

Multiple studies attest to 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 subjects generally performing slightly higher after the second administration, but the differences were not statistically significant. 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 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 et al. (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 experienced loss of consciousness and varying degrees of post-traumatic amnesia (PTA). Based on their findings, the authors concluded that scores derived from the BADS and other measures used in their study were only moderately useful in assessing executive dysfunction. On the other hand, several studies have found the BADS to discriminate between patients and controls. Krabbendam et al. (1999) were able to discriminate between schizophrenic patients and controls, while Katz et al. (2007) were able to discriminate between acute and chronic schizophrenics, 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 individuals (SDI). They concluded that the BADS yielded greater effect sizes for differences between SDI and controls than traditional measures of executive function. SDI performance on the BADS was also useful as a predictor of problems in daily activities. Third, deficits in BADS scores persisted following protracted abstinence, even when other neuropsychological indices showed recovery.

Canali et al. (2011) examined the reliability of the BADS in its ability to discriminate Brazilian older adults with and without mild Alzheimer's disease. Intergroup differences were reported on most components of the measure, including task switching, time monitoring, and rule-shifting subtests. The highest level of discrimination between controls and patients was found on the Modified Six Elements, with a sensitivity index of 80% and specificity index of 90%.

Clinical Uses

Wilson et al. (1996) developed the BADS to aid those involved in the assessment of individuals with brain injury to determine the extent of executive dysfunction that is present and the likelihood that it will interfere with everyday life. It can also be used to determine the presence of executive dysfunction in other patient groups, such as schizophrenics and substance abusers. The BADS can be a useful part of the rehabilitation process as a tool that can pick up subtle difficulties with planning and organization, which are then amenable to intervention. For example, Baba et al. (2010) found that the executive functioning of 20 Japanese adults with remitted major depressive disorder were more impaired on the Modified Six Elements subtest relative to 29 healthy comparison subjects.

See Also

► Frontal Lobe

Further Reading

- Baba, K., Baba, H., Noguchi, I., Arai, R., Suzuki, T., & Mimura, M. (2010). Executive dysfunction in remitted late-life depression: Juntendo University mood disorder projects (JUMP). *The Journal of Neuropsychiatry* and Clinical Neurosciences, 22, 70–74.
- 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.
- Canali, F., Brucki, S. M. D., Bertolucci, P. H. F., & Bueno, O. F. A. (2011). Reliability study of the behavioral assessment of the dysexecutive syndrome adapted for

a Brazilian sample of older-adult controls and probable early Alzheimer's disease patients. *Revista Brasileira de Psiquiatria*, *33*, 338–346.

- 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 operation on the frontal lobes. *Acta Psychiatrica Neurologica*, (30).
- Shallice, T. (1982). Specific impairments of planning. *Philosophical Transactions of the Royal Society of London.* Series B, Biological Sciences, 298, 199-209.
- 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 individuals. *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. London: Harcourt Assessment.

Behavioral Inattention Test (BIT)

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The Behavioral Inattention Test (BIT) was developed in the United Kingdom in 1987 to assess hemi-inattention and has predominantly been used with stroke patients to assess unilateral spatial neglect (Wilson et al. 1987b; Halligan et al. 1991). Unilateral spatial neglect is commonly defined as an inability to respond to or notice stimuli that is present on the side opposite of the brain lesion location. This spatial neglect is not attributed to motor or sensory deficits (Heilman et al. 1993). In an attempt to improve ecological validity, the BIT incorporated nine behavioral subtests in addition to six conventional subtests. The behavioral subtests aim to assess unilateral spatial neglect as well as an individual's functioning on activities of daily living.

The BIT takes approximately 40 min to complete and can be administered to individuals ages 19-83. The conventional subtests consist of line crossing, star cancellation, letter cancellation, line bisection, figure/ shape copying, and representation drawing. Line crossing and the cancellation subtests require the examinee to cross out the target items, where the cancellation subtests add a level of difficulty with the presence of various nontarget items. Line bisection requires the examinee to estimate and mark the center of three horizontal lines. In the figure copying portion of the subtest, the examinee is presented with a drawing of a four-pointed star, a cube, and a daisy in a vertical orientation where each of the items are pointed out to the examinee prior to asking the examinee to draw the items. The shape copying portion of the same subtests consists of the examinee drawing three geometric shapes that are presented but not distinctly pointed out to the examinee. The representation drawing subtest requires the examinee to draw a clock face with numbers, a man or woman, and a butterfly. Both of the drawing subtests include images that tend to be bilaterally symmetric (Halligan et al. 1991).

The behavioral subtests of the BIT are comprised of: menu reading, article reading, address/ sentence copying, telling/setting the time, telephone dialing, picture scanning, coin sorting, card sorting, and map navigation. Menu reading consists of a list of common food items presented in columns. The article reading subtest contains three columns of text that are to be read by the examinee. Address/Sentence copying asks the examinee to copy four lines of an address and three lines of sentences. Telling/setting time requires the examinee to read the time on a digital clock as well as analogue clock and to set time on an analogue clock with moveable hands based on the examiner's verbal instructions. Telephone dialing utilizes a disconnected telephone and requires the examinee to dial three numbers presented in large print on separate cards. The picture scanning subtest presents the examinee with three large color photographs of a plate of food, a bathroom, and a hospital room. The examinee is asked to look at each of the photos one at a time and to point as well as name the major items in each of the photos. Coin sorting presents three rows of coins with six different denominations where the examinee is asked to identify and locate the coins as the examiner names various coins. The card sorting subtest involves the examiner pointing out each of the 16 cards to the examinee and then asking the examinee to point to the card being named by the examiner. Map navigation contains a grid of different paths marked by a letter and as the examiner says letter pairs the examinee is asked to follow the path using their finger (Halligan et al. 1991; Lezak et al. 2012).

The reliability of the BIT was initially based on a small sample size but contained excellent testretest reliability with the behavioral subtest at r = 0.97 and the conventional subtests at r = 0.89. The conventional and behavioral subtests are also highly correlated with each other at r = 0.75. In terms of inter-rater reliability, the BIT is also highly reliable with both the behavioral and conventional subtests being r = 0.99 (Halligan et al. 1991). Thus, the BIT has been a common neuropsychological measure used to assess unilateral neglect postinjury and throughout recovery (Azouvi 2016). Maximum total score for the conventional subtests is 146 with a clinical cutoff of 129. While the behavioral subtests total maximum score is 81 with a clinical cutoff score of 67. When an individual scores lower on either one of these they are classified as having unilateral spatial neglect via BIT (Wilson et al. 1987a).

Using the BIT to predict functional outcomes, one study found BIT scores to be significantly correlated with Functional Independence Measure (FIM) scores at the time of discharge from rehabilitation, 0.385 (P = 0.004) for the conventional BIT and 0.396 (P = 0.003) for the behavioral BIT subtests (Di Monaco et al. 2011). These results indicate that the severity of unilateral spatial neglect should be accounted for when estimating functional outcome poststroke and is consistent with prior findings (Buxbaum et al. 2004; Cherney et al. 2001). In general, studies have found that unilateral spatial neglect contributes to worse functional outcomes and longer rehabilitation durations (Gillen et al. 2005; Franceschini et al. 2010). In fact, having unilateral spatial neglect was indicative of poorer functional outcomes in 25 out of 26 studies examined and the BIT has been shown to be the greatest predictor of function poststroke at 3, 6, and 12 months postinjury (Jehkonen et al. 2006; Jehkonen et al. 2000). To examine the ecological validity of the BIT, the behavioral subtests have been compared to task performance as well as to Activities of Daily Living (ADLs) checklist in patient samples. The results found that six out of the nine subtests correlated with task performance and ADLs. Additionally, seven out of the nine behavioral subtests were able to differentiate between individuals with and without spatial neglect (Hartman-Maeir and Katz 1995).

Research studies often implement one or several of the BIT subtests but this may not be an accurate way to distinguish unilateral neglect (Lopes et al. 2007). In cases where only one subtest is administered, the sensitivity of the measure may be lost. Lopes and colleagues found that all of the patients with hemi-neglect were properly identified using figure and shape copying as well as the representational drawing subtests. However, this was not the case with other subtests of the BIT when examined in a stand-alone manner. Ultimately, the full BIT test administration is still recommended for greater sensitivity.

References

- Azouvi, P. (2016). The ecological assessment of unilateral neglect. Annals of Physical and Rehabilitation Medicine. https://doi.org/10.1016/j.rehab.2015.12.005.
- Buxbaum, L. J., Ferraro, M. K., Veramonti, T., Farne, A., Whyte, J., Ladavas, E., et al. (2004). Hemispatial

neglect: Subtypes, neuroanatomy, and disability. *Neurology, 62*, 749–756.

- Cherney, L. R., Halper, A. S., Kwasnica, C. M., Harvey, R. L., & Zhang, M. (2001). Recovery of functional status after right hemisphere stroke: Relationship with unilateral neglect. *Archives of Physical Medicine and Rehabilitation*, 82, 322–328.
- Di Monaco, M., Schintu, S., Dotta, M., Barba, S., Tappero, R., & Gindri, P. (2011). Severity of unilateral spatial neglect is an independent predictor of functional outcome after acute inpatient rehabilitation in individuals with right hemispheric stroke. *Archives of Physical Medicine and Rehabilitation*, 92, 1250–1255.
- Franceschini, M., La Porta, F., Agosti, M., & Massucci, M. (2010). Is health-related-quality of life of stroke patients influenced by neurological impairments at one year after stroke? *European Journal of Physical and Rehabilitation Medicine*, 46, 389–399.
- Gillen, R., Tennen, H., & McKee, T. (2005). Unilateral spatial neglect: Relation to rehabilitation outcomes in patients with right hemisphere stroke. *Archives of Physical Medicine and Rehabilitation*, 86, 763–767.
- Halligan, P. W., Cockburn, J., & Wilson, B. (1991). The behavioural assessment of visual neglect. *Neuropsychological Rehabilitation*, 1, 5–32.
- Hartman-Maeir, A., & Katz, N. (1995). Validity of the behavioral inattention test (BIT): Relationships with functional tasks. *American Journal of Occupational Therapy*, 49, 507–516.
- Heilman, K. M., Watson, R. T., & Walenstein, E. (1993). Neglect and related disorders. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (pp. 279–336). New York: Oxford University Press.
- Jehkonen, M., Ahonen, J. P., Dastidar, P., Koivisto, A. M., Laippala, P., Vilkki, J., & Molnar, G. (2000). Visual neglect as a predictor of functional outcome one year after stroke. *Acta Neurologica Scandinavica*, 101, 195–201.
- Jehkonen, M., Laihosalo, M., & Kettunen, J. E. (2006). Impact of neglect on functional outcome after stroke: A review of methodological issues and recent research findings. *Restorative Neurology and Neuroscience*, 24, 209–215.
- Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (2012). *Neuropsychological assessment* (5th ed.pp. 428–439). New York: Oxford University Press.
- Lopes, M. A., Ferreira, H. P., Carvalho, J. C., Cardoso, L., & Andre, C. (2007). Screening tests are not enough to detect hemineglect. *Arquivos de Neuro-Psiquiatria*, 65, 1192–1195.
- Wilson, B., Cockburn, J., & Halligan, P. W. (1987a). Behavioural inattention test manual. Hants/Los Angeles: Thames Valley Test Company/Western Psychological Services.
- Wilson, B., Cockburn, J., & Halligan, P. W. (1987b). Development of a behavioural test of visuospatial neglect. Archives of Physical Medicine and Rehabilitation, 68, 98–102.

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" have a very long history. It is based on the idea that all behaviors are learned and in the case of psychotherapy, these unhealthy behaviors can be changed.

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 Frederic (B. F.) 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 releases 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, obsessivesmoking cessation, anorexia, 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 (but not limited to):

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

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 systematic desensitization.

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 (e.g., insight oriented) 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.
- Modeling patients learn responses simply by observing other individuals and repeating their behavior.
- 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. These problems can range from aggression to agitation and from depression to nonadherence. 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). One approach to examining these behaviors sees behavioral dysfunction as more of a signal that a person is beyond their personal capacities to manage presenting challenges and thus requiring support that is contextually relevant. In this paradigm, "behavior" is seen as both a person's competencies and incompetencies in managing their environment, personal functioning, emotional/ behavioral stability, and independence.

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 selfmonitoring, 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, 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. Mark and Linda Sobell view alcoholic drinking as a discriminated, operant response that can be treated through aversive conditioning (electric shocks). Their research has important implications for future treatments since the experimental subjects functioned significantly better than controls post-intervention.

Cross-References

- Applied Behavior 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.
- Jacobs, H. (1993). Behavior analysis guidelines and brain injury rehabilitation: People, principles and programs. Gaithersburg: Aspen Publishing Company.
- Jacobs, H. (2014). Perspectives on behavior and acquired brain injury. *NeuroRehabilitation*, 34, 597–599.
- 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.
- Sobell, L., & Sobell, M. (2016). Individualized behavior therapy for alcoholics. *Behavior Therapy*, 47(6), 937–949.
- Wilson, K. (1997). Science and treatment development: Lessons from the history of behavior therapy. *Behavior Therapy*, 28, 547–558.

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. Despite behaviorism's historical avoidance of physiological explanations of behavior (Skinner 1950), those clinical neuropsychologists who include psychotherapy as part of their professional practice make use of classic and cognitive behavioral approaches in their work with brain injury survivors.

Cross-References

- Behavior Modification
- Cognitive Behavioral 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: Academic.
- 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: Sage.

- Skinner, B. F. (1950). Are theories of learning necessary? *Psychological Review*, 57(4), 193–216.
- Staddon, J. E. R. (2000). The new behaviorism: Mind, mechanism, and society. Philadelphia: Psychology Press.

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 statistics (e.g., t-tests, ANOVA). Standardized scores derived from neuropsychological measures are based upon (assume) normal distribution of the standardization sample. While this assumption provides a common metric that allows for direct comparison of performance between different measures, it is important to note that score distributions for a number of neuropsychological tests are non-normal (e.g., Boston Naming Test, Wisconsin Card Sorting Test, Mini-Mental Status Exam, Test of Memory Malingering). For this reason, selection of measures and interpretation of test findings must include consideration of score distributions (Strauss et al. 2006).

Cross-References

- Base Rate (Population)
- Cutoff Scores, Cutting Scores
- Intelligence Quotient
- ► Mental Age
- Percentiles
- Standard Scores

References and Readings

- Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (2012). *Neuropsychological assessment* (5th ed.). New York: Oxford University Press.
- Sattler, J. M. (2001). Assessment of children: Cognitive foundations (5th ed.). La Mesa: Jerome M. Sattler.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). A compendium of neuropsychological tests: Administration, norms, and commentary (3rd ed., pp. 3–43). New York: Oxford University Press.

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 and 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 reinnervation. 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 Lymepositive 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.

Bender Visual-Motor Gestalt Test II

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Synonyms

Bender-Gestalt, Second Edition; BG-II

Description

The Bender-Gestalt test was first published in 1938, as a brief measure of visual-motor functioning. 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 (BG-II), which was revised by Brannigan and Decker in 2003. The BG-II maintains many historical properties that appealed to clinicians, while improving the psychometric adequacy of the test. The BG-II is divided into two phases: copy and recall. The copy phase requires the duplication of 16 geometric images, shown sequentially on separate 3×5 cards, onto a blank piece of paper with a No. 2 pencil. The recall phase involves drawing these images from memory on a new sheet of paper. Using the Global Scoring System, the drawing productions are rated on a 5-point scale based on the similarity to the original image and yield percentile ranks, scaled scores, T-scores, and confidence intervals. Additionally, the BG-II also contains two supplemental measures to screen for specific difficulties and can be used to better understand low performance.

Like the original, the BG-II 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 BG-II 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 et al. 2006). Additionally, research with the BG-II has shed insight on the development of visual-motor abilities across the life-span(-Decker 2008). Specifically, using this measure it has been demonstrated that visual-motor integration rapidly matures into adolescence, gradually declines through adulthood, and rapidly decreases in late adulthood.

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 eventually the 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 techniques 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 sustained use of the measure.

Clinical Uses

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

The BG-II is appropriate for use with children as young as 4 years old to individuals over the age of 85 years old and typically takes no longer than 15 min to administer. It has been used extensively for educational, medical, and other purposes, particularly 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. (2008). Measuring growth and decline in visual motor processes with the Bender-Gestalt, Second Edition. *Journal of Psychoeducational Assessment*, 26, 3–15.
- 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.

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 et al. 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 and 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 and 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 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 ten 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 et al. (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 et al. 2005; Strauss et al. 2006). A multiple-choice recognition administration (Administration M, with alternate forms F and G) is also sometimes used to assess visual memory without visuoconstructional or motor coordination demands (Amieva et al. 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 and 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 auditoryvocal), 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. Later editions included a design copy administration and updated norms. The most recent revision was authored by Abigail Benton Sivan (Sivan 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.

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 to 69. (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 visuospatial disturbance 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 et al. 1989), as well as on copy and multiple-choice administrations (Arena and 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. Participants with right hemisphere damage achieved a lower total correct score on Administration D than 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). Poorer performance on the BVRT in learning disabilities has been linked with deficits in the identification of facial emotional expression (Dimitrovsky et al. 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 and Bowers 1993). Poorer performance is also evident in a subset of patients with schizophrenia and may result from abnormal patterns of visual scanning and fixation related to deficient attention (Obayashi et al. 2003) or be related to poor executive functions (Egan et al. 2011). Another clinical application is the inclusion of the BVRT in a neuropsychological battery for the prediction of driving safety in patients with early dementia (Dawson et al. 2009). The BVRT may also be useful in detecting malingering, which has been characterized by a greater number of errors, particularly distortion errors, than seen in neuropsychologically impaired patients (Suhr et al. 1997).

