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Saccadic Eye Movements

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Definition

Saccades are rapid, abrupt, usually conjugate eye movements that redirect the fovea to new areas of interest in the visual field.

Current Knowledge

A saccade takes about 150–250 ms to plan and execute. The execution of a single saccade takes about 30 ms, but it can vary between 20 and 100 ms depending on the angular distance traveled by the eye (amplitude). During this interval, the eyes can move at a speed of up to 900° per second. Head-fixed saccades have amplitudes ranging between 1.2 and 90°, but saccades with amplitudes larger than 20° tend to be accompanied by head movements. Saccades are also very accurate bringing the line of gaze typically within a 1° of the target. As ballistic movements, that is, movements whose trajectory cannot be changed during their execution, saccades often require and are followed by additional corrective saccades. For instance, saccades made to targets across a distance of more than 10° often undershoot the target by 10%, and after a short latency of about 150 ms, are followed by a corrective saccade.

Between two consecutive saccades, the eyes fixate the area of interest for a variable interval averaging at about 300 ms. During *fixation*, the area fixated is processed at a higher resolution taking advantage of the fact that it projects on the fovea. Most of visual perception takes place during this interval. On the other hand, vision is attenuated during the execution of a saccade, a mechanism known as *saccadic suppression*. This mechanism prevents the experience of blurred vision due to the image displacement taking place during a saccade. Given a visual scene, saccades facilitate the process of collecting high-resolution information from different parts of the scene, information collected, and integrated over a sequence of fixations.

Saccades, along with all types of eye movements, result from the action of the extraocular muscles, which control eye rotations in horizontal, vertical, and torsional directions. Two main types of neurons are responsible for saccade generation, known as premotor neurons as they project monosynaptically to ocular motor neurons. Premotor neurons in the paramedian pontine reticular formation control horizontal eye movements, while premotor neurons in the rostral interstitial nucleus in the midbrain control vertical movements. Oblique saccades are the result of joint horizontal and vertical eye movements. Omnipause neurons lying close to the midline in the raphe interpositus nucleus inhibit premotor neurons and thus prevent new saccades and allow for fixations. The selection of a saccade target is provided by two neural structures, the superior colliculus of the midbrain and the frontal eye field in the frontal lobe. Both these structures contain topographical motor maps. Activation of a particular site in these structures produces saccadic eye movements in a specified direction and for a specified distance independent of the current position of the eyes. The superior colliculus provides the motor command to the premotor neurons and controls the omnipause neurons, allowing the generation of saccades. The frontal eye field projects to the superior colliculus as well as to the premotor neurons. As a result, the frontal eye field can also control eye movements independently of the superior colliculus. In addition to these two structures, the cerebellum also influences the premotor and omnipause neurons, its main role being to control the accuracy of the saccades.

Saccade disorders include *opsoclonus*, slow saccades, and "stuttering" saccades. Opsoclonus consists of bursts of high-frequency oscillations of the eyes. Each burst of oscillations can be analyzed as a continuous series of saccades lacking intersaccadic intervals (Ashe et al., 1991). Patients with opsoclonus can experience vertigo and oscillopsia (illusory motion of the visual world) and often have

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clinical evidence of cerebellar ataxia. Slow saccades, affecting the horizontal, the vertical, or both components of eye motions, are associated with a variety of conditions. They can be caused by a number of genetic and degenerative diseases affecting neurons in the brainstem and cerebellum, by diseases affecting the extraocular muscles as well as by brainstem stroke (Leigh & Zee, 2006). Another disorder, transient deceleration of eye movement during saccades, also known as "stuttering" saccades, is found in certain diseases, such as the late-onset Tay–Sachs disease (Rucker et al., 2004), which affects the brainstem and the cerebellum.

Cross References

- ▶ Cerebellum
- ▶ Fovea
- ▶ Frontal Eye Fields
- ▶ Oculomotor Nerve
- ▶ Superior Colliculus

References and Readings

Ashe, J., Hain, T. C., Zee, D. S., & Schatz, N. J. (1991). Microsaccadic flutter. *Brain: A Journal of Neurology, 114*(Pt 1B), 461–472.