In evaluating results, it is important to consider that the BVRT may also be sensitive to individual differences that do not 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). Declines in executive function and attention with normal aging have been associated with lower BVRT scores and may be related to educational level or "cognitive reserve." In a large sample of healthy elderly adults, those with higher education performed better by using a more exhaustive search strategy in the multiple choice administration (Le Carret et al. 2003). The BVRT is used worldwide, and normative data have been published from more than a dozen countries (Mitrushina et al. 2005). Most studies have shown no gender differences. 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). Results from a large Columbian sample of school-aged children did not differ from North American norms (Rosselli et al. 2001), suggesting that when educational quality is similar, as is increasingly more common in developed countries, cross-cultural differences, if present, are relatively small.

See Also

- Short-Term Memory
- Visual-Motor Function
- ► Visuoperceptual
- Wechsler Memory Scale All Versions

Further Reading

- 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 de rétention visuelle (1965). Paris: Les Editions du Centre de Psychologie Applique.
- Benton, A. L. (1974). Revised visual retention test: Clinical and experimental applications (4th ed.). New York: Psychological Corporation.

- Dawson, J. D., Anderson, S. W., Uc, E. Y., Dastrup, E., & Rizzo, M. (2009). Predictors of driving safety in early Alzheimer disease. *Neurology*, 72, 521–527.
- Dimitrovsky, L., Spector, H., Levy-Shiff, R., & Vakil, E. (1998). Interpretation of facial expressions of affect in children with learning disabilities with verbal or nonverbal deficits. *Journal of Learning Disabilities*, 31, 286–292.
- Egan, G. J., Hasenkamp, W., Wilcox, L., Green, A., Hsu, N., Boshoven, W., Lewison, B., Keyes, M. D., & Duncan, E. (2011). Declarative memory and WCST-64 performance in subjects with schizophrenia and healthy controls. *Psychiatry Research*, 188, 191–196.
- Kawas, C. H., Corrada, M. M., Brookmeyer, R., Morrison, A., Resnick, S. M., Zonderman, A. B., & Arenberg, D. (2003). Visual memory predicts Alzheimer's disease more than a decade before diagnosis. *Neurology*, 60, 1089–1093.
- Le Carret, N., Rainville, C., Lechevailler, N., Lafont, S., Letenneur, L., & Fabrigoule, C. (2003). Influence of education on the Benton visual retention test performance as mediated by a strategic search component. *Brain and Cognition*, 53, 408–411.
- 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: Psychological Corporation.
- Sivan, A. B., & Spreen, O. (1996). *Der Benton-Test* (7th ed.). Bern: 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.

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 brainbehavior studies in aphasia, visuospatial abilities, hemispheric specialization, and other cognitive processes. Through the development of standardized tasks that stressed specific abilities, together with the 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 (Fig. 1)



Benton, Arthur (1909–2006), Fig. 1 Benton, Arthur (1909–2006)

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 childhoodonset damage to the 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 Dr. Russell Meyers (Chairman of the Division of Neurosurgery) and Dr. 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."

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 hypothesis-testing а 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 the 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 anymore 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 two 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

- American Board of Professional Psychology (ABPP)
- American Psychological Association (APA)
- Aphasia
- Benton Visual Retention Test
- Clinical Neuropsychology
- Facial Recognition Test
- ► Flexible Battery
- ► Frontal Lobe
- 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

- Ackerly, S. S., & Benton, A. L. (1948). Report of a case of bilateral frontal lobe defect. In *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 fingerlocalization 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. (1964a). Contributions to aphasia before Broca. Cortex, 1, 314–327.
- Benton, A. L. (1964b). 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., & 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.
- Benton, A. L., Hamsher, K. deS., Varney, N. R., & Spreen, O. (1983). Contributions to neuropsychological assessment. New York: Oxford University Press.
- Levin, H. S., & Benton, A. L. (1975). Temporal orientation in patients with brain disease. *Applied Neurophysiol*ogy, 38, 56–60.

Ben-Yishay, Yehuda (1933-)

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Major Appointments

 1964–1966 Assistant Professor of Clinical Rehabilitation Medicine, New York University School of Medicine

- 1967–1974 Associate Professor of Clinical Rehabilitation Medicine, New York University School of Medicine
- 1975–2011 Tenured Professor of Clinical Rehabilitation Medicine, New York University School of Medicine
- 1975–1976 Visiting Professor, Tel Aviv University, Department of Psychology
- 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 Steinach, Germany
- 1996–2011 Assistant Chief of Behavioral Sciences, Rusk Institute of Rehabilitation
- 1975–2011 Tenured Professor of Clinical Rehabilitation Medicine, New York University of School of Medicine

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 Lifetime 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: 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.
- Dr. Ben-Yishay's work 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 on 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 a research project 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.

Ben-Yishay 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. In 1964, Dr. Ben-Yishay joined the faculty at New York University. 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 to 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 of the study were impressive and were followed in September of 1978 by a 5-year research grant on brain injury rehabilitation at New York University (NYU) Rusk Rehabilitation Head Trauma 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 Ben-Yishay's work and established programs built upon principles learned under his tutelage. Ben-Yishay maintains that the objective of all neurorehabilitation interventions is to optimize the person's compensatory repertoire, including helping the individual in mastering and reliably applying learned compensatory skills in his or her postrehabilitation life.

Dr. Ben-Yishay's work in the area of holistic brain injury rehabilitation continues to the present day, and he has earned worldwide recognition for his work. He has received 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 his many international committee and consultant positions, Dr. Ben-Yishay has 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.*

Of all of his achievements, Dr. Ben-Yishay greatest satisfaction stems from the programs all over the world that subscribe to his philosophy of brain injury rehabilitation and the many acknowledgments of his influence on clinical practice (personal communication, July 15, 2016).

Dr. Ben-Yishay formally retired in 2009 and has since volunteered at the NYU Rusk Rehabilitation Day Program. He continues to work on several publications, including work identifying the major predictors of successful outcomes of intensive neuropsychological rehabilitation and patient acceptance of the limitations imposed by brain injury. In regard to the future of the field, Dr. Ben-Yishay believes the "therapeutic community" portion of the holistic approach merits a wider application in order to improve outcomes (personal communication, July 15, 2016).

Cross-References

- ► Christensen, Anne-Lise (1926–)
- Cognitive Rehabilitation
- ▶ Goldstein, Kurt (1878–1965)

References and Readings

- Ben-Yishay, Y. (1996). Reflections on the evolution of the therapeutic milieu concept. *Neuropsychological Rehabilitation*, 6(4), 327–343.
- Ben-Yishay, Y. (2000). Postacute neuropsychological rehabilitation: A holistic perspective. In A. Christensen & B. P. Uzzell (Eds.), *International*

handbook of neuropsychological rehabilitation (pp. 127–135). Dordrecht: Kluwer Academic/Plenum Publishers.

- Ben-Yishay, Y. (2008). Foreword. In F. Gracey and T. Ownsworth (Eds). The self and identity in rehabilitation: A special issue of the journal *Neuropsychological Rehabilitation*,18(5), 513–521.
- Ben-Yishay, Y., & Diller, L. (1993). Cognitive remediation in traumatic brain injury: Update and issues. Archives of Physical Medicine and Rehabilitation, 74(2), 204–213.
- Ben-Yishay, Y., & Diller, L. (2008). Kurt Goldstein's holistic ideas: An alternative, or complementary, approach to the management of traumatically brain injured individuals. US Neurology, 4(1), 79–80.
- Ben-Yishay, Y., & Diller, L. (2016). Turning points: Positive outcomes of insensitive psychotherapeutic interventions in holistic neuropsychological rehabilitation settings. Youngsville: Lash and Associates Publishing.
- Ben-Yishay, Y., & Gold, J. (1990). Therapeutic milieu approach to neuropsychological rehabilitation. In R. L. Wood (Ed.), *Neurobehavioral sequelae of traumatic brain injury* (pp. 194–215). London: Taylor and Francis.
- Ben-Yishay, Y., & Lakin, P. (1989). Structured group treatment for brain-injury survivors. In D. Ellis & A. L. Christensen (Eds.), *Neuropsychological treatment after brain injury* (pp. 271–295). Boston: Kluwer Academic.
- Ben-Yishay, Y., Ben-Nachum, Z., Cohen, A., Gross, Y., Hoofien, D., Rattok, J., & Diller, L. (1978). Digest of a two-year comprehensive clinical research program for out-patient head injured Israeli veterans. NYU Rehabilitation Monograph, 60, 1–61.
- Ben-Yishay, Y., Rattok, J., Lakin, P., Piasetsky, E. B., Ross, B., Silver, S., ... & Ezrachi, O. (1985). Neuropsychologic rehabilitation: Quest for a holistic approach. *Seminars in Neurology*, 5(3), 252–259.
- Ben-Yishay, Y., Silver, S. M., Piasetsky, E., & Rattok, J. (1987). Relationship between employability and vocational outcome after intensive holistic cognitive rehabilitation. *The Journal of Head Trauma Rehabilitation*, 2(1), 35–48.
- Ezrachi, O., Ben-Yishay, Y., Kay, T., Diller, L., & Rattok, J. (1991). Predicting employment in traumatic brain injury following neuropsychological rehabilitation. *The Journal of Head Trauma Rehabilitation*, 6(3), 71–84.
- Prigatano, G. P., & Ben-Yishay, Y. (1999). Psychotherapy and psychotherapeutic interventions in brain injury rehabilitation. In M. Rosenthal, E. R. Griffith, J. Korutzer, & B. Pentland (Eds.), *Rehabilitation of the adult and child with traumatic brain injury* (3rd ed.). Philadelphia: F.A. Davis.
- Rattok, J., Ross, B., Ben-Yishay, Y., Ezrachi, O., Silver, S., Lakin, P., & ... Diller, L. (1992). Outcome of different treatment mixes in a multidimensional neuropsychological rehabilitation program. *Neuropsychology*, 6(4), 395–415.

Benzodiazepines

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Synonyms

Anxiolytics; Sedative-hypnotics

Definition

Benzodiazepines belong to a class of medications known as sedative-hypnotics. The benzodiazepine molecule binds to the subtype A portion of the protein receptor of the primary inhibitory neurotransmitter substance in the brain, gamma aminobutyric acid (GABA). The simultaneous binding of the endogenous neurotransmitter GABA on the GABA-A postsynaptic receptor increases the frequency of the opening of the chloride channel, allowing greater amounts of this negatively charged anion, chloride, to rapidly enter the cell due to the concentration gradient. The additional entry of chloride into the cytoplasm hyperpolarizes the cell, which reduces depolarization, or firing, of the cell. Hence, greater stimulation is required for cell firing. This is known as the GABA-benzodiazepine receptor complex (Stahl 2004).

Benzodiazepines 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 et al. 2009), and alcohol withdrawal syndrome (Ntais et al. 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 and Stewart 2002), and postural instability (with increased risk of falls) among the elderly (Allain et al. 2005). Chronic use also carries the risk of substance dependence and abuse and cognitive impairment with prolonged use at high doses (Stewart 2005; Barker et al. 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.).

Current Knowledge

Current uses of benzodiazepines include the treatment of spasticity (Gold and Oreja-Guevara 2013) and tremor (Meador et al. 2016) in patients with multiple sclerosis. 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 disorder (Bourin and 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).

See Also

- Anxiolytics
- Barbiturates

References

- 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 hypnotics 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 longterm 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.
- Gold, R., & Oreja-Guevara, C. (2013). Advances in the management of multiple sclerosis spasticity: Multiple sclerosis spasticity guidelines. *Expert Review of Neurotherapeutics*, 13(12 Suppl), 55–59.
- Iversen, L. L., Iversen, S. D., Bloom, F. E., & Roth, R. H. (2009). Antidepressants and anxiolytics. In *Introduction to neuropsychopharmacology* (pp. 306–335). New York: Oxford University Press.
- Meador, W., Salter, A. R., & Rinker, J. R., 2nd. (2016). Symptomatic management of multiple sclerosisassociated tremor among participants in the NARCOMS registry. *International Journal of MS Care*, 18, 147–153.
- Montplaisir, J., Hawa, R., Moller, H., Morin, C., Fortin, M., Matte, J., Reinish, L., & Shapiro, C. M. (2003). Zopiclone and zaleplon vs benzodiazepines 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.
- Stahl, S. M. (2013). Anxiolytics and sedative hypnotics. In Essential psychopharmacology: Neuroscientific basis and practical applications (pp. 297–333). New York: Cambridge University Press.

Berg Balance Scale

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Synonyms

7-item BBS-3P; BBS

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 to 21, and 20 to 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. For individuals with multiple sclerosis, the minimal clinically important difference has been determined to be 3 points.

Although originally designed to screen older adults for fall risk, the BBS has subsequently been validated for persons with stroke, multiple sclerosis, Parkinson's disease, and chronic obstructive pulmonary disease (COPD).

Cross-References

- Balance Disorders
- Multiple Sclerosis
- Parkinson's Disease
- ► 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., & Gayton, D. (1989). Measuring balance in the elderly: Preliminary development of an instrument. *Physiotherapy Canada*, 41, 304–311.
- 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.
- 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.
- Gervasoni, E., Jonsdottir, J., Montesano, A., & Cattaneo, D. (2016). Minimal clinically important difference of berg balance scale in people with multiple sclerosis.

Archives of Physical Medicine and Rehabilitation, epub.

- Internet Stroke Center. (2007). *Berg balance scale*. http:// www.strokecenter.org/Trials/scales/berg.html. Accessed 19 May 2010.
- Jacome, C., Cruz, J., Olivera, A., & Marques, A. (2016). Validity, reliability, and ability to identify fall status of the berg balance scale, BESTest, Mini-BESTest, and Brief-BESTest in patients with copd. *Physical Therapy*, 96, 1807–1815.
- 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. *The 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.

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, and research does not support that performance on cognitive testing is uniform across different tests or cognitive domains. In fact, abnormal performance on some proportion of neuropsychological testing has proven to be psychometrically normal (Binder et al. 2009).

Cross-References

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

References and Readings

- Binder, L. M., Iverson, G. L., & Brooks, B. L. (2009). TO err is human: "Abnormal" neuropsychological scores and variability are common in healthy adults. *Archives* of *Clinical Neuropsychology*, 24, 31–46.
- Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (2012). The rationale of deficit measurement. In *Neuropsychological assessment* (5th ed.). 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 proinflammatory proteins (cytokines). There are three FDA approved beta interferons available in the US - Avonex[®] (INF- β 1a), Betaseron[®] (INF- β 1b), and Rebif[®] (INF- β 1a). 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 15 Feb 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.

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.

Bias

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Synonyms

Partiality; Prejudice

Definition

Faust et al. (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 fourcomponent 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 Jr., J. B. (1991). Brain damage claims: Coping with neuropsychological evidence. Marina del Rey: 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: 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 (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 and 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 et al. 1994) and found bicycle drawing to be more sensitive to brain dysfunction in children with visualspatial 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, 1997a, b), 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 et al. 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 Visual-Motor Gestalt Test II
- 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: 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.

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 Ansaldo and Ghazi Saidi 2014; Lorenzen and Murray 2008; Pearce 2005), 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. Neuroimaging research suggests that different languages are most likely to share the same brain regions when the second language is acquired earlier, with age of second-language acquisition treated as a continuous variable (Nichols and Joanisse 2016) or as a categorical variable where a late age of acquisition is defined as 10 years or older (e.g., Wartenburger et al. 2003). Bilinguals' languages also show more overlap when both are highly proficient (e.g., Abutalebi and Green 2007; Chee et al. 1999; Golestani et al. 2006; Klein et al. 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 et al. 2007).

Additional brain areas have also been identiduring bilingual language processing, fied pointing to a complex neural network involved in the cognitive control of bilinguals' two languages (e.g., Green and Abutalebi 2013). For example, 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 et al. 2004). Increased activation in dorsolateral prefrontal cortex, anterior cingulate gyrus, and supramarginal gyrus has been observed during language switching (Hernandez et al. 2000; Price et al. 1999; Wang et al. 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). Altogether, the neural network that supports language control in bilinguals likely includes anterior cingulate cortex, presupplementary motor area, prefrontal and inferior frontal cortex, caudate nucleus/putamen/thalamus, left inferior parietal lobule, and the cerebellum (Green and Abutalebi 2013).

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 postonset). During this phase, availability of representations may vary because of diaschisis. As impairment patterns stabilize during the postacute phase (up to 5 months postonset), language impairment becomes more directly related to site of lesion and damage to specific linguistic and cognitive 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 et al. 1999).

Parallel impairment and recovery is the most prevalent pattern observed in bilingual aphasia (e.g., Paradis 2001), 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 inadvertent and uncontrolled language by 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 frequently less impaired in bilingual individuals with aphasia than naming and translation of noncognate words (key – llave, Goral et al. 2006; Kohnert 2004; Roberts and Deslauriers 1999). However, variability in such cognate effects has been demonstrated across individuals with aphasia, ranging from facilitation to interference effects (Hughes and Tainturier 2015). In general, aspects of bilinguals' languages that are more shared are also more resistant to impairment (e.g., Kiran and 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 et al. 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 higher-ranked cues more likely to be preserved. For example, English-speaking individuals with aphasia were 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 reviews of bilingual aphasia by Lorenzen and Murray (2008) and Ansaldo and Ghazi Saidi (2014).

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, providing systematic ways to assess aphasia in more than 59 languages, including cross-linguistic interactions in an even larger combination of language pairs. Tasks on the BAT are equivalent in linguistic complexity across languages and cover assessment of multiple 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 premorbid proficiency in each language that is equivalent to at least 400 languagelearning 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. It may be possible to distinguish pathological mixing from nonpathological mixing, although such an analysis should be embedded within a careful documentation of code-switching practices characterizing the patient's community.