Leigh, R. J., & Zee, D. S. (2006). *The neurology of eye movements.* New York: Oxford University Press.

Rucker, J. C., Shapiro, B. E., Han, Y. H., Kumar, A. N., Garbutt, S., Keller, E. L. et al. (2004). Neuro-ophthalmology of late-onset Tay-Sachs disease (LOTS). *Neurology, 63*(10), 1918–1926.

Sagittal Sinus Thrombosis

- ▶ Central Venous Thrombosis

Sarcoma

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Definition

Sarcomas are cancerous lesions comprising connective or supportive tissue (e.g., bone, cartilage, and soft tissue) that frequently affect the limbs. These tumors are formed by the proliferation of mesodermal cells and can be very malignant. However, they comprise only 1% of malignant neoplasms and central nervous system (CNS) sarcomas encompass approximately 0.5% of all soft tissue sarcomas. There are two types of brain sarcomas: one occurs as a component within a primary brain tumor and does infiltrate the brain surface, and the other develops from mesenchymal tissue. Sarcomas also manifest following the late effects of radiotherapy (5–15 years after treatment). Osteosarcomas are the most common type of malignant bone cancers, accounting for nearly 35% of primary bone malignancies (Adigun & Rahman, 2007). Ewing's sarcoma, a rare variant of this disease, can infiltrate the skull and compress the brain parenchyma. The neurological sequelae correspond to the size and region in which the tumor is located. Low-grade sarcomas are usually treated surgically, whereas high-grade lesions are treated more aggressively with adjuvant therapy. Since sarcomas are relatively rare, there is scant reported medical experience in treating this disease.

References and Readings

Adigun, I. A., & Rahman, G. A. (2007). A review of soft tissue sarcoma. *Nigerian Journal of Medicine, 16*, 94–101.

Merimsky, O., Lepechoux, C., Terrier, P., Vanel, D., Delord, J. P., LeCesne, A. (2008). Primary sarcomas of the central nervous system. *Oncology, 58*, 210–214.

SAS

- ▶ Stroke Activity Scale

SA-SIP

- ▶ Stroke-Adapted Sickness Impact Profile

SA-SIP30

- ▶ Stroke-Adapted Sickness Impact Profile

SAT

► Scholastic Aptitude Test

Satisfaction with Life Scale

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Synonyms

Diener life satisfaction scale; SWLS

Description

The SWLS is a 5-item measure of life satisfaction, conceptualized as an individual's judgment of their overall quality of life (Diener, Emmons, Larsen, & Griffin, 1985). Each item is rated on a 7-point scale, ranging from 1 (*strongly disagree*) to 7 (*strongly agree*), with a score of 4 (*neither agree nor disagree*), indicating a neutral position on an item. The scores are summed to provide a total score ranging from 5 to 35, with higher scores indicating greater subjective life satisfaction. It is intended to be a measure of global life satisfaction, and thus does not assess satisfaction in specific life domains (e.g., work satisfaction, relationship satisfaction).

Background

The construct of life satisfaction is conceptualized as one of two components of an individual's sense of well-being, the other component being affective state. While affective state is the emotional component of an individual's experience of well-being, life satisfaction is a cognitive process by which individuals judge their life. The premise underlying the construction of the SWLS was that this process of judging life satisfaction is purely subjective, that is, the individual has a set of internal standards on which evaluation of life satisfaction is based. If their perception of their life does not meet these internal standards, life satisfaction is judged to be poorer. These subjective, internal standards that the individual uses to evaluate life satisfaction contrast with judgments based on externally provided standards (i.e., standards believed by researchers or clinicians to be important to life satisfaction).

The SWLS was constructed to fill a gap in life satisfaction research and assessment by providing a scale that captures an individual's *global* evaluation of *subjective* (i.e., standards that are internally generated) life satisfaction. At the time of the development of the SWLS (Diener et al., 1985), other life satisfaction scales were available only for geriatric populations or were confounded the assessment of the cognitive process of life satisfaction judgment with the affective facet of well-being.

Psychometric Data

Reliability

Reliability estimates are generally limited to samples without neurological involvement. Internal consistency (Cronbach's α) estimates range from 0.79 to 0.89 across different samples of varying size and composition (Pavot & Diener, 1993, 2008), with the typical internal consistency in the low to mid 0.80s. Test–retest reliabilities range from 0.50 to 0.84 in different time frames from 2 weeks to 4 years. In general, the longer the test–retest interval, the lower the test–retest reliability value. Interrater reliability has been estimated at 0.73 (see Pavot & Diener, 1993, 2008 for reviews).

Validity

Factor structure. The initial report of the SWLS (Diener et al., 1985) found that the items represented a single factor, and this finding has been consistently replicated in numerous studies. Item–total correlations range from 0.57 to 0.81. Some reports find that item five of the SWLS has the lowest item-total correlation, and the authors suggest that this item can be deleted without significant reduction in psychometric quality (Pavot & Diener, 2008).

Construct validity. Construct validity has been explored in numerous studies through correlations with other measures of life satisfaction and related constructs such as negative affect and self-esteem. These studies consistently find that the SWLS has generally moderate negative correlations (typically in the -0.40s to -0.50s) with measures of depression and negative affect (e.g., ► Beck Depression Inventory, Positive and Negative Affect Schedule), and moderate positive correlations with

self-esteem (Rosenberg Self-Esteem Scale). Correlations between the SWLS and other measures of life satisfaction tend to be in the 0.60s to 0.80s, and the SWLS shows considerably more convergence with other measures of life satisfaction than it does with measures of related constructs such as affect (Lucas, Diener, & Suh, 1996).

Criterion-related validity. Criterion-related validity is seen in evidence of significantly lower SWLS scores in individuals hypothesized to have lower life satisfaction (e.g., prisoners, abused women). In addition, the SWLS appears sensitive to life changes that would be expected to alter judgment of life satisfaction (Pavot & Diener, 2008). For example, caregivers of spouses with a progressive dementia process indicate lower SWLS scores across time, which correspond to the increasing severity of the spouse's dementia. Individuals undergoing psychotherapy also show significantly higher SWLS scores after 1 month of therapy as compared to the beginning of therapy.

Discriminant validity. Lucas et al. (1996) used multitrait–multimethod matrix analyses to explore the discriminant validity of the SWLS. This study found that the SWLS converged much more highly with other measures of life satisfaction than with other measures of related constructs such as affect. Factor analytic studies show that life satisfaction and affect, while significantly related, load on separate factors under the superordinate factor of well-being.

Clinical Uses

The SWLS has the advantage of being a brief and focused measure of life satisfaction appropriate for use across different settings (e.g., community samples, psychiatric samples, college students) and across the age range. It has been translated into many languages, including Spanish, German, Japanese, Korean, Russian, and Arabic. Extensive normative data (Pavot & Diener, 2008) allows the clinician/researcher to identify the most suitable comparative base. The authors (Pavot & Diener, 1993) recommend ranges of scores as reflecting different levels of life satisfaction: scores of 5–9 represent *extreme dissatisfaction with life*, scores of 10–14 represent *dissatisfaction with life*, scores of 15–19 represent *slight dissatisfaction* with life, scores of 21–25 represent *slight satisfaction with life*, scores of 26–30 represent *satisfaction with life*, and scores of 31–35 represent *extreme satisfaction with life*. A score of 20 is the midpoint of the scale, and reflects a neutral judgment of life satisfaction. SWLS total scores in most samples generally range from 23 to 28, but there is a large amount of variability. Since the SWLS is sensitive to change across time, it may be useful as an indicator of efficacy of a treatment or intervention in clinical studies.

The SWLS has been used in samples of individuals with health concerns, including traumatic brain injury, spinal cord injury, and stroke. While individuals with health concerns tend to endorse lower life satisfaction on the SWLS, the authors are explicit in stating that there is a large amount of variability among these samples (Pavot & Diener, 2008). In the traumatic brain injury recovery literature (e.g., Corrigan, Bogner, Mysiw, Clinchot, & Fugate, 2001), the SWLS appears most related to psychological well-being and social functioning, while physical factors and injury severity appear to have at most weak relations with life satisfaction. The relation between life satisfaction on the SWLS and social functioning is also consistently seen in samples of individuals with spinal cord injury and stroke survivors.