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 cross-linguistic 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 nontarget 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 and 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. Cross-linguistic generalization is most likely to occur when shared representations are targeted for treatment, cross-linguistic 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 a patient's weaker or equally dominant language may generalize to their other language, especially when treatment targets are similar across languages (e.g., Edmonds and Kiran 2004). Generalization of treatment effects across languages has been shown when cognate words

are used in treatment (Kohnert 2004, but see Kurland & Falcon for cognate interference during treatment 2011), when semantic features are treated (Edmonds and Kiran 2004, 2006; Knoph et al. 2015), when general cognitive function is treated (Kohnert 2004), and may be more likely when languages are structurally similar to each other (e.g., Ansaldo and Ghazi Saidi 2014; Knoph et al. 2015). However, generalization from a stronger to a weaker language is less likely and, in general, cross-linguistic generalization does not always occur (e.g., Galvez and Hinckley 2003). In contrast, *within*-language generalization of treatment effects has been shown to be more substantial in the more proficient language (Edmonds and Kiran 2006; Kurland and Falcon 2011), a result that must also be considered when selecting treatment languages. Although language dominance patterns are frequently the same preand postmorbidly, this is not always the case. While much of the current treatment literature suggests that premorbid proficiency levels influence cross-linguistic generalization of treatment, others have argued that generalization patterns may also be determined by postmorbid proficiency levels (for a review, see Ansaldo and Ghazi Saidi 2014).

Future Directions

In 2011, 21% of the population older than 5 years of age (more than 60 million people) spoke a language other than English at home, up from 18% in 2000, 14% in 1990, and 11% in 1980 (Ryan 2013). 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 and Murray 2008). The US Department of Health and Human Services found that individuals of Latino origin were 33% less likely to receive necessary health-care services, compared to non-Latino white peers (Lorenzen and Murray 2008). With these changes in population dynamics, systematic evidence-based **Cr** research on the efficacy of various treatment approaches for bilingual aphasia has become

One avenue of research in bilingual aphasia that has been virtually unexplored is in the area of cognitive control, and more broadly, in attention and executive function skills. There exists a significant body of literature attempting to link language and communication outcomes in people with aphasia to deficits in nonverbal cognitive domains of attention and executive function (Erickson et al. 1996; Fridriksson et al. 2006; Helm-Estabrooks 2002; Hula and McNeil 2008; Keil and Kaszniak 2002; Murray 2012; Ramsberger 2005; Zinn et al. 2007). These nonverbal cognitive skills may be honed with bilingual experience, as bilinguals must suppress one language in favor of another language every time they speak. Although the evidence for bilingual effects on cognitive control abilities remains divided (e.g., Hilchey and Klein 2011; Paap and Greenberg 2013), balanced bilinguals have been shown to have cognitive advantages over monolinguals on nonlinguistic executive function tasks, especially as they age (Bialystok et al. 2004; Kave et al. 2008; Zied et al. 2004). Examination of the relation between cognitive control and language skills in bilinguals with aphasia will extend current understanding of the cognitive foundations of bilingual language processing (e.g., Dash and Kar 2014). Furthermore, given the important role of the executive control system in both aphasia and 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. Recent evidence suggests that bilingualism may influence cognitive outcomes after stroke. In a study of 608 patients with ischemic stroke, Alladi et al. (2016) found that a larger proportion of bilinguals had normal cognition compared with monolinguals, although there were no differences in the frequency of aphasia for bilinguals and monolinguals. 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., & Green, D. (2007). Bilingual language production: The neurocognition of language representation and control. *Journal of Neurolinguistics*, 20, 242–275.
- 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 eventrelated fMRI study in bilinguals. *Journal of Neuroscience*, 27, 13762–13769.
- Alladi, S., Bak, T. H., Mekala, S., Rajan, A., Chaudhuri, J. R., Mioshi, E., Krovvidi, R., Surampudi, B., Duggirala, V., & Kaul, S. (2016). Impact of bilingualism on cognitive outcome after stroke. *Stroke*, 47, 1–4.
- Ansaldo, A. I., & Ghazi Saidi, L. (2014). Aphasia therapy in the age of globalization: Cross-linguistic therapy effects in bilingual aphasia. *Behavioral Neurology*, 2014, 1–10.
- 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: 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: 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.
- Dash, T., & Kar, B. R. (2014). Bilingual language control and general purpose cognitive control among individuals with bilingual aphasia: Evidence based on negative priming and flanker tasks. *Behavioral Neurology*, 2014, 1–20.
- 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

increasingly necessary.

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.
- Erickson, R. J., Goldinger, S. D., & LaPointe, L. L. (1996). Auditory vigilance in aphasic individuals: Detecting nonlinguistic stimuli with full or divided attention. *Brain and Cognition*, 30, 244–253.
- Fabbro, F. (2001). The bilingual brain: Bilingual aphasia. Brain and Language, 79, 201–210.
- Fridriksson, J., Nettles, C., Davis, M., Morrow, L., & Montgomery, A. (2006). Functional communication and executive function in aphasia. *Clinical Linguistics* & *Phonetics*, 20, 401–410.
- 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.
- Green, D. W., & Abutalebi, J. (2013). Language control in bilinguals: The adaptive control hypothesis. *Journal of Cognitive Psychology*, 25, 515–530.
- Helm-Estabrooks, N. (2002). Cognition and aphasia: A discussion and a study. *Journal of Communication Disorders*, 35, 171–186.
- 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.
- Hilchey, M. D., & Klein, R. M. (2011). Are there bilingual advantages on nonlinguistic interference tasks? Implications for the plasticity of executive control processes. *Psychonomic Bulletin & Review*, 18, 625–658.
- Hughes, E., & Tainturier, M.-J. (2015). The cognate advantage in bilingual aphasia: Now you see it, now you don't. Frontiers in Psychology, 6, Conference Abstract: Academy of Aphasia 53rd Annual Meeting. https://doi. org/10.3389/conf.fpsyg.2015.65.00086.
- Hula, W. D., & McNeil, M. R. (2008). Models of attention and dual-task performance as explanatory constructs in aphasia. *Seminars in Speech and Language*, 29, 169–187.
- 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: 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.

- Keil, K., & Kaszniak, A. W. (2002). Examining executive function in individuals with brain injury: A review. *Aphasiology*, 16, 305–335.
- 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.
- Knoph, M. I. N., Lind, M., & Simonsen, H. G. (2015). Semantic feature analysis targeting verbs in a quadrilingual speaker with aphasia. *Aphasiology*, 29, 1473–1496.
- Kohnert, K. (2004). Cognitive and cognate-based treatments for bilingual aphasia: A case study. *Brain and Language*, 91, 294–302.
- Kurland, J., & Falcon, M. (2011). Effects of cognate status and language of therapy during intensive semantic naming treatment in a case of severe nonfluent bilingual aphasia. *Clinical Linguistics & Phonetics*, 25, 584–600.
- 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 Lan*guage, 66, 249–274.
- Murray, L. (2012). Attention and other cognitive deficits in aphasia: Present and relation to language and communication measures. *American Journal of Speech-Language Pathology*, 21, S51–S64.
- Nichols, E. S., & Joanisse, M. F. (2016). Functional activity and white matter microstructure reveal the independent effects of age of acquisition and proficiency on second-language learning. *NeuroImage*, 143, 15–25.
- Paap, K. R., & Greenberg, Z. I. (2013). There is no coherent evidence for a bilingual advantage in executive processing. *Cognitive Psychology*, 66, 232–258.

- Paradis, M. (1987). *The assessment of bilingual aphasia*. Hillsdale: 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: Erlbaum.
- 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.
- Ramsberger, G. (2005). Achieving conversational success in aphasia by focusing on non-linguistic cognitive skills: A potentially promising new approach. *Aphasiology*, 19, 1066–1073.
- Roberts, P. M., & Deslauriers, L. (1999). Picture naming of cognate and non-cognate nouns in bilingual aphasia. *Journal of Communication Disorders*, 32, 1–22.
- Ryan, C. (2013). Language use in the United States: 2011. http://www.census.gov/content/dam/Census/library/ publications/2013/acs/acs-22.pdf. Retrieved 11 Mar 2016.
- 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.
- 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 crosslinguistic 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.
- Zinn, S., Bosworth, H. B., Hoenig, H. M., & Swartwelder, H. S. (2007). Executive function deficits in acute stroke. Archives of Physical Medicine and Rehabilitation, 88, 173–180.

Binge-Eating Disorder

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Synonyms

Compulsive eating

Definition

Binge-eating disorder is defined in the *Diagnostic* and Statistical Manual of Mental Disorders (5th ed.; DSM-5; American Psychiatric Association 2013) as a feeding and eating disorder characterized by repeated instances of binge eating in which the individual consumes an amount of food during a limited time period that is proportionally larger than what would be typically eaten during a similar time period. Binge-eating episodes are accompanied by a sense of lack of control. While a binge-eating episode may occur in one setting, it may also begin in one setting, such as a restaurant, and continue upon returning home. Binge-eating episodes are characterized by the amount of food consumed, and by marked distress. Individuals may report eating very quickly and eating until uncomfortably full. These episodes typically occur in secrecy or social isolation and individuals report negative affect.

Categorization

The disorder is classified with the feeding and eating disorders in DSM-5.

Current Knowledge

Development and Course

The development of binge-eating disorder is common in adolescents and young adults but can occur later in life. Individuals typically fall within a normal to obese weight range; however, bingeeating disorder is distinct from obesity. The disorder is slightly more common in females (1.6%) than in males (0.8%; APA 2013). Binge-eating disorder is persistent, but remission rates are higher for than for anorexia nervosa or bulimia nervosa. The condition is associated with social maladjustment, poor quality of life, obesity, and increased mortality. Mood disorders and anxiety disorders commonly co-occur, as do substance abuse disorders to a lesser extent.

Assessment and Treatment

Diagnosis typically involves medical and psychiatric evaluation. Individuals seeking treatment for binge-eating disorder are typically older than those seeking treatment for bulimia nervosa or anorexia nervosa. Treatment typically involves pharmacotherapy and psychotherapy. Medications with some empirical evidence include selective serotonin reuptake inhibitors, tricyclic antidepressants, anticonvulsants, and antiobesity medications (Bulik et al. 2007). Empirically supported psychotherapeutic interventions include cognitive-behavioral therapy and dialectical behavior therapy.

See Also

- Anorexia Nervosa
- Bulimia Nervosa
- ► Feeding and Eating Disorders

References and Readings

- American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (DSM-5[®]).
 Washington, DC: American Psychiatric Association.
- Berkman, N. D., Brownley, K. A., Peat, C. M., Lohr, K. N., Cullen, K. E., Morgan, L. C., ..., Bulik, C. M. (2015). Management and outcomes of binge-eating disorder. Comparative Effectiveness Review No. 160. (Prepared by the RTI International-University of North Carolina Evidenced-based Practice Center under Contract No. 290-2012-00008-I.) AHRQ Publication No. 15(16)-EHC030-EF. Rockville, MD: Agency for Healthcare Research and Quality; December 2015. www.effectivehealthcare.ahrq.gov/reports/final.cfm.

- Bulik, C., Brownley, K., & Shapiro, J. (2007). Diagnosis and management of binge eating disorder. *World Psychiatry*, 6(3), 142–148.
- Cossrow, N., Pawaskar, M., Witt, E. A., Ming, E. E., Victor, T. W., Herman, B. K., ..., Erder, M. H. (2016). Estimating the prevalence of binge eating disorder in a community sample from the United States: Comparing DSM-IV-TR and DSM-5 criteria. *The Journal of Clinical Psychiatry*, 77(8), e968–e974.

Binocular Disparity

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Human eyes are separated by about 50-75 mm between pupils (Dodgson 2004). Therefore, each eye views the world in a slightly different way. The difference between these images is referred to as binocular disparity and provides important information that is not available from either image alone. The amount of disparity depends on the difference in the distance of the two objects and the distance of the fixation point. The greater the disparity, or distance, between the two images, the closer the object is to the fixation point. Binocular disparity is a necessary condition for stereopsis, which is the sense of depth the brain generates from information obtained by the left and right eye. This helps us to see the world in three dimensions, rather than two dimensions.

The idea that binocular disparity contributes to depth perception was first described by Sir Charles Wheatstone in the nineteenth century after he invented the stereoscope, a device used for observing pictures in three dimensions (Qian 1997). Since then, much research has focused on unraveling how the brain processes these disparities, particularly in animal models. A seminal study by Barlow et al. (1967) conducted in the cat's primary visual cortex found that neurons are tuned, or excited, at different distances. A subsequent study by Poggio and Fischer (1977) described four types of depth cells in the striate and prestriate cortex of the rhesus monkey: (1) "tuned excitatory neurons," which respond to a narrow range of depth around the fixation point, (2) "tuned inhibitory neurons," whose responses are suppressed by stimuli at or close to the fixation point, (3) "near neurons," which respond to stimuli near the fixation point and suppress information behind it, and (4) "far neurons," which respond to information behind the fixation point and suppress information near it. While Poggio and collaborators classified cells into these discrete categories, additional research suggests there is a continuous distribution of these cells (LeVay and Voigt 1988). Nevertheless, recordings indicate that disparity neurons are primarily located in V2, V3/V3A, and MT/V5, with weak clustering shown in V1 (Parker 2007).

In humans, the architecture of disparity is less understood. This is partially due to differences in neuronal firing for absolute versus relative disparities (Neri 2005). When looking at a pair of objects that are different distances apart, absolute disparity refers to the differences between the two retinal images generated by the object alone with respect to the fixation point and relative disparity refers to the difference between the two absolute disparities (Neri 2005; Parker 2007). Humans are more sensitive to relative, compared to absolute, disparities (Westheimer 1979). Both the dorsal and ventral streams process binocular cues; however, each stream carries out different types of processing. In animals, the dorsal stream is involved in processing relative disparity, particularly as it relates to spatially extended surfaces (Roy et al. 1992; Upadhyay et al. 2000). In contrast, the ventral stream is also sensitive to relative disparity but appears to be selectivity involved in the relative depth of objects and their three-dimensional configurations (Janssen et al. 2000, 2001; Uka et al. 2005; Umeda et al. 2007). Neurons in the dorsal and ventral streams do fire for both relative and absolute disparities, but, to date, no single cortical region in humans has been found to

be solely involved in relative disparity (Parker 2007), though a recent functional magnetic resonance imaging study showed that disparity preferences for depth perception were clustered in dorsal visual areas, including V3A and V3B/KO (Goncalves et al. 2015).

References

- Barlow, H. B., Blakemore, C., & Pettigrew, J. D. (1967). The neural mechanism of binocular depth discrimination. *The Journal of Physiology*, 193(2), 327–342.
- Dodgson, N. A. (2004). Variation and extrema of human interpupillary distance. In M. T. Bolas, A. J. Woods, J. O. Merritt, & S. A. Benton (Eds.), *Proceedings of SPIE: Stereoscopic displays and virtual reality systems* XI (Vol. 5291, pp. 36–46). San Jose. https://doi.org/ 10.1117/12.529999
- Goncalves, N. R., Ban, H., Sanchez-Panchuelo, R. M., Francis, S. T., Schluppeck, D., & Welchman, A. E. (2015). 7 tesla FMRI reveals systematic functional organization for binocular disparity in dorsal visual cortex. *The Journal of Neuroscience*, 35(7), 3056–3072.
- Janssen, P., Vogels, R., & Orban, G. A. (2000). Threedimensional shape coding in inferior temporal cortex. *Neuron*, 27(2), 385–397.
- Janssen, P., Vogels, R., Liu, Y., & Orban, G. A. (2001). Macaque inferior temporal neurons are selective for three-dimensional boundaries and surfaces. *The Journal of Neuroscience*, 21(23), 9419–9429.
- LeVay, S., & Voigt, T. (1988). Ocular dominance and disparity coding in cat visual cortex. *Visual Neuroscience*, 1(4), 395–414.
- Neri, P. (2005). A stereoscopic look at visual cortex. Journal of Neurophysiology, 93(4), 1823–1826.
- Parker, A. J. (2007). Binocular depth perception and the cerebral cortex. *Nature Reviews. Neuroscience*, 8(5), 379–391.
- Poggio, G. F., & Fischer, B. (1977). Binocular interaction and depth sensitivity in striate and prestriate cortex of behaving rhesus monkey. *Journal of Neurophysiology*, 40(6), 1392–1405.
- Qian, N. (1997). Binocular disparity and the perception of depth. *Neuron*, 18(3), 359–368.
- Roy, J. P., Komatsu, H., & Wurtz, R. H. (1992). Disparity sensitivity of neurons in monkey extrastriate area MST. *The Journal of Neuroscience*, 12(7), 2478–2492.
- Uka, T., Tanabe, S., Watanabe, M., & Fujita, I. (2005). Neural correlates of fine depth discrimination in monkey inferior temporal cortex. *The Journal of Neuroscience*, 25(46), 10796–10802.
- Umeda, K., Tanabe, S., & Fujita, I. (2007). Representation of stereoscopic depth based on relative disparity in macaque area V4. *Journal of Neurophysiology*, 98(1), 241–252.

- Upadhyay, U. D., Page, W. K., & Duffy, C. J. (2000). MST responses to pursuit across optic flow with motion parallax. *Journal of Neurophysiology*, 84(2), 818–826.
- Westheimer, G. (1979). Cooperative neural processes involved in stereoscopic acuity. *Experimental Brain Research*, 36(3), 585–597.

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 diffuse white matter lesions and scattered multiple lacunes (Akiguchi et al. 2014).

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 (Akiguchi et al. 2014; Caplan 1995; Hachinski et al. 2006; Olsen and Clasen 1998; Pantoni and Garcia 1995; Rosenberg et al. 2015). 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% to 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. There is growing evidence that white matter pathology in BD is related to endothelial dysfunction and neuroinflammation (Huisa and Rosenberg 2014).

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 et al. 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., selective serotonin 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 et al. 2005). Antiplatelet therapy and statins have also been recommended for stroke prevention in BD (Huisa and Rosenberg 2014). 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

- Akiguchi, I., Budka, H., Shirakashi, Y., Woehrer, A., Watanabe, T., Shiino, A., Yamamoto, Y., Kawamoto, Y., Krampla, W., Jungwirth, S., & Fischer, P. (2014). MRI features of Binswanger's disease predict prognosis and associated pathology. *Annals of Clinical and Translational Neurology*, 1(10), 813–821.
- 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.

- Huisa, B. N., & Rosenberg, G. A. (2014). Binswanger's disease: Toward a diagnosis agreement and therapeutic approach. *Expert Review of Neurotherapeutics*, 14(10), 1203–1213.
- 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: Wiley.
- Roman, G. C. (2005). Vascular dementia prevention: A risk factor analysis. *Cerebrovascular Diseases*, 20(Suppl. 2), 91–100.
- Rosenberg, G. A., Wallin, A., Wardlaw, J. M., Markus, H. S., Montaner, J., Wolfson, L., Costantino, I., Zlokovic, B. V., Joutel, A., Dichgans, M., Duering, M., Schmidt, R., Korczyn, A. D., Grinberg, L. T., & Hachinski, V. (2015). Consensus statement for diagnosis of subcortical small vessel disease. *Journal of Cerebral Blood Flow and Metabolism, 36*(1). 1–13.
- Santamaria Ortiz, J., & Knight, P. V. (1994). Review: Binswanger's disease, leukoaraiosis and dementia. *Age and 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.

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 or blast 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 (TBI)

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.
- Goriely, A., Geers, M. G., Holzapfel, G. A., Jayamohan, J., Jerusalem, A., Sivaloganathan, S., Squier, W., van Dommelen, J. A., Waters, S., & Kuhl, E. (2015). Mechanics of the brain: Perspectives, challenges, and opportunities. *Biomechanical Models of Mechanobiology*, 14(5), 931–965.
- Oeur, R. A., Karton, C., Post, A., Rousseau, P., Hoshizaki, T. B., Marshall, S., Brien, S. E., Smith, A., Cusimano, M. D., & Gilchrist, M. D. (2015). A comparison of head dynamic response and brain tissue stress and strain using accident reconstructions for concussion, concussion with persistent postconcussive symptoms, and subdural hematoma. *Journal of Neurosurgery*, 123(2), 415–422.
- 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 582

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

Radiosurgery, Stereotactic 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.

Blast Effects

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Synonyms

Blast injury

Definition

Blast effects are injuries or symptoms resulting from a shock wave or blast caused by high-order or low-order explosives such as dynamite, bombs, or C-4 (CDC 2016). Effects resulting from blasts may vary from auditory problems to brain injury and are classified as primary, secondary, tertiary, or quaternary (CDC 2016). Primary blast effects are unique to high-order explosives and may include injuries such as concussion, eye rupture, or abdominal hemorrhage (CDC 2016). Secondary and tertiary effects may result from bomb fragments, flying debris, or blast wind with any body part being affected. Last, quaternary effects include explosion-related injuries, illness, or disease, such as burns, close or open brain injury, and breathing problems due to toxic fumes, dust, or smoke from a blast.

See Also

- ▶ Blast Injury
- Mild Traumatic Brain Injury
- Severe Brain Injury
- Traumatic Brain Injury (TBI)

References and Readings

- Center for Disease Control Injury Prevention (2016). Explosions and blast injuries: A primer for clinicians. Retrieved from: https://www.cdc.gov/masstrauma/pre paredness/primer.pdf
- Lash, M. (2017). Understanding the effects of concussion, blast, and brain injuries: A guide for families, veterans, and caregivers. Brainline Military. Retrieved from: http://www.brainlinemilitary.org/content/2008/11/under standing-effects-concussion-blast-and-brain-injuries-gui de-families-veterans-and-caregi pageall.html

Blast Injury

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Synonyms

This term is highly associated (but technically not synonymous) with mild traumatic brain injury and post-concussive symptoms.

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 a 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 front line) duty. Initially, these symptoms were considered to be 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 recent conflicts in the Middle East, given the high incidence of explosives utilized terrorists non-Coalition by and combatants.

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% during the recent conflicts in Iraq and Afghanistan. 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 recent conflicts. Overall, it has been estimated that up to 30% of all combat troops in Operations Iraqi Freedom and Enduring Freedom (OIF/OEF) 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 shock-wave approaching a speed of 8000 m/s 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-liquid interface, such as the tympanic membrane, lung, colon, etc. 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 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 a person's physical wounds from the 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 an object, a structure, the ground, other individuals, etc., often resulting in blunt force wounds.

4. Quaternary Blast Injury

Quaternary blast injuries 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 brain injuries resulting from explosions are classified as mild in nature. "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 (ACRM) are coming into wider acceptance and have largely been adopted by the military. The ACRM definition of mTBI includes at least one of the following symptoms: less than half an hour of loss of consciousness, less than 24 h of posttraumatic 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, many studies have indicated that factors such as loss of consciousness, resultant symptom profiles, and recovery courses do not appear to substantially 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., nervousness, irritability) symptoms are commonly seen initially after blast injuries. In civilian mTBI samples, these symptoms usually 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, these persistent symptoms have not been consistently defined, and many point out that the constellation of symptoms present are vague and lack specificity needed to identify them as constituting a true syndrome.

There is debate over whether the more chronic symptom constellation after mild TBI reflects a true neurological condition; this has particular relevance in blast injury, as the brief history of "shell shock" above illustrates. Those favoring a more neurological position cite animal models in which direct and indirect exposure to primary blasts causes structural, chemical, and electrophysiological changes in the brain. Additionally, some studies using functional MRI and diffuse tensor imaging in humans have reported cerebral alterations in some (albeit not all) persons who have sustained blast injuries. Conversely, 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 symptoms seen in disorders such as posttraumatic stress disorder (PTSD) is particularly noteworthy, given the high incidence of PTSD in military personnel who have experienced combat: Gaylord et al. (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 et al. (2008) reported that approximately 15% of soldiers surveyed after being returned home might meet criteria for both 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, concomitantly, mTBI symptoms can exacerbate and prolong PTSD symptoms). 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/her brain injury recovery.

Treatment of blast injuries 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 been made to carefully screen every wounded individual for other symptoms (e.g., tinnitus) that place them at higher risk for having sustained a TBI in a blast, to help ensure mTBIs are not underdiagnosed. The assessment process includes a thorough medical evaluation of the patient's current condition and a comprehensive interview that elicits historical information about past psychological treatment/coping, substance use, and combat exposure.

Neuropsychological evaluation is recommended to occur as early as possible to help identify postconcussive symptoms and clarify the diagnostic picture, enabling education and treatment efforts to proceed more quickly. Whereas there are cognitive deficits commonly seen after most mTBIs (e.g., slowed attention and information processing speed, motor slowness, executive dysfunction, and memory difficulties), the profile of cognitive weaknesses can be quite variable, necessitating a broad-based neuropsychological assessment (i.e., a sampling of all major cognitive domains). Additionally, as is the case in sports concussions, the symptom picture for many blast survivors 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., Neurobehavioral Symptom Inventory, 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 (including objective personality measures such as the MMPI-2 or PAI and instruments such as the PTSD Checklist), 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 performance on an effort test should not automatically be interpreted as an indicator 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 after mTBI is delivered. Specific treatments need to be tailored to the individual, in recognition that not all mTBIs are expressed identically. For instance, in a cluster analysis performed on 1341 servicemen who had sustained an mTBI in combat over the previous 2 years, Bailie et al. (2016) found four different subtypes: good recovery (low overall cognitive and affective symptoms; 37.8% of the sample), high PTSD but few cognitive symptoms (21.9%), elevated cognitive but few affective symptoms (21.5%), and mixed symptoms (18.6%). These results suggest an array of treatment strategies that is necessary to effectively address appropriate needs. Such treatment strategies would include medication (e.g., analgesics for pain, soporific medication for insomnia, antidepressants for affective symptoms), relaxation strategies for anxiety symptoms, psychotherapy for PTSD symptoms, and evidence-based cognitive rehabilitation.

Future Directions

The research literature in blast injury is still evolving. 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 other sources; investigation of the effect of multiple blast exposures; and investigation of how PTSD/affective distress differs from and interacts with mTBI. Studies should take into account situational factors such as combat exposure, combat intensity, length and number of deployments, as well as potentially moderating variables on symptom expression and recovery (e.g., substance use, pain intensity, sleep integrity). More prospective research is

clearly needed. Within neuropsychology, development of alternate forms for many tests is encouraged.

See Also

- ► Concussion
- Mild Traumatic Brain Injury
- Post-concussive Syndrome
- Post-Traumatic Stress Disorder
- Traumatic Brain Injury (TBI)

References and Readings

- Bailie, J., Kennedy, J., French, L., Marshall, K., Prokhorenko, O., Asmussen, S., Reid, M., Qashu, F., Brickell, T., & Lange, R. (2016). Profile analysis of the neurobehavioral and psychiatric symptoms following combat-related mild traumatic brain injury: Identification of subtypes. *The Journal of Head Trauma Rehabilitation*, 31(1), 2–12.
- 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, DC.
- Bogdanova, Y., & Verfaellie, M. (2012). Cognitive sequelae of blast-induced traumatic brain injury: Recovery and rehabilitation. *Neuropsychological Review*, 22, 4–20.
- 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.

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 selfcare (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 6 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 0.5 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 are 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 et al. 1968, 1988). A total cutoff 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 et al. 1983). Stern et al. (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, 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 3 sequencing tasks requiring concentration (Blessed et al. 1968, 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, 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 et al. 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 et al. 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).

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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 et al. 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 et al. 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 cutoff 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 State 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 cutoff 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 (Erkinjuntti et al. 1988). Education appears unrelated to DS scores (Erkinjuntti et al. 1988). African-American patients score higher on the DS than white patients (Hargrove et al. 1998). The DS has been translated and validated in Chinese, Korean, and Czech (Lam et al. 1997; Lee et al. 1999; Vajdickova et al. 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 healthcare 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 et al. 1988) because it measures functional aspects of dementia.

Cross-References

- Alzheimer's Disease
- Bristol Activities of Daily Living Scale
- Clinical Dementia Rating
- ▶ Dementia
- Dementia Rating Scale-2
- 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 Geriatrics 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 Korean Neuropsychiatric Association*, 38, 1365–1375.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
- 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 practitioners' opinion with assessments using the minimental 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., 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.
- Stern, Y., Hesdorffer, D., Sano, M., & Mayeux, R. (1990). Measurement and prediction of functional capacity in Alzheimer's disease. *Neurology*, 40(1), 8–14.
- 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.

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. Lawrence 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

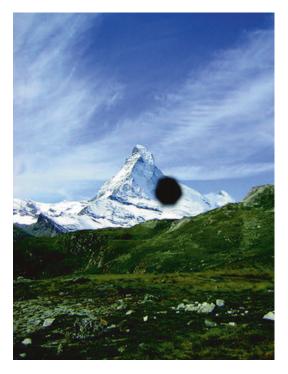
Blindsight results from brain damage to the primary visual cortex (V1) located in the posterior region of the occipital cortex, typically caused by a tumor, a hemorrhage, or some sort of brain trauma.

Natural History, Prognostic Factors, and Outcomes

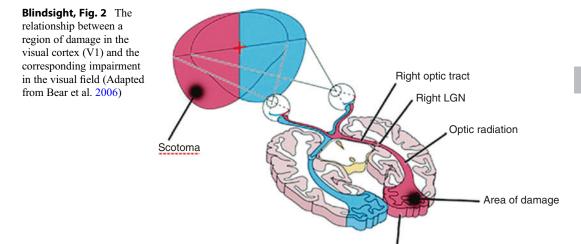
The first cases of blindsight were observed in war veterans with damage to their occipital lobe (Pöppel et al. 1973). These veterans had no conscious perception of stimuli in the damaged portion of their visual field yet were able to track a moving light presented there. The most extensive experimental 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).

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 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 the 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 - such 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 - such a condition is termed quadranopia. Figure 2 illustrates the pattern of



Blindsight, Fig. 1 The visual perception of a scotoma



Primary visual cortex

visual impairment and the corresponding scotoma that arises when only a small region of the 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 et al. 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 of 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, 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 (nonmatched 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 et al. 2006) and nonhuman primates (Dineen and 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 the 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 DB's 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: 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 neuro-sciences*. Cambridge, MA: MIT Press.

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 twodimensional 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.

Edith Kaplan: deceased.

Psychometric Data

Successful completion of the BD test requires a host of cognitive abilities (Kramer et al. 1991) including specific analytic and synthetic problem-solving strategies (Schorr et al. 1982). Analytic strategies refer to mental segmentation of the stimulus design into individual blocks. After mentally dividing the blocks into segments, 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, overreliance 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 the so-called constructional apraxia (Kleist, 1923, cited in Benton and 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 have undergone a commissurotomy who (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 \times 2 or 3 \times 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 \times 2 or 3 \times 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 et al. (1981) noted that similar behavior occurs in patients with focal right and left hemisphere lesions. Patients with right-sided lesions often break the 2 \times 2 or 3 \times 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 singleblock, 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 four or nine blocks as prescribed by the Wechsler test manual, Kaplan et al. (1991) suggests presenting the patient with nine blocks on all four-block test items and 12 blocks on all nine-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 \times 2 or 3 \times 3 grid configuration of the stimulus matrix and often produce distorted responses. Kaplan et al. (1991) provides examples of BD constructions produced by rightanterior 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.
- 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.
 - 1. **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.
 - 2. 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 (Zip-Williams et al. 2000) or brain injury (Wilde et al. 2000) lateralized to one side of the brain may also make these errors.

- 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° in relation to the model. Spatial, perceptual, or executive problems might underlie this difficulty.
- 2. 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.
- 3. 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 selfcorrect errors.
- 4. 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.
- 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 agerelated 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 psychomotor slowing are all more prevalent in individuals diagnosed with a dementia relative healthy older adult controls.

Cross-References

Constructional Apraxia

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: 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 nonpenetrating 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.

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 level (BAL) is 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 (4 oz) 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 to 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. The tendency to underestimate one's own blood alcohol level seems to pose an additional risk of impairment and injury risk. Additionally, speed of cognitive performance recovers as alcohol is metabolized and BAL decrease; however, accuracy may continue to remain impaired.

Cross-References

- ► Coma
- Executive Functioning
- 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.
- Harrison's Online Harrison's principles of internal medicine (16th edn.). Chapter 372: Alcohol and alcoholism.
- Laude, J. R., & Filmore, M. (2016). Drivers who selfestimate lower blood alcohol concentrations are riskier drivers after drinking. *Psychopharmacology (Berl)*, 233(8), 1387–1394. https://doi.org/10.1007/s00213-016-4233-x.
- Schweizer, T. A., & Vogel-Sprott, M. (2008). Alcoholimpaired speed and accuracy of cognitive functions: A review of acute tolerance and recover of cognitive performance. *Experimental and Clinical Psychopharmacology*, 16(3), 340–350.

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 and veins, as well as chambers and valves of the heart. Doppler ultrasound (US) may be used to diagnose vascular conditions such as cerebral vasospasm, cerebral or limb thrombosis, vascular stenosis, valvular heart disease, and 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 are used most often to study blood flow particularly in the legs, neck, and brain. 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 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 - usually a superficial one. 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 those of duplex Doppler. It is commonly used to evaluate

the flow of blood through vessels within solid organs.

Transcranial Doppler (TCD) is used to measure blood flow through the brain's blood vessels. It is becoming more widely used to evaluate for emboli, stenosis, vasospasm, and the risk of stroke.

Limitations to studies include obesity, cardiac arrhythmias, heart disease, and smoking within an hour of study. Studies are done in the inpatient and outpatient settings monitoring.

Variations in blood flow can affect microvascular and macrovascular beds for a wide range of clinical situations. There are new expanding techniques to study blood flow but ultrasound remains the commonly used techniques in hospital an outpatient clinical settings.

See Also

- Cerebral Blood Flow
- 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(5), S224–S234.
- 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.
- Roberts, D. R., & Forfia, P. R. (2011). Diagnosis and assessment of pulmonary vasculature disease by doppler echocardiography. *Pulmonary Circulation*, 1(2), 160–181.
- Rubens, D. J., Bhatt, S., Nedelka, S., & Cullinan, J. (2006). Doppler artifacts and pitfalls. *Radiologic Clinics of North America*, 44(6), 805–835.
- Webb, R. C., Ma, Y., Krishnan, L. Y., Yoon, S., et al. (2015). Epiderman devices for noninvasive, preise, and continuous mapping of macrovascular and microvascular blood flow. *Science Advances*, 1(9), 1–13.
- 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 (BOLD)

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Synonyms

BOLD

Definition

Blood-oxygen-level-dependent (BOLD) imaging is a technique used to generate images in functional MRI (fMRI) studies. The goal of this technique is to discern regional differences in cerebral blood flow in an effort to delineate more specific regional activity. This version of magnetic resonance imaging 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

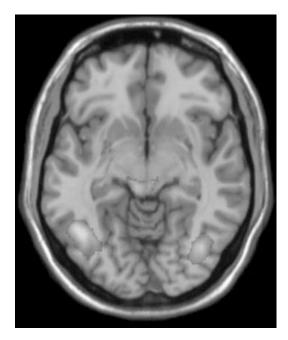
As early as 1890, Roy and Sherrington noted that regional cerebral blood flow increased in areas of neural activity. This increase in perfusion became detectable in vivo with the advent of positron emission tomography (PET), in which radioactive tracers are injected and their emitted radiation detected. It was not until the discovery of BOLD contrast in 1990 by Ogawa and colleagues at Bell Laboratories that it was possible to measure neuron-mediated changes in blood flow without radiation exposure. While fMRI relies on an evolving understanding of both the physiologic and biophysical origins of BOLD, this technique has become a powerful research modality in mapping brain activation in animals and humans.

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 neuronal and glial tissue. BOLD technique represents the relationship between oxygen delivery and oxygen extraction, rather than oxygen consumption itself – a more direct measure of tissue metabolic activity. The clinician and research scientist should appreciate the delay of several seconds between the changes in neural activity and changes in associated blood flow. Thus, BOLD imaging technique requires mathematical modeling of the "hemodynamic response function" (the increase and subsequent return to baseline of flow associated with a neural event). The derived BOLD signal should correlate to resting neuronal states or in response to specific behavioral and cognitive events that 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 neuronal activity, its signal detected by fMRI reflects changes in deoxyhemoglobin derived from cerebral mediated changes in blood oxygenation and blood flow. This phenomenon appears to be closely linked to neuronal activity, a process referred to as neurovascular coupling. Research exploring the neurovascular coupling mechanisms and the BOLD signal are opportunities to more completely understand the underlying pathobiological mechanisms underlying brain development, disease states, and aging.