The use of the SWLS has several potential qualifying factors. It (and judgment of life satisfaction in general) has been suggested to be modified by personality characteristics, such as extraversion and neuroticism, which should be considered by the clinician/researcher in interpretation. Furthermore, the clinician/researcher should be mindful that the SWLS appears to be influenced by cultural factors; cross-cultural studies suggest that there are SWLS total score differences among countries depending on the type of society (e.g., individualistic vs. collectivistic, wealthy vs. poor; Pavot & Diener, 2008). It can also be affected by momentary mood, which may diminish the fidelity of assessing global life satisfaction.

Cross References

- ▶ Beck Depression Inventory
- ▶ Center for Epidemiological Studies-Depression
- ▶ Coping
- ▶ Hamilton Rating Scale for Depression
- ▶ Quality of Life
- ▶ Self-Report Measures
- ▶ Stress
- ▶ Test Reliability
- ▶ Test Validity
- ▶ Zung Self-Rating Depression Scale

References and Readings

Corrigan, J. D., Bogner, J. A., Mysiw, W. J., Clinchot, D., & Fugate, L. (2001). Life satisfaction after traumatic brain injury. *Journal of Head Trauma Rehabilitation, 16*, 543–555.

Diener, E., Emmons, R. A., Larsen, R. J., & Griffin, S. (1985). The satisfaction with life scale. *Journal of Personality Assessment, 49*, 71–75.

Lucas, R. E., Diener, E., & Suh, E. (1996). Discriminant validity of well-being measures. *Journal of Personality and Social Psychology, 71*, 616–628.

Pavot, W., & Diener, E. (1993). Review of the satisfaction with life scale. *Psychological Assessment, 5*, 164–172.

Pavot, W., & Diener, E. (2008). The satisfaction with life scale and the emerging construct of life satisfaction. *Journal of Positive Psychology, 3*, 137–152.

SB-5

► Stanford–Binet Intelligence Scales and Revised Versions

SBS

► Personality Inventory for Children
► Sick Building Syndrome

Schizotypal Disorder

► Schizotypal Personality Disorder

Schizotypal Personality Disorder

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Synonyms

Borderline schizophrenia; Latent schizophrenia; Prodromal schizophrenia; Schizotypal disorder; SPD

Short Description or Definition

Schizotypal personality disorder (SPD) is characterized as an enduring and pervasive pattern of deficits in social and interpersonal functioning and is associated with peculiarities in thinking, behavior, and appearance. Individuals with SPD frequently experience ideas of reference and paranoia, odd beliefs or magical thinking, unusual perceptual experiences, odd thinking manifested by odd speech that is not grossly disorganized, unusual or constricted affect, a lack of close friends and discomfort in close relationships, and social anxiety (APA, 2000). If psychotic symptoms do occur, they are brief and transient.

Categorization

SPD is classified as a Cluster A personality disorder by the American Psychiatric Association's DSM-IV-TR ("odd/eccentric" cluster) (APA, 2000). In ICD-10 classification, more emphasis is placed on this pattern of symptoms reflecting a prodromal, borderline, or latent schizophrenia. Three main underlying factors have been identified as the three core symptom clusters of schizotypal personality: cognitive-perceptual impairments (magical thinking, unusual perceptual experiences, ideas of reference, and paranoid ideation), interpersonal impairments (paranoid ideation, social anxiety, and a lack of close friends), and disorganized features (odd/eccentric behavior and odd speech). This three-factor structure has been widely validated among ethnically and clinically diverse populations (Raine, 2006).

Epidemiology

Lifetime prevalence is approximately 3%; with some evidence, males are more frequently affected than females.

Natural History, Prognostic Factors, and Outcomes

Established thinking is that the disorder most frequently has onset