BOLD technique is also an important concept in resting state fMRI studies. These concepts offer a broader understanding of human brain connectivity and especially how it is influenced by a variety of disease states and its inherent underlying pathophysiologic mechanisms. In addition, BOLD imaging techniques and fMRI 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



Blood Oxygen Level Dependent (BOLD), Fig. 1 Blood–oxygen-level dependent. 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

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 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 considerable distances may separate components of these neural networks, it is of interest to understand how they communicate with each other in the performance of specific cognitive 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. 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 do this. 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.

Conclusions

Although BOLD and other fMRI 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. A crucial perspective in interpreting BOLD and fMRI results must comprehensively consider BOLD's vascular and metabolic underpinnings. This understanding is important to analyze under both resting conditions and in disease states. 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.

Cross-References

- Functional Imaging
- Magnetic Resonance Imaging

References and Readings

- Barkhof, F., Haller, S., & Rombouts, S. A. (2014). Restingstate functional MR imaging: A new window to the brain. *Radiology*, 272(1), 29–49.
- Hillman, E. M. (2014). Coupling mechanism and significance of the BOLD signal: A status report. *Annual Review of Neuroscience*, 37, 161–181.
- Kida, I., & Hyder, F. (2006). Physiology of functional magnetic resonance imaging: Energetics and function. *Methods in Molecular Medicine*, 124, 175–195.
- Kim, S. G., & Ogawa, S. (2012). Biophysical and physiological origins of blood oxygenation level-dependent fMRI signals. *Journal of Cerebral Blood Flow and Metabolism*, 32(7), 188–1206.
- Lee, M. H., Smyser, C. D., & Shimony, J. S. (2013). Resting-state fMRI: A review of methods and clinical applications. *AJNR. American Journal of Neuroradiol*ogy, 34(10), 1866–1872.
- Mark, C. I., Mazerolle, E. L., & Chen, J. J. (2015). Metabolic and vascular origins of the BOLD effect: Implications for imaging pathology and resting-state brain function. *Journal of Magnetic Resonance Imaging*, 42 (2), 231–246.
- Veldsman, M., Cumming, T., & Brodtmann, A. (2015). Beyond BOLD: Optimizing functional imaging in stroke populations. *Human Brain Mapping*, 36(4), 1620–1636.
- Yablonskiy, D. A., Sukstanskii, A. L., & He, X. (2013). Blood oxygenation level-dependent (BOLD)-based techniques for the quantification of brain hemodynamic and metabolic properties – Theoretical models and experimental approaches. *NMR in Biomedicine*, 26(8), 963–986.

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 the 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 abluminal 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 the 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 the brain endothelium compared with the endothelium from the periphery. These findings suggest a procoagulant environment in the brain that may predispose the brain to strokes.

Cross-References

▶ Neuroglia

Body Dysmorphic Disorder

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Synonyms

Dysmorphia

Definition

Previously known a dysmorphia, body dysmorphic disorder (BDD) is an obsessive-compulsive disorder (OCD)-related condition characterized by a preoccupation with one or more perceived defects or flaws in one's physical appearance that are not observable or appear slight to others. The disorder is manifested through repetitive behaviors such as excessive grooming, skin picking, or mental acts such as comparing one's appearance to others, in efforts to assuage appearance concerns. Muscle dysmorphia is a subtype of BDD, in which there is a preoccupation with one's body build being too small or insufficiently muscular.

Categorization

The disorder is classified with the obsessivecompulsive and related disorders in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychiatric Association 2013).

Current Knowledge

Epidemiology

National prevalence rates of BDD range from 1.7% to 2.4%, with higher rates in outpatient (1–8-6.7%) and inpatient (13.1–16%) clinical samples, as well as in patients seeking cosmetic surgery and dermatologic treatments (7.7–24.5%). Risk factors for BDD include childhood neglect and abuse and a family history of OCD. The mean age of onset is 16–17 years and most individuals develop symptoms by age 18. Nevertheless, BDD does occur among the elderly as well, though much less is known about the nature of the disorder in this segment of the population.

Etiology and Clinical Issues

The etiology of BDD is complex, encompassing biological, psychological, and socioenvironmental factors. Biological factors that have been implicated include hyperactivity in the left orbitofrontal cortex and volume abnormalities in the orbitofrontal cortex and anterior cingulate cortex. Additionally, maladaptive beliefs and cognitive biases about physical appearance, reinforced through life experiences, likely play a role. Compared to obsessive-compulsive disorder, BDD tends to be associated with higher suicidal ideation, greater psychiatric comorbidity, and poorer insight. Associated/comorbid conditions include major depressive disorder, social anxiety disorder, and substance-related disorders.

Assessment and Treatment

With regard to assessment, the primary rule out is eating disorders, however, BDD must also be differentiated from other OCD-related disorders, illness anxiety, depressive and anxiety disorders, and psychotic disorders. It is recommended that patients receiving mental health treatment be screened for BDD, by asking whether individuals are worried or unhappy with their appearance and through use of standardized measures such as the Yale-Brown Obsessive-Compulsive Scale. Effective pharmacologic interventions include serotonin reuptake inhibitors such as escitalopram and fluoxetine. Psychotherapeutic interventions include BDDspecific cognitive-behavioral therapy (CBT). There is emerging evidence for the utility of internet-based CBT.

See Also

Obsessive-Compulsive and Related Disorders

References and Readings

- American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (DSM-5[®]). Washington, DC: American Psychiatric Association.
- Enander, J., Andersson, E., Mataix-Cols, D., Lichtenstein, L., Alström, K., Andersson, G., ... & Rück, C. (2016). Therapist guided internet based cognitive behavioural therapy for body dysmorphic disorder: Single blind randomised controlled trial. *BMJ*, 352, i241.
- Fang, A., Matheny, N. L., & Wilhelm, S. (2014). Body dysmorphic disorder. *Psychiatric Clinics of North America*, 37(3), 287–300.
- Harrison, A., de la Cruz, L. F., Enander, J., Radua, J., & Mataix-Cols, D. (2016). Cognitive-behavioral therapy for body dysmorphic disorder: A systematic review and meta-analysis of randomized controlled trials. *Clinical Psychology Review*, 48, 43–51.
- Phillips, K. A., & Hollander, E. (2008). Treating body dysmorphic disorder with medication: Evidence,

misconceptions, and a suggested approach. *Body Image*, 5(1), 13–27.

- Phillips, K. A., Pinto, A., Menard, W., Eisen, J. L., Mancebo, M., & Rasmussen, S. A. (2007). Obsessive-compulsive disorder versus body dysmorphic disorder: A comparison study of two possibly related disorders. *Depression and Anxiety*, 24(6), 399–409.
- Phillips, K. A., Keshaviah, A., Dougherty, D. D., Stout, R. L., Menard, W., & Wilhelm, S. (2016). Pharmacotherapy relapse prevention in body dysmorphic disorder: A double-blind, placebo-controlled trial. *American Journal of Psychiatry*, 173(9), 887–895.

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 hemiinattention 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: 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.

Borderline Personality Disorder

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Synonyms

Emotional intensity disorder

Definition

Borderline personality disorder (BPD) is characterized in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association 2013) by pervasive instability in relationships, affect, and self-image, present in adulthood and across contexts. Criteria include frantic avoidance of real or imagined abandonment; impulsivity; self-harm, including self-injury and suicidal behavior; marked emotional reactivity with intense, disproportionate anger; persistent feelings of emptiness; and transient paranoid ideation or dissociation.

Categorization

BPD is classified with the cluster B personality disorders in DSM-5.

Current Knowledge

Prevalence

Rates of BPD are estimated at 1-5.9% in the community, with 75% female. Treatment settings have higher rates: about 6% in primary care, 10% in outpatient mental health clinics, and 20% in inpatient psychiatric programs. Increased risk for BPD is found in first-degree relatives, Native Americans, and Blacks (Tomko et al. 2014).

Diagnostic Considerations

Men with BPD may report greater symptom severity, separation anxiety, and body image concerns in childhood and odd thinking in adolescence (Goodman et al. 2013; Busch et al. 2016). Impulsivity diminishes, but affective reactivity persists with aging (Arens et al. 2013). Symptoms vary across cultures, with self-harm/suicidality more common in developed countries (Jani et al. 2016; Paris and Lis 2013).

Clinical Correlates

BPD has been proposed as a mood disorder, noting similar affective variability, impulsivity, and related limbic dysregulation to bipolar disorder (BD; Sjåstad et al. 2012; Perugi et al. 2013). While comorbidity is high, both BD and BPD exist primarily without the other (Zimmerman and Morgan 2013). Mood disorders, eating disorders, PTSD, ADHD, and other personality diagnoses are frequent comorbid conditions. Premature death, suicide, and significant physical injuries are not uncommon.

Physiology and Neuropsychology

In BPD, the hippocampus, anterior cingulate cortex, dorsolateral prefrontal cortex, and amygdala are implicated (Mak and Lam 2013; O'Neill and Frodl 2012; Ruocco et al. 2016). Prefrontal gray matter deficits may increase with age, while parieto-occipital deficits may be more pronounced in younger individuals (Kimmel et al. 2016). This population may also have atypical sensitivity to stress hormones, dysregulation of the oxytocinergic system, and atypical sleep patterns (Herpertz and Bertsch 2015; Winsper et al. 2016). Neurocognitive findings include increased selective attention to negative stimuli, difficulty with dichotomous thinking, emotional processing, and poor visuospatial working memory (Mak and Lam 2013; Winter et al. 2017; Thomsen et al. 2017).

Treatment

Dialectical behavior therapy (DBT) has been the most frequently studied model of psychotherapy, with mentalization-based therapy (MBT), schema-focused therapy (SFT), and Systems Training for Emotional Predictability and Problem-Solving (STEPPS) also frequently used. No treatment can boast a broad and sound evidence base, and replication studies are desperately needed (Stoffers et al. 2012).

No medication is currently approved for BPD, although common pharmacologic adjunctives may include mood stabilizers, antipsychotics, and antidepressants. When alcohol use disorder co-occurs, anticonvulsants and second-generation antipsychotics may be prescribed. Oxytocin yields some promise, with some studies showing reduced emotional reactivity, while others have found increased interpersonal anxiety and uncooperative behaviors (Amad et al. 2015).

See Also

Personality Disorders

References and Readings

- Amad, A., Thomas, P., & Perez-Rodriguez, M. M. (2015). Borderline personality disorder and oxytocin: Review of clinical trials and future directions. *Current Pharmaceutical Design*, 21(23), 3311–3316. doi:CPD-EPUB-68145 [pii].
- American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). Arlington: American Psychiatric Association Publishing.
- Arens, E. A., Stopsack, M., Spitzer, C., Appel, K., Dudeck, M., Volzke, H., ... Barnow, S. (2013). Borderline personality disorder in four different age groups: A cross-sectional study of community residents in Germany. *Journal of Personality Disorders*, 27(2), 196–207. 10.1521/pedi.2013.27.2.196
- Busch, A. J., Balsis, S., Morey, L. C., & Oltmanns, T. F. (2016). Gender differences in borderline personality disorder features in an epidemiological sample of adults age 55–64: Self versus informant report. *Journal* of Personality Disorders, 30(3), 419–432. https://doi. org/10.1521/pedi_2015_29_202.
- Goodman, M., Patel, U., Oakes, A., Matho, A., & Triebwasser, J. (2013). Developmental trajectories to male borderline personality disorder. *Journal of Personality Disorders*, 27(6), 764–782. https://doi.org/ 10.1521/pedi 2013 27 111.
- Herpertz, S. C., & Bertsch, K. (2015). A new perspective on the pathophysiology of borderline personality disorder: A model of the role of oxytocin. *The American Journal of Psychiatry*, 172(9), 840–851. https://doi.org/ 10.1176/appi.ajp.2015.15020216.

- Jani, S., Johnson, R. S., Banu, S., & Shah, A. (2016). Cross-cultural bias in the diagnosis of borderline personality disorder. *Bulletin of the Menninger Clinic, 80* (2), 146–165. https://doi.org/10.1521/ bumc.2016.80.2.146.
- Kimmel, C. L., Alhassoon, O. M., Wollman, S. C., Stern, M. J., Perez-Figueroa, A., Hall, M. G., ... Radua, J. (2016). Age-related parieto-occipital and other gray matter changes in borderline personality disorder: A meta-analysis of cortical and subcortical structures. *Psychiatry Research*, 251, 15–25. https://doi.org/ 10.1016/j.pscychresns.2016.04.005
- Mak, A. D., & Lam, L. C. (2013). Neurocognitive profiles of people with borderline personality disorder. *Current Opinion in Psychiatry*, 26(1), 90–96. https://doi.org/ 10.1097/YCO.0b013e32835b57a9.
- O'Neill, A., & Frodl, T. (2012). Brain structure and function in borderline personality disorder. *Brain Structure* & Function, 217(4), 767–782. https://doi.org/10.1007/ s00429-012-0379-4.
- Paris, J., & Lis, E. (2013). Can sociocultural and historical mechanisms influence the development of borderline personality disorder?. *Transcultural Psychiatry*, 50(1), 140–151.
- Perugi, G., Angst, J., Azorin, J. M., Bowden, C., Vieta, E., Young, A. H., & BRIDGE Study Group. (2013). Is comorbid borderline personality disorder in patients with major depressive episode and bipolarity a developmental subtype? Findings from the international BRIDGE study. *Journal of Affective Disorders*, 144 (1–2), 72–78. https://doi.org/10.1016/j.jad.2012.06.008.
- Ruocco, A. C., Rodrigo, A. H., McMain, S. F., Page-Gould, E., Ayaz, H., & Links, P. S. (2016). Predicting treatment outcomes from prefrontal cortex activation for self-harming patients with borderline personality disorder: A preliminary study. *Frontiers in Human Neuroscience*, 10, 220. https://doi.org/10.3389/ fnhum.2016.00220.
- Sjåstad, H. N., Grawe, R. W., & Egeland, J. (2012). Affective disorders among patients with borderline personality disorder. *PloS One*, 7(12), e50930. https://doi.org/ 10.1371/journal.pone.0050930 [doi].
- Stoffers, J. M., Vollm, B. A., Rucker, G., Timmer, A., Huband, N., & Lieb, K. (2012). Psychological therapies for people with borderline personality disorder. *The Cochrane Database of Systematic Reviews*, 8, CD005652. https://doi.org/10.1002/14651858. CD005652.pub2.
- Thomsen, M. S., Ruocco, A. C., Uliaszek, A. A., Mathiesen, B. B., & Simonsen, E. (2017). Changes in neurocognitive functioning after 6 months of mentalization-based treatment for borderline personality disorder. *Journal of Personality Disorders*, 31(3), 306–324.
- Tomko, R. L., Trull, T. J., Wood, P. K., & Sher, K. J. (2014). Characteristics of borderline personality disorder in a community sample: Comorbidity, treatment utilization, and general functioning. *Journal of Personality Disorders*, 28(5), 734–750. https://doi.org/ 10.1521/pedi_2012_26_093.

- Winsper, C., Tang, N. K., Marwaha, S., Lereya, S. T., Gibbs, M., Thompson, A., & Singh, S. P. (2016). The sleep phenotype of borderline personality disorder: A systematic review and meta-analysis. *Neuroscience* and Biobehavioral Reviews, 73, 48–67.
- Winter, D., Niedtfeld, I., Schmitt, R., Bohus, M., Schmahl, C., & Herpertz, S. C. (2017). Neural correlates of distraction in borderline personality disorder before and after dialectical behavior therapy. *European Archives of Psychiatry and Clinical Neuroscience*, 267(1), 51–62. https://doi.org/10.1007/s00406-016-0689-2.
- Zimmerman, M., & Morgan, T. A. (2013). The relationship between borderline personality disorder and bipolar disorder. *Dialogues in Clinical Neuroscience*, 15(2), 155–169.

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. The complete BDAE-3 test kit includes stimulus cards, test booklets for Standard and Short forms, the 60-item Boston Naming Test with record booklets, a DVD, and a hardbound text that contains the test manual.

The Boston Diagnostic Aphasia Examination-3 (BDAE-3; Goodglass et al. 2001a) 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. The test manual is part of the text by Goodglass et al. (2001b). It provides suggestions for administering, scoring, and interpreting performance on subtests, 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. 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, Greek, Hindi, Finnish, Mandarin Chinese, Japanese, and Portuguese.

Psychometric Data

Norms

Standardization of the BDAE-3 is based on a population of individuals with aphasia (IwA) who were referred concurrently by field examiners working in inpatient, outpatient, and private practice settings. Means and standard deviations

for the BDAE-3 subtests for IwA are provided in the test manual. The number of IwA administered the 50 subtests varies from a maximum of 85 to a low of 31. Means are also provided for 15 nonclinical individuals who, on average, failed less than one item per subtest. Rosselli et al. (1990) and Pineda et al. (2000) provide norms for the Spanish version of the BDAE-2 (Goodglass and Kaplan 1986) that is based on 156 healthy individuals living in Columbia, South America.

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. No stability coefficients for test-retest are provided. The authors state that test-retest reliability is difficult to attain with IwA. The current reliability coefficients demonstrate very good internal consistency in terms of what the items within the subtests are measuring (Goodglass et al. 2001b). For most subtests, correlations are very high between the short and standard forms (>0.90;Goodglass et al. 2001b). No reliability information is provided in the BDAE-3 manual regarding the Severity Rating Scale, Language Competency Index, praxis assessment, or Spatial-Quantitative Battery.

Validity

A correlation matrix was obtained for all the scores in the BDAE-3 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 subtests for the standardization sample. Based on these, "a number of sharply defined clusters" are indicated by the authors (p. 16). Strauss et al. (2006), however, pointed out that the lack of data on the entire correlational matrix makes it "difficult to estimate convergent and discriminant validity within and across BDAE-3 clusters" (p. 896) especially given the fact that the more than 50 subtests were administered to just 31–85 subjects. 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 adults with aphasia, 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) for the BDAE-2 Spanish version.

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 and Robinson 1989). Brookshire and Nicholas (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 and Graves 1975).

Ecological validity of the BDAE for predicting progress with aphasia therapy has been described by various authors (e.g., Davidoff and Katz 1985; Marshall and Neuburger 1994).

Clinical Uses

The BDAE is derived from samples of 85 adult individuals with stroke and 15 elderly nonclinical volunteers. Therefore, it is most useful when assessing adult populations with language impairments resulting from strokes, but it may be used effectively with persons who have sustained traumatic brain injury (e.g., Theodoros et al. 2008) and forms of dementia (e.g., Tsantali et al. 2013). 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. 2001b).

BDAE results can be used to guide aphasia treatment programs (Helm-Estabrooks et al. 2014) and to measure the effects of treatment (Robey 1998).

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-braindamaged 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: Lea & Febiger.
- Goodglass, H., & Kaplan, E. (1986). La evaluacion de la afasia y transformos relacionados (2nd ed.). Madrid: Editorial Medica Panamericana.
- Goodglass, H., Kaplan, E., & Barresi, B. (2001a). Boston diagnostic aphasia examination (3rd ed.). Austin: Pro-Ed.
- Goodglass, H., Kaplan, E., & Barresi, B. (2001b). The assessment of aphasia and related disorders (3rd ed.). Austin: Pro-Ed.
- Helm-Estabrooks, N., Albert, M. L., & Nicholas, M. (2014). *Manual of aphasia and aphasia therapy* (3rd ed.). Austin: Pro-ED.
- Marshall, R. C., & Neuburger, S. I. (1994). Verbal selfcorrection 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.
- Robey, R. R. (1998). A meta-analysis of clinical outcomes in the treatment to aphasia. *Journal of Speech, Lan*guage, and Hearing Research, 41(1), 172–188.
- Rosselli, M., Ardila, A., Florez, A., & Castro, C. (1990). Normative data on the Boston diagnostic aphasia evaluation in a Spanish speaking population. *Journal of Clinical and Experimental Neuropsychology*, 12, 313–322.
- Spreen, O., & Risser, A. H. (Eds.). (2003). Assessment of aphasia. New York: Oxford University Press.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). A compendium of neuropsychological tests (3rd ed.). New York: Oxford University Press.
- Sussman, H., Marquardt, T., Hutchinson, J., & MacNeilage, P. (1986). Compensatoryarticulation in Broca's aphasia. *Brain and Language*, 27, 56–74.
- Theodoros, D., Hill, A., Russell, T., Ward, E., & Wootton, R. (2008). Assessing acquired language disorders in adults via the internet. *Telemedicine Journal and E-Health*, 14(6), 552–559.
- Tsantali, E., Economidis, D., & Tsolaki, M. (2013). Could language deficits really differentiate mild cognitive impairment (MCI) from mild Alzheimer's disease? *Archives of Gerontology & Geriatrics*, 57(3), 263–270.

Boston Naming Test

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Synonyms

BNT

Description

Boston Naming Test (2nd (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-3 (Goodglass et al. 2001) from Pro-Ed.

The Boston Naming Test (BNT) is a widely used tool for assessing confrontation naming ability. The BNT consists of 60 black and white line drawings of objects that are ordered according to vocabulary word frequency from bed to abacus. The order of the pictured stimuli takes into account the finding that individuals 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.. This type of picture-naming vocabulary test is useful in the evaluation of children with learning disabilities and 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, adults with aphasia, and nonclinical adults. The drawings are shown to the examinee one at a time, and the examinee is asked to name each of them. Item 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, e.g., "used by a carpenter" for saw) and a "phonemic cue" (the beginning sound of the target word, e.g., "s ... " for saw) A stimulus cue is presented when the examinee clearly misperceives the picture (e.g., "worm" for pretzel) or indicates a lack of recognition of the picture. A phonemic cue is presented after each error response, including following a stimulus cue. The examinee is given up to 20 s to respond following each stimulus presentation and after the cues. All responses are recorded as a "correct response" or as an error with the actual error response written verbatim for later coding by type. Types of cues ("stimulus cue" or "phonemic cue") presented are noted. Response latencies in seconds are also documented. The total correct score is the sum of the accurate spontaneous responses given within 20 s of picture presentation or 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 Mack et al. 15- item version has been adopted by the Consortium to Establish a Registry for Alzheimer's Disease (CERAD).

In a 2011 study, Hobson, et al. explored whether the 15-item BNT/CERAD version and two 30-item (even and odd) versions could predict scores earned on the 60-item version by participants with and without AD. Estimated 60-item scores created from the shorter versions were then correlated with actual scores. The 60-item scores estimated from the 30-item versions had good predictive value for actual 60-item BNT scores and the 15-item version less so.

The new BNT-2 also includes a multiplechoice version that can be administered following the standard presentation, specifically to further assess the examinee's recognition of the lexicon for items previously missed. The BNT-2 is available separately and as part of the revised BDAE-3. 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, testretest reliability ranged between 0.62 and 0.89 (Mitrushina and Satz 1995); and high retest reliability (0.92) in a normal or neurologically stable adult population (Dikmen et al. 1999). In 2012, Sachs and colleagues published a BNT reliability study of 844 cognitively unimpaired, Caucasian adults who were over age 55. The BNT was readministered between 9 and 24 months after the baseline exam. During a 9–15-month retest period, a 4-point decline occurred. A 6-point decline occurred during a 16–24-month retest period. The participant's age and family history of dementia further characterized the cutoff values for reliable changes in BNT performance.

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.

Data on BNT norms for children is limited. The BNT record form presents norms for ages 5 years and 0 months (5-0) through 12-5, based on small groups in successive 6-month age increments. 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.

Martielli and Blackburn (2015) collected normative BNT-2 data for 100 male and 100 female adolescents aged 15–18 years. None of the 200 participants had neurologic, psychiatric, or academic problems. No statistically significant differences in BNT scores based on gender, age, or grade occurred. Martielli and Blackburn provide normative means and standard deviations, collapsed across age and gender.

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 and Adams 1999; Steinberg et al. 2005; Tombaugh and 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 and 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).

It can be found in the literature a number of normative reports for adult English speakers (see pp. 905-907, Strauss et al. 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 aboveaverage intelligence who were administered the 60-item version. Their data was presented in 5-year increments, ranging from ages 25–84 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 ten and a standard deviation (SD) of three (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), and 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).

Pedraza and colleagues (2009) used item response theory (IRT) and methods to detect differential item functioning (DIF) of BNT items with 336 Caucasian and 334 African American participants. Twelve items were shown to have DIF between the two groups. Additional analyses showed that six of these items (*dominoes*, *escalator*, *muzzle*, *latch*, *tripod*, and *palette*) represent the strongest evidence for race-/ethnicity-based DIF. This study demonstrates that psychometric and sociocultural factors can lead to BNT score discrepancies between groups.

Zec and colleagues (2007a) published the results of a BNT study conducted with 1111 "normal elderly" adults aged 50–101 years and 61 younger adults aged 20–49 years. They found both significantly lower scores and increasing variability among increasing age groups and with lower educational levels. In a subsequent study (Zec et al. 2007b), BNT raw scores earned by 1,026 participants ranging in age from 50–95 were converted to scaled scores and percentiles.

Zec and colleagues present these norms and recommend them for use in assessing people with suspected dementia.

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, individuals with aphasia a, and typical adults, although there is limited and poorly described normative data and no test-retest reliability for children.

In their 2013 chapter on using a process approach to aphasia, Helm-Estabrooks and Nicholas describe the clinical and diagnostic utility of response patterns to the BNT. Responses typical of a person with Broca's aphasia and a person with Wernicke's aphasia are used to illustrate the clinical value of looking beyond BNT scores to analyzing transcribed responses.

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. (2001). Boston diagnostic aphasia examination (3rd ed.). Austin: Pro-Ed.
- 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: PAR.
- Helm-Estabrooks, N., & Nicholas, M. (2013). The process approach to aphasia. In L. Ashendorf & D. J. Libon (Eds.), *Neuropsychological assessment using the Boston process approach: A practitioner's guide* (pp. 170–199). New York: Oxford Press.
- Hobson, V. L., Hall, J. R., Harvey, M., Munro Cullum, C., Lacritz, L., Massman, P. J., Waring, S. C., & O'Bryant, S. E. (2011). An examination of the Boston Naming Test: Calculation of "estimated" 60-item score from 30and 15-item scores in a cognitively impaired population. *International Journal of Geriatric Psychiatry*, 26, 351–335.
- 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.

- 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.
- Kaplan, E., Goodglass, H., & Weintrab, S. (1978, 1983). The Boston naming test: Experimental edition (1978). Boston: Kapan & Goodglass. (2nd ed.) Philadelphia: Lea & Febiger.
- Kaplan, E., Goodglass, H., & Weintrab, S. (2001). The Boston naming test (2nd ed.). Austin: Pro-Ed.
- 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., & 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.
- Martielli, T. M., & Blackburn, L. B. (2015). When a funnel becomes a martini glass: Adolescent performance on the Boston Naming Test. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 1–13.
- 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.
- 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, 641–652.
- 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-braindamaged adults. *Clinical Aphasiology*, 18, 103–115.

- Pedraza, O., Graff-Radford, N. R., Smith, G. E., Ivnik, R. J., Willis, F. B., Petersen, R. C., & Lucas, J. A. (2009). Differential item functioning of the Boston Naming Test in cognitively normal African American and Caucasian older adults. *Journal of the International Neuropsychological Society*, 15, 758–768.
- Randolph, C., Lansing, A., Ivnick, R. J., Cullum, C. M., & Hermann, B. P. (1999). Determinants of confrontation naming performance. *Archives of Clinical Neuropsycology*, 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.
- Sachs, B. C., Lucas, J. A., Smith, G. E., Ivnik, R. J., Peterson, R. C., Graff-Radford, N. R., & Pedrazal, O. (2012). Reliable change on the Boston Naming Test. *Journal of International Neuropsychological Society*, 18, 375–378.
- 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 shortforms. *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 Dutchspeaking 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 neuropsychological 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. *Neuropsychologia*, 27, 1073–1079.
- Zec, R. F., Burkett, N. R., Markwell, S. J., & Larsen, D. L. (2007a). A Cross-sectional study of the effects of age, education, and gender on the Boston Naming Test. *The Clinical Neuropsychologist*, 21(4), 587–616.
- Zec, R. F., Burkett, N. R., Markwell, S. J., & Larsen, D. L. (2007b). Normative data stratified for age, education, and gender on the Boston Naming Test. *The Clinical Neuropsychologist*, 21(4), 617–637.

Boston Process Approach

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Synonyms

BPA

Definition

Born out of the work of A. R. Luria (e.g., Higher Cortical Function in Man, 1966), the Boston process approach (BPA) to neuropsychological testing 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 (Kaplan 1988; Strauss et al. 2006).

Current Knowledge

According to Edith Kaplan (1988, 1990), the "achievement"-oriented approach to assessment, where performance is based on the scores obtained on a particular test, 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 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, as each of these variables can influence a patient's performance. According to Kaplan (1990), 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. In addition, the test may be administered differently from the standardized approach, and additional measure may be introduced in order to better understand the component processes that influence or are involved in 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 et al. 1986).

While right or wrong answers are deemphasized in the BPA, Kaplan (1990) felt that it was essential for the qualitative observations to be quantifiable and subjected to statistical analyses. This led to the development of a wide variety of measures including the NEPSY – Second Edition (Korkman et al. 2007) and Delis-Kaplan Executive Function System (Delis et al. 2001). 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, enabling complex processes to be broken down into simpler components, so that the area(s) of weakness and strength can be more readily identified.

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. This can result in practice effects but can also change how the patient approaches the test when it is readministered.

See Also

- Fixed Battery
- ► Flexible Battery
- Hypothesis Testing Approach to Evaluation

Further Readings

- Delis, D. C., Kaplan, E., & Kramer, J. H. (2001). Delis-Kaplan executive function system. San Antonio: 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.
- Korkman, M., Kirk, U., & Kemp, S. L. (2007). NEPSY II. Administrative manual. San Antonio: Psychological Corporation.
- 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.

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, and 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.

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 (Kumaret al. 2014). 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. 2014).

Symptoms

Clinically, cerebral abscesses can be devastating and often lead to an increase in intracranial pressure and localized deficits (Kumar et al. 2014). 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%. Earlier treatment predicts a better outcome, although long-term neurological deficits may persist despite all intervention approaches (Kumar et al. 2014 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. (2014). Robbins and Cotran pathologic basis of disease (9th ed.). Philadelphia: W.B. Saunders Company.

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 and 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.

Brain	Death,	Table	1	Assessment	of	neurologic
devasta	ation					

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 $> 32 \degree C$

1. Coma, profound state of unconsciousness	ciousne	unconscio	ate o	profound	Coma,	1.
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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 Paco₂ 60 mmHg or 20 mmHg above patients baseline^a

^aPaco₂ is the partial pressure of arterial carbon dioxide (Reprinted from Wijdicks (2001) with permission)

These often include cerebral angiography, transcranial doppler, electroencephalography (EEG), or 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 Injury Association of America

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Synonyms

BIAA

Membership as of 2016

The Brain Injury Association of America (BIAA) consists of more than 27 divisions and state affiliates across the USA, 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 25,000 individuals. Approximately two-thirds of the list members are traumatic brain injury (TBI) survivors and their family members, while the remaining represents a wide variety of professional providers and researchers (Ayotte, personal communication, February, 2016).

Major Areas or Mission Statement

"Our mission is to advance brain injury prevention, research, treatment and education and to improve the quality of life for all individuals impacted by brain injury. Through advocacy, we bring help, hope and healing to millions of individuals living with brain injury, their families and the professionals who serve them" (www. biausa.org).

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 reauthorized as Title XIII of the Children's Health Act of 2000 (PL 106-310), the S. 793 TBI Act of 2008, and most recently the TBI Reauthorization Act of 2014 (S. 2539). 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 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 the congress to join the Congressional Brain Injury Task Force. The latest version of the bill provided new emphasis on brain injury management in children by tasking the CDC to study TBI care in children and identify potential opportunities for new research. The BIAA, in cooperation with the Mount Sinai Brain Injury Research Center, published a 2013 position paper on this topic (Gordon et al. 2013).

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 serve veterans and active-duty military TBI victims via clinical care, research initiatives, and ongoing education of victims, families, providers, and policy makers. The program has nearly tripled in size from its 6 initial locations in 1992 to 16 that now offer specialized care; it acts as the operational component of the Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury.

In 1996, the BIAA founded the Academy of Certified Brain Injury Specialists (ACBIS) which to date has certified over 6500 members. The mission of ACBIS 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*, which is now published in its fifth edition (Academy for the Certification of Brain Injury Specialists 2016). Certification is granted to those with appropriate work experience who have successfully completed the training and written examination.

In 1999, the US Supreme Court decided the case of L.C. & E.W. vs. Olmstead. The court held 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 appropriate. 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. Although the BIAA is no longer doing trainings specifically for this purpose, their advocacy and legislative efforts continue to be driven toward increasing access to medical care, including rehabilitation, for all brain injury survivors. In 2006, these efforts included the publication Cognitive Rehabilitation: The Evidence, Funding, and Case for Advocacy of Brain Injury (Katz et al. 2006), which included ten recommendations to increase access and delivery of cognitive rehabilitation services across the nation.

In 2000, 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. The BIAA's newest effort involves partnering with the Icahn School of Medicine at Mount Sinai to provide recommendations for post-acute TBI care; this will be the first guideline focused upon treatment efforts after the inpatient stage. These guidelines should be published by 2017 and will address outpatient rehabilitation efforts as well as chronic disease

management for those suffering from moderate to severe TBI.

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 on brain injury. 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 to facilitate clinical care for military service-related brain injury, has been a crucial area of intervention. Public policy initiatives have also sought to address trauma care, child abuse prevention, transportation safety, brain injury education, and respite care (Ayotte, personal communication, February, 2016).

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. In recent years it has focused on Internet-friendly methods for disseminating information and connecting survivors with educational materials and possible providers. The BIAA also publishes TBI Challenge!, a quarterly newsletter with a distribution that includes 25,000 households. It continues to host/cohost educational meetings and conferences and has added both live and recorded webinars to its array of education options. This webinar series, free to survivors, included topics geared toward survivors and caregivers, as well as professional providers and researchers. BIAA continues to maintain a comprehensive website and makes electronically available the National Directory of Brain Injury Rehabilitation Services. On an annual basis, the BIAA responds to over 100,000 requests for assistance through either its national information call center or its website.

See Also

Traumatic Brain Injury (TBI)

References and Readings

- Aarabi, B., Alden, T. D., Chestnut, R. M., Downs J. H., Ecklund, J. M., Eisenberg, H. M., ... Walters, B. C. (2001). Management and prognosis of penetrating brain injury – Guidelines. *Journal of Trauma, Injury, Infection and Critical Care*, 51, S1–S86.
- Academy of certified brain injury specialists. (2016). *The essential brain injury guide* (5th ed.). McLean: Brain Injury Association of America.
- Brain Injury Association of America. (2005). Academy of certified brain injury specialists (ACBIS). Retrieved from http://www.aacbis.net/index.html.
- Brain Injury Association of America. (n.d.). *Brain injury* association USA home page. Retrieved from http:// www.biausa.org.
- 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 from http:// www.dvbic.dcoe.mil.
- Gordon, W. A., Oswald, J. M., Vaughn, S. L., Connors, S. H., & Brown, M. (2013). States of the states: Meeting the educational needs of children with traumatic brain injury. Retrieved from www.biausa.org/biaa-positionpapers.htm.
- 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: 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: McGraw-Hill.
- Kolb, B. (1995). *Brain plasticity and behavior*. Mahwah: Erlbaum.

- Kolb, B., Gibb, R., & Robinson, T. E. (2003). Brain plasticity and behavior. *Current Directions in Psychologi*cal 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: Massachusetts Institute of Technology.

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.

Postmortem examination of elderly individuals provides evidence that there is a discrepancy between the clinical manifestation of Alzheimer's disease and the neuropathology of the disorder (Katzman et al. 1988). 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 (Stern et al. 2006).

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. Symptomatic behaviors are less likely to be prevalent in individuals with greater brain reserve capacity. Research has 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. 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. It has also been argued that innate intelligence of life experiences, including educational and professional achievements, may increase cognitive reserve by helping a set of behavoiral skills that allow people to manage their behaviors better (Vasile 2013). The interplay among cognitive activity, physical activity, diet, and brain reserve capacity is being carefully studied in an attempt to understand the complex 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 and other types of dementia. 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

- 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). In L. Bieliauskas (Ed.), Cognitive reserve, theory and application. New York: Psychology Press.
- Vasile, C. (2013). Cognitive reserve and cortical plasticity. Procedia – Social and Behavioral Sciences, 78, 601–604.

Brain Swelling

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Definition

Expansion of the size of the brain that occurs following head trauma and brain injury.

Current Knowledge

Brain swelling can elevate intracranial pressure immediately following brain injury and can continue hours or days after the onset of brain injury. 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. Mitochondrial function is now thought to be directly related to brain edema. Acute management of brain swelling is important at the gross and molecular level. 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, administering medications to reduce potential oxidative stress, and using an electronic intracranial pressure monitor with a valve to adjust pressure over time.

- ▶ 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.
- Vlodavsky, E., Palzur, E., Shehadeh, M., & Soustiel, J. F. (2015). Post-traumatic cytoxic edema is directly related to mitochondrial function. *Journal* of Cerebral Blood Flow and Metabolism, 37 (1):166–177.

Brain Training

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Synonyms

Brain games

Brain training is a term used primarily by companies which market cognitive intervention products or by the lay public, rather than clinicians or researchers. The term is somewhat of a misnomer as only a very small fraction of published studies have assessed neural activity directly. Companies use this term to refer to "... practicing core cognitive abilities with the goal of improving performance on other cognitive tasks, including those involved in everyday activities..." (Simons et al. 2016, pp. 105). Support for the term and particularly its effectiveness is highly controversial, and scientific effectiveness should be differentiated from marketing and other claims. Care should be used in that the term brain training is not synonymous with concepts like cognitive training or cognitive rehabilitation.

Cross-References

Cognitive Rehabilitation

References and Readings

Simons, D. J., Boot, W. R., Charness, N., Gathercole, S. E., Chabris, C. F., Kambrick, D. Z., & Stine-Morrow, A. L. (2016). Do "brain-training" programs work? *Psychological Science in the Public Interest*, 17(3), 103–186.

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 et al. 2001; Price et al. 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: 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.

Brainstem Auditory Evoked Responses

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Synonyms

Auditory brainstem responses (ABR); Auditory brainstem response audiometry; Auditory evoked response (AER); BAER; Brainstem auditory evoked potentials (BAEP); 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 et al. 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 morphology and interwave differences are compared to healthy controls to establish 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 infant hearing screening, 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 et al. 1996).

Cross-References

Evoked Potentials

References and Readings

- Huszar, L. (2006). Clinical utility of evoked potentials. *eMedicine*. Retrieved 9 July 2007 from http://www. emedicine.com/neuro/topic69.htm. Accessed 23 Mar 2017.
- 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.
- Lew, H. L., Tanaka, C., Hirohata, E., & Goodrich, G. L. (2016). Auditory, vestibular and visual impairments. In D. X. Cifu (Ed.), *Physical medicine and rehabilitation* (pp. 1137–1161). Philadelphia: Elsevier.
- Luauté, J., Fischer, C., Adeleine, P., Morlet, D., Tell, L., & Boisson, D. (2005). Late auditory and event-related potentials can be useful to predict good functional outcome after coma. *Archives of Physical Medicine* and Rehabilitation, 86, 917–923.
- Luaute, J., Maucort-Boulch, D., Tell, L., Quelard, F., Sarraf, T., Iwaz, J., Boisson, D., & Fischer, C. (2010). Long-term outcomes of chronic minimally conscious and vegetative states. *Neurology*, *75*, 246–252.
- Vernet, M., Bashir, S., Enam, S. F., Kumru, H., & Pascual-Leone, A. (2012). Electrophysiologic techniques. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine* (pp. 236–238). 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

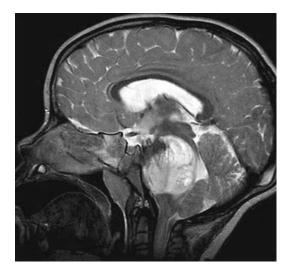
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Synonyms

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). This type of tumors often originates from the left side and typically involves one of three anatomical locations within the brainstem. Pontine brainstem gliomas are associated with the poorest prognosis



Brainstem Glioma, Fig. 1 (Picture credit: Michael Phillips and Peter C. Fisher)

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.

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 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.

Brexipiprazole

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Generic Name

Brexpiprazole

Brand Name

Rexulti

Class

Atypical Neuroleptic, Serotonin-Dopamine Modulators (SDAM)

Proposed Mechanism(s) of Action

The efficacy of brexpiprazole may be mediated through a combination of partial agonist activity as serotonin 5-HT1A and dopamine D2 receptors, and antagonist activity at serotonin 5-HT2A receptors.

Indication

Schizophrenia, adjunctive treatment of major depressive disorder

Off Label Use

No common offlabel use

Side Effects

Serious

Increased risk of death in elderly people with dementia-related psychosis, CVA among elderly patients, hyperglycemia, low white blood cell count, orthostatic hypotension, seizures.

Common

Headache, weight gain, somnolence, dyspepsia, constipation, fatigue, dizziness, anxiety, restlessness, increased appetite.

References and Readings

Physicians' desk reference (71st ed.) (2017). Montvale: Thomson PDR.

Additional Information

CenterWatch: https://www.centerwatch.com/druginformation/fda-approved-drugs/drug/100084/rexultibrexpiprazole.

http://www.drugs.com/drug_interactions.html.

Drug Molecule Images: http://www.worldofmolecules. com/drugs/.

Free Drug Online and PDA Software: www.epocrates.com. Free Drug Online and PDA Software.

Gene-Based Estimate of Drug interactions: http://mhc. daytondcs.com: 8080/cgi bin/ddiD4?ver=4&task= getDrugList.

Medscape Psychiatry.

Pill Identification.: http://www.drugs.com/pill_identification.html.

https://rexulti.com/us/mdd.

Brief Cognitive Rating Scale

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Synonyms

BCRS

Description

The Brief Cognitive Rating Scale (BCRS; Reisberg and 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 et al. 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 is comprised of two parts and provides objective ratings for a number of domains which include cognitive functions, functional abilities, mood, and behavior. 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 high test-retest reliability (Foster et al. 1988; Reisberg et al. 1989). Validity studies indicate that the BCRS has strong correlations with the GDS, the Mini-Mental State Examination, some neuropsychological measures of memory abilities, and 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 et al. 1992). The BCRS has been used with elderly persons in India and compared to the Hindi Cognitive Screening Test (HCST) during its development, which correlates with the BCRS total score (r = -0.87) and composite axes (Tripathi and Tiwari 2013). 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
- Modified 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: 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.
- Tripathi, R. K., & Tiwari, S. C. (2013). Cognitive functioning of community dwelling urban older adults with reference to socio-demographic variables. *Indian Journal of Clinical Psychology*, 40(2), 92–102.

Brief Psychiatric Rating Scale

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Synonyms

BPRS

Definition

The Brief Psychiatric Rating Scale (BPRS) (Overall and 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 rate, 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 severe psychopathology.

Current Knowledge

The BPRS continues to be a very commonly used instrument. Between 2013 and 2015, there were well over 300 articles cited in MedLine which employed the BPRS in clinical and experimental studies. Expanded versions of the BPRS have been developed. These include behavioral anchors and structured interview questions (Woemer et al. 1988). The BPRS has a wellestablished history of acceptable psychometric properties. For instance, using a behaviorally anchored 24-item version 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. Furthermore, scores on the BPRS are highly correlated with those of other similar instrument constructs although, as with all such clinician-rated scales, relationships with external criteria are modest (Mortimer 2007).

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 to course of the clinical condition over time. It is most commonly used with psychotic disorders such as schizophrenia (e.g., Samara et al. 2015; Dunayevich et al. 2006), but is appropriate with other psychiatric conditions, including bipolar disorder (Picardi et al. 2008) and major depression (Zanello et al. 2013). It can also be useful with persons having neurological conditions with psychotic or other psychiatric symptoms including Alzheimer's disease, dementia with Lewy bodies, Parkinson's disease (Cummings et al. 2007; Devanand 1998; Tariot et al. 2004), and traumatic brain injury (Diaz et al. 2012).

Versions of the BPRS are widely available and may be found online at http://uwaims.org/files/mea sures/BPRS.pdf (18-item version) and http://www. public-health.uiowa.edu/icmha/outreach/documents/ BPRS_expanded.PDF (24-item expanded version).

See Also

- ► Affective Disorder
- Alzheimer's Disease
- Anxiety
- Clinical Interview
- Dementia with Lewy Bodies
- Parkinson's Disease
- Psychosis
- Psychotic Disorder
- Structured Clinical Interview for DSM-IV (SCID-I/SCID-II)

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-comparison of a trial of haloperidol for psychosis and disruptive behaviours in Alzheimer's disease. *American Journal of Psychiatry*, 155, 1512–1520.
- Diaz, A. P., Schwarzbold, M. L., Thais, M. E., Hohl, A., Bertotti, M. M., Schmoeller, R., Nunes, J. C., Prediger, R., Linhares, M. N., Guarnieri, R., & Walz, R. (2012). Psychiatric Disorders and Health-Related Quality of Life after Severe Traumatic Brain Injury: A Prospective Study. *Journal of Neurotrauma*, 29(6):1029–1037
- 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., Gummattira, P., Kopecky, C. R., & Wassef, A. (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.
- Mortimer, A. M. (2007). Symptom rating scales and outcome in schizophrenia. *The British Journal of Psychiatry Supplement*, 50, s7–14.
- 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., & Biondi, M. (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.
- Samara, M. T., Leucht, C., Leeflang, M. M., et al. (2015). Early improvement as a predictor of later response to antipsychotics in schizophrenia: A diagnostic test review. *American Journal of Psychiatry*, 172, 617–629.
- Tariot, P. N., Profenno, L. A., & Ismail, M. S. (2004). Journal of Clinical Psychiatry, 65(Suppl 11), 11–15.
- Woerner, M. G., Mannuzza, S., & Kane, J. M. (1988). Anchoring the BPRS. *Psychopharmacology Bulletin*, 24, 112–117.
- Zanello, A., Berthoud, L., Ventura, J., & Merlo, M. C. (2013). The Brief Psychiatric Rating Scale (version 4.0) factorial structure and its sensitivity in the treatment of outpatients with unipolar depression. *Pssychiatry Research*, 15, 626–633.

Brief Symptom Inventory

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Synonyms

BSI; BSI-18

Description

The BSI (Derogatis and Melisaratos 1983) is a shortened, 53-item version of the Symptom

Checklist-90 (SCL-90; Derogatis et al. 1973) that measures emotional-behavioral functioning in nine dimensions: somatization, obsessivecompulsive, 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 short form of the BSI intended as a screen for psychiatric disorders and psychological distress, consists of three 6-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 and 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 et al. 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 et al. 1974). The BSI was published in 1983 (Derogatis and 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 et al. 1984), college students (Cochran and Hale 1985), individuals with spinal cord injury (Heinrich et al. 1994), British psychiatric outpatients (Ryan 2007) and community-dwelling adults (Francis et al. 1990), Israeli adolescents (Canetti et al. 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 0.68–0.91 (BSI) to 0.68–0.84 (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 and 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 et al. 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 and Lewinsohn 1981), drug-using adults (Buckner and Mandell 1990), and the elderly (Hale et al. 1984). Screening studies completed with medical cohorts (e.g., Kuhn et al. 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 et al. 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 and Boss 1991). Several cross-cultural studies of the BSI-18 (e.g., Asner-Self et al. 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, and psychotic or have motivation to distort their responses are not good candidates for either measure. Given factor structure concerns noted above (e.g., Boulet and 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 (TBI; Meachen et al. 2008). The Federal Interagency TBI Outcomes Workgroup recommends the BSI-18 as a core common data element for use in TBI research as an indicator of psychological status and response to treatment (Hicks et al. 2013; Wilde et al. 2010). 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 obsessivecompulsive 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 Epidemiological Studies: Depression
- Geriatric Depression Scale
- Hamilton Depression Rating Scale
- 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: National Computer Systems.
- Derogatis, L. R. (2000). Brief Symptom Inventory-18 (BSI-18): Administration, scoring, and procedures manual. Minneapolis: National Computer Systems.
- Derogatis, L. R., & Melisaratos, N. (1983). The Brief Symptom Inventory: An introductory report. *Psychological Medicine*, 13, 595–605.
- 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.
- 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.
- Hicks, R., Giacino, J., Harrison-Felix, C., Manley, G., Valadka, A., & Wilde, E. A. (2013). Progress in developing common data elements for traumatic brain injury research: Version two–the end of the beginning. *Journal of Neurotrauma*, 30, 1852–1861.
- 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.
- Wilde, E. A., Whiteneck, G. G., Bogner, J., Bushnik, T., Cifu, D. X., Dikmen, S., ..., & von Steinbuechel, N. (2010). Recommendations for the use of common outcome measures in traumatic brain injury research. *Archives of Physical Medicine and Rehabilitation*, 91, 1650–1660.e1617.

Brief Test of Attention

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The Brief Test of Attention (BTA) is a test of attention based on Cooley and Morris's conceptualization of attentional processes (Cooley and Morris 1990; Schretlen et al. 1996a). It was developed to be a pure measure auditory divided attention, and as such attempts to eliminate possible confounds of other attentional tasks such as motor and reasoning component (Schretlen et al. 1996a). It has also been suggested the BTA may be a useful embedded measure of cognitive effort (Busse and Whiteside 2012). The BTA has been used to assess attention in a variety of populations including Parkinson's disease, sleep apnea, cancer, and traumatic brain injury (TBI; Aloia et al. 2003; Butler et al. 2008; Rao et al. 2010; Tröster et al. 1997; Wong 1999). Of note, the BTA is not intended to measure normal attention but instead to be a screening tool for attentional deficits (Schretlen 1997; Strauss et al. 2006). Additionally, the BTA does not assess visual attention (Strauss et al. 2006).

The BTA takes approximately 10 min to administer and has two parts (Schretlen 1997; Strauss et al. 2006). In both parts, individuals are asked to listen to a voice on a recording read 10 lists of letters and numbers. The length of each list ranges from 4 to 18 items. In the first part, individuals are asked count how many numbers are read, ignoring the letters in each list. In the second part, individuals count how many letters are read, ignoring the number for each list. The specific numbers and letters do not need to be recalled, just the total amount read per list. One point is given for each correctly counted trial. Possible scores range from 0 to 20 points. Normative data is available for individuals age 6-14 and 17-82 (Strauss et al. 2006). Recently, normative data of the BTA was published for Spanish speaking adults in 11 Latin America countries (Rivera et al. 2015).

The BTA has been shown to correlate most strongly with other known attentional test, and specifically may be more related to more complex attentional tasks (Trails B; digit backwards; Schretlen et al. 1996a). Initial validity and reliability measures for the BTA indicate acceptable internal consistency (coefficient $\alpha = 0.82$) in adults (Schretlen et al. 1996a). When both clinical and healthy populations were combined internal consistency was high (coefficient $\alpha = 0.91$; Schretlen et al. 1996a). There have been variable results from examination of test-retest reliability for the BTA. In one study of adolescent girls tested 3 months apart, test-retest reliability was low (r = 0.45), however it was suggested that limited range may have contributed to this finding (Schretlen 1997). In contrast, an examination of older adults assessed at baseline and 9 months reported adequate test-retest reliability (r = 0.70; Schretlen 1997). While practice affects appear to be small or nonexistent, there have been concerns regarding a ceiling effect in the test (Schretlen

1997; Strauss et al. 2006). Age is the largest demographic variable predicting performance on the BTA, with older age associated with poorer performance (Schretlen 1997; Schretlen et al. 1996a; Strauss et al. 2006). Other demographic variables that may contribute to BTA performance include ethnicity, education level, and gender (Schretlen et al. 1996a; Schretlen 1997; Strauss et al. 2006). Demographic variables as a whole account for 17.5% of the variance in scores on the BTA, with age being the largest contributor to variance (Schretlen 1997).

BTA has been shown to be sensitive to individuals with mild head injury (Wong 1999). A report examining the validity of the BTA in Huntington's disease patients found this group performed significantly worse than controls on the BTA. In contrast, a small group on amnestic patients were not found to perform differently from controls on the BTA, suggesting intact memory function may not be needed to successfully complete the BTA (Schretlen et al. 1996b).

Overall, the BTA appears to be a valid and relatively consistent measure of divided auditory attention. More research examining validity and reliability in minority populations is needed, as is further evaluation of test-retest reliability in different populations.

References

- Aloia, M. S., Ilniczky, N., Di Dio, P., Perlis, M. L., Greenblatt, D. W., & Giles, D. E. (2003). Neuropsychological changes and treatment compliance in older adults with sleep apnea. *Journal of Psychosomatic Research*, 54(1), 71–76. https://doi.org/10.1016/ S0022-3999(02)00548-2.
- Busse, M., & Whiteside, D. (2012). Detecting suboptimal cognitive effort: Classification accuracy of the Conner's continuous performance test-II, brief test of attention, and trail making test. *The Clinical Neuropsychologist, 26*(4), 675–687. https://doi.org/10.1080/ 13854046.2012.679623.
- Butler, R. W., Copeland, D. R., Fairclough, D. L., Mulhern, R. K., Katz, E. R., Kazak, A. E., Noll, R. B., Patel, S. K., & Sahler, O. J. Z. (2008). A multicenter, randomized clinical trial of a cognitive remediation program for childhood survivors of a pediatric malignancy. *Journal* of Consulting and Clinical Psychology, 76(3), 367.
- Cooley, E. L., & Morris, R. D. (1990). Attention in children: A neuropsychologically based model for assessment.

Developmental Neuropsychology, 6(3), 239–274. https://doi.org/10.1080/87565649009540465.

- Rao, V., Bertrand, M., Rosenberg, P., Makley, M., Schretlen, D. J., Brandt, J., & Mielke, M. M. (2010). Predictors of new-onset depression after mild traumatic brain injury. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 22(1), 100–104. https://doi.org/ 10.1176/appi.neuropsych.22.1.100.
- Rivera, D., Perrin, P. B., Aliaga, A., Garza, M. T., Saracho, C. P., Rodriguez, W., Justo-Guillen, E., Aguayo, A., Schebela, S., Gulin, S., Weil, C., Longoni, M., Ocampo-Barba, N., Galarza-del-Angel, J., Rodriguez, D., Esencarro, L., Garcia-Egan, P., Martinez, C., & Arango-Lasprilla, J. C. (2015). Brief test of attention: Normative data for the Latin American Spanish speaking adult population. *Neuropsychological Rehabilitation*, 37, 663–676.
- Schretlen, D. (1997). Brief test of attention professional manual. Odessa: Psychological Assessment Resources.
- Schretlen, D., Bobholz, J. H., & Brandt, J. (1996a). Development and psychometric properties of the brief test of attention. *The Clinical Neuropsychologist*, 10(1), 80–89. https://doi.org/10.1080/13854049608406666.
- Schretlen, D., Brandt, J., & Bobholz, J. H. (1996b). Validation of the brief test of attention in patients with Huntington's disease and amnesia. *The Clinical Neuropsychologist*, 10(1), 90–95. https://doi.org/10.1080/ 13854049608406667.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). A compendium of neuropsychological tests: Administration, norms, and commentary. New York: Oxford University Press.
- Tröster, A. I., Fields, J., Wilkinson, S., Pahwa, R., Miyawaki, E., Lyons, K., & Koller, W. (1997). Unilateral pallidal stimulation for Parkinson's disease: Neurobehavioral functioning before and 3 months after electrode implantation. *Neurology*, 49(4), 1078–1083.
- Wong, T. M. (1999). Validity and sensitivity of the brief test of attention with acute brain injury and mild head injury patients. *Archives of Clinical Neuropsychology*, 14(8), 617–818. [Abstract].

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 fourfactor structure consisting of instrumental ADLs (seven items explaining 40.3% of variance), self care (six items explaining 10.3% of variance), orientation (five items explaining 7.5% of variance), and mobility (two 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 et al. 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 toward 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 openlabel 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 moderatesevere AD (Jones et al. 2009), and RCTs of reminiscence therapy (Woods et al. 2009) and interpersonal psychotherapy (Burns et al. 2005) for people with Alzheimer'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

- Activities of Daily Living Questionnaire
- Alzheimer's Disease Cooperative Study ADL Scale
- Disability Assessment for Dementia
- Lawton-Brody Instrumental Activities of Daily Living Scale

References and Readings

- AD2000 Collaborative Group. (2008). Aspirin in Alzheimer's disease (AD2000): A randomised openlabel 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., & Haworth, J. (2002). Bristol activities of daily living scale: A critical evaluation. *Expert Reviews* in Neurotherapeutics, 2, 669–676.
- 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.
- 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.

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) and 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 1850.

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 wellrespected 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 leftfrontal 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 leftfrontal 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 two 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

- Broca, P. P. (1850). Memoire sur l'anatomie pathologique du cancer. Bulletin de la Societe Anatomique de Paris, 25, 45.
- Broca, P. P. (1856). Traite des anevrismes et leur traitement. Paris: Labe & Asselin.
- Broca, P. P. (1861a). Sur le principe es localisations cerebrales. Bulletin de la Societe d'Anthropologie, 2, 190–204.
- Broca, P. P. (1861b). Nouvelle observation d'aphemie produite par une lesion de la moitie posterieure de deuxieme et troisieme circonvolution frontales gauches. *Bulletin de la Societe Anatomique, 36*, 398–407.
- Broca, P. P. (1863). Localisations des fonctions cerebrales. Siege de la faculte du language articule. Bulletin de la Societe d'Anthropologie, 4, 200–208.
- 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. (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 Neurosur*gery, 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 wordfinding 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 et al. 2008). The debate about localization of Broca's aphasia is part of a larger theoretical discussion of the modularity of language and other cognitive functions, in which localizationist views are contrasted with processing accounts (e.g., that Broca's aphasia results from slow lexical activation) (Patil et al. 2016). For the treatment implications of this debate, see discussion in Basso and 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 and Hassan 2002). Scales designed to limit verbal demands, such as the Cornell Depression Scale (Alexopoulos et al. 1988), have been used in studies of aphasia but have not been validated for this population (Townend et al. 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 et al. 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 postonset (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 et al. 2009), constraintinduced aphasia therapy (Cherney et al. 2008), training of communication partners (Kagan et al. 2001), and direct training of underlying grammatical structures (Thompson and 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 Coammunication Disorders and Sciences. Evidence-based practice guidelines (association). www.ancds.org. Retrieved 1 Oct 2008.

- 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: Academic.
- Helm-Estabrooks, N. (2001). Cognitive linguistic quick test (1st ed.). San Antonio: 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.
- Patil, U., Hanne, S., Burchert, F., De Bleser, R., & Vasishth, S. (2016). A computational evaluation of sentence processing deficits in aphasia. *Cognitive Science*, 40(1), 5–50.
- 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 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 Nisslstained 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 (trans: Carey L. J.). 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 groups 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. John E. Mendoza Department of Psychiatry and Neuroscience, Tulane Medical School and SE Louisiana Veterans Healthcare System, New Orleans, LA, USA

Synonyms

Hemisection of spinal cord

Definition

Brown-Séquard 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
- Posterior Columns

B

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 taskspecific 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. Each item is scored using a pass/fail criteria based upon task-specific performance or time standards. 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 itemtotal correlations of more than 0.20. Cronbach's alpha, a measure of internal consistency, was 0.93 (Tyson and 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 and 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 poststroke in 102 individuals (Tyson et al. 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 poststroke 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.

Minimal Clinically Important Difference (MCID): Due to the hierarchical nature of the scale, the MCID is considered to be a change of one level.

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.

Bulimia Nervosa

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Synonyms

Bulimia

Definition

Bulimia nervosa is defined in the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5; American Psychiatric Association 2013) as a feeding and eating disorder characterized by episodes of binge eating that occur during a discrete period of time and are accompanied by a sense of lack of control. Behaviors to expel food eaten (e.g., self-induced vomiting, laxatives) and/or restrictive caloric intake behaviors typically follow binge eating episodes. Additionally, individuals engage in inappropriate behaviors to prevent weight gain, and self-evaluation is disproportionately influenced by body shape and weight. Individuals typically experience fear of gaining weight and a desire to lose weight, as is common in anorexia nervosa.

Categorization

The disorder is classified with the Feeding and Eating Disorders in DSM-5.

Current Knowledge

Development and Course

The development of bulimia nervosa is common during adolescence and young adulthood and is often associated with stressful life events or the occurrence of binge eating behavior during or following a period of dieting. The disorder is 10 times more common in females than males, with a prevalence of approximately 1-3% (APA 2013). Risk of suicidal ideation or behaviors is greater in individuals with this disorder.

Associated Features and Current Research

Individuals with bulimia nervosa are typically within the normal weight to overweight range (body mass index \geq 18.5 and <30). The disorder can produce a number of medical problems, including amenorrhea, electrolyte imbalance, and gastrointestinal dysfunction, and there is a significant risk of mortality. A wide range of functional limitations, such as social disturbances, are associated with bulimia nervosa. Additionally, studies have identified neuropsychological impairments in the areas of attention and executive functioning that are associated with bulimia nervosa (Lauer 2002). Common comorbid conditions include mood disorders, anxiety disorders, substance abuse, and personality disorders (most commonly borderline personality disorder).

Assessment and Treatment

Diagnosis typically involves psychiatric assessment, physical examination, and laboratory testing for associated medical conditions. Course of treatment is also impacted by the information gathered in the diagnostic phase, as well as factors such as interpersonal history and functioning, medical and psychiatric comorbidity, and previous treatment attempts. Treatment often combines pharmacological (e.g., antidepressants) and psychological interventions. Cognitive behavioral therapy has emerged as the superior treatment approach (McGilley and Pryor 1998). Differential diagnoses include anorexia nervosa, binge eating disorder, major depression, and borderline personality disorder.

See Also

- Anorexia Nervosa
- Binge-Eating Disorder
- Body Dysmorphic Disorder
- ► Feeding and Eating Disorders
- Rumination Disorder

References and Readings

- American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (DSM-5[®]). Washington, DC: American Psychiatric Association.
- Jenkins, P. E., Luck, A., Cardy, J., & Staniford, J. (2016). How useful is the DSM-5 severity indicator in bulimia nervosa? A clinical study including a measure of impairment. *Psychiatry Research*, 246, 366–369.
- Lauer, C. J. (2002). Neuropsychological findings in eating disorders. In *Biological psychiatry* (pp. 1167–1172). New York: Wiley.
- McGilley, B. M., & Pryor, T. L. (1998). Assessment and treatment of bulimia nervosa. *American Family Physician*, 57(11), 2743–2750.
- Wonderlich, S. A., Peterson, C. B., Crosby, R. D., Smith, T. L., Klein, M. H., Mitchell, J. E., & Crow, S. J. (2014). A randomized controlled comparison of integrative cognitive-affective therapy (ICAT) and enhanced cognitive-behavioral therapy (CBT-E) for bulimia nervosa. *Psychological Medicine*, 44(03), 543–553.

Bupropion

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Generic Name

Bupropion, Bupropion hydrobromide

Brand Name

Wellbutrin, Wellbutrin SR, Wellbutrin XL, Zyban, Aplenzin, Buproban, Budeprion SR, and Forfivo XL

Class

Antidepressants, smoking cessation aids, and dopamine reuptake inhibitors

Proposed Mechanism(s) of Action

Increases norepinephrine/noradrenaline and dopamine, blocks norepinephrine reuptake pump, may increase dopamine neurotransmission in the frontal cortex, and blocks dopamine reuptake pump.

Indication

Major depressive disorder, seasonal affective disorder, and smoking cessation.

Off-Label Use

Bipolar disorder, ADHD, and neuropathic pain.

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, tremor, palpitation, anorexia, urinary frequency, and sweating.

References and Readings

Physicians' Desk Reference (71st ed.). (2017). Montvale: Thomson PDR.

Stahl, S. M. (2007). Essential psychopharmacology: The prescriber's guide (2nd ed.). New York: Cambridge University Press.

Additional Information

- Centerwatch: https://www.centerwatch.com/druginformation/fda-approved-drugs/drug/986/aplenzinbupropion-hydrobromide.
- 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. Free Drug Online and PDA Software: www.medscape.com.

Gene-Based Estimate of Drug interactions: http://

- mhc.daytondcs.com:8080/cgibin/ddiD4?ver=4&task= getDrugList.
- Pill Identification: http://www.drugs.com/pill_identification.html.

Burden of Proof

Moira C. Dux US Department of Veteran Affairs, 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 of 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.

Buspirone

Efrain Antonio Gonzalez College of Psychology, Nova Southeastern University, Fort Lauderdale, FL, USA Utah State University, Logan, UT, USA

Generic Name

Buspirone

Brand Name

BuSpar, Buspirex, Bustab, and Linbuspirone

Class

Antianxiety agents, anxiolytics, and nonbenzodiazepines

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, and smoking cessation.

Side Effects

Serious

Very rare cardiac symptoms.

Common

Dizziness, sedation, nervousness, and nausea.

References and Readings

- *Physicians' Desk Reference* (71st ed.). (2017). Montvale: Thomson PDR.
- Stahl, S. M. (2007). Essential psychopharmacology: The prescriber's guide (2nd ed.). New York: 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.
- Free Drug Online and PDA Software: www.medscape. com.
- Gene-Based Estimate of Drug interactions: http:// mhc.daytondcs.com:8080/cgibin/ddiD4?ver=4&task= getDrugList.
- Pill Identification: http://www.drugs.com/pill_identification.html.

Butters, Nelson (1937–1995)

Meryl A. Butters and James T. Becker Department of Psychiatry, University of Pittsburgh School of Medicine, WPIC, Pittsburgh, PA, USA

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 and sometimes implicit - that was woven into the fabric of Butters' research career.
- An early series of studies in the 1970s 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, nonamnestic 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

- 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.

٠ 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 preclinical 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 biological 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 Veterans 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.

Cross-References

- Alzheimer's Disease
- ► Amnesia
- Amnestic Disorder
- Amnestic Syndromes
- Anterograde Amnesia
- Dementia
- Episodic Memory
- ► Goodglass, Harold (1920–2002)
- Implicit Memory
- ► Kaplan, Edith (1924–2009)
- ► 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 Lobe
- ► Thalamus

References and Readings

- Albert, M. S., Butters, N., & Brandt, J. (1981). Patterns of remote memory in amnesic and demented patients. *Archives of Neurology*, 38(8), 495–500. PMID: 6454407.
- Brandt, J., Butters, N., Ryan, C., & Bayog, R. (1983). Cognitive loss and recovery in long-term alcohol abusers. *Archives of General Psychiatry*, 40(4), 435–442.
- Butters, N. (1966). The effect of LSD-25 on spatial and stimulus perseverative tendencies in rats. *Psychopharmacologia*, 8(6), 454–460.
- Butters, N. (1984). The clinical aspects of memory disorders: Contributions from experimental studies of amnesia and dementia. *Journal of Clinical Neuropsychology*, *6*(1), 17–36. PMID: 6230375.
- Butters, N. (1992). Memory remembered: 1970–1991. *Archives of Clinical Neuropsychology*, 7(4), 285–295. PMID:14591284.
- Butters, N. (1996). Memoirs, in Nelson Butters remembered. APA Division 40 Newsletter, 14(3), 1, 3–7.
- 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.

- Butters, N., & Cermak, L. S. (1974). The role of cognitive factors in the memory disorders of alcoholic patients with the Korsakoff syndrome. *Annals of the New York Academy of Sciences*, 233, 61–75. PMID: 4523804.
- Butters, N., & Pandya, D. (1969). Retention of delayedalternation: Effect of selective lesions of sulcus principalis. *Science*, 165(3899), 1271–1273. PMID: 4979528.
- Butters, N., & Rosvold, H. E. (1968). Effect of caudate and septal nuclei lesions on resistance to extinction and delayed-alternation. *Journal of Comparative and Physiological Psychology*, 65(3), 397–403. PMID: 4970002.
- Butters, N., Lewis, R., Cermak, L. S., & Goodglass, H. (1973). Material-specific memory deficits in alcoholic Korsakoff patients. *Neuropsychologia*, 11(3), 291–299. PMID: 4792180.
- Butters, N., Tarlow, S., Cermak, L. S., & Sax, D. (1976). A comparison of the information processing deficits of patients with Huntington's chorea and Korsakoff's syndrome. *Cortex*, 12(2), 134–144. PMID: 133786.
- Butters, N., Wolfe, J., Martone, M., Granholm, E., & Cermak, L. S. (1985). Memory disorders associated with Huntington's disease: Verbal recall, verbal recognition and procedural memory. *Neuropsychologia*, 23(6), 729–743. PMID: 2934642.
- Butters, N., Granholm, E., Salmon, D. P., Grant, I., & Wolfe, J. (1987). Episodic and semantic memory: A comparison of amnesic and demented patients. *Journal of Clinical and Experimental Neuropsychology*, 9(5), 479–497. PMID: 2959682.
- 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.
- Cermak, L. S. (Ed.). (1994). Neuropsychological explorations of memory and cognition: Essays in honor of Nelson Butters. New York: Plenum.
- Chan, A. S., Butters, N., Paulsen, J. S., Salmon, D. P., Swenson, M. R., & Maloney, L. T. (1993). An assessment of the semantic network in patients with Alzheimer's disease. *Journal of Cognitive Neuroscience*, 5(2), 254–261. https://doi.org/10.1162/ jocn.1993.5.2.254. PMID: 23972157.
- Granholm, E., & Butters, N. (1988). Associative encoding and retrieval in Alzheimer's and Huntington's disease. *Brain and Cognition*, 7(3), 335–347. PMID: 2969744.
- Heindel, W. C., Salmon, D. P., Shults, C. W., Walicke, P. A., & Butters, N. (1989). Neuropsychological evidence for multiple implicit memory systems: A comparison of Alzheimer's, Huntington's, and Parkinson's disease patients. *The Journal of Neuroscience*, 9(2), 582–587. PMID: 2521896.
- Hodges, J. R., Salmon, D. P., & Butters, N. (1990). Differential impairment of semantic and episodic memory in Alzheimer's and Huntington's diseases: A controlled prospective study. *Journal of Neurology, Neurosurgery, and Psychiatry, 53*(12), 1089–1095. PMID: 2149861.

- Jernigan, T., & Butters, N. (1989). Neuropsychological and neuroradiological distinctions between Alzheimer's and diseases. *Neuropsychology*, 3, 283–290.
- Moss, M. B., Albert, M. S., Butters, N., & Payne, M. (1986). Differential patterns of memory loss among patients with Alzheimer's disease, Huntington's disease, and alcoholic Korsakoff's syndrome. *Archives of Neurology*, 43(3), 239–246. PMID: 2936323.
- Rosen, J., Stein, D., & Butters, N. (1971). Recovery of function after serial ablation of prefrontal cortex in the rhesus monkey. *Science*, 173(3994), 353–356. PMID: 4997798.
- Shimamura, A. P., Salmon, D. P., Squire, L. R., & Butters, N. (1987). Memory dysfunction and word priming in dementia and amnesia. *Behavioral Neuroscience*, 101(3), 347–351. PMID: 2955793.
- Tröster, A. I., Butters, N., Salmon, D. P., Cullum, C. M., Jacobs, D., Brandt, J., & White, R. F. (1993). The diagnostic utility of savings scores: Differentiating Alzheimer's and Huntington's diseases with the logical memory and visual reproduction tests. *Journal of Clinical and Experimental Neuropsychology*, 15(5), 773–788. PMID: 8276935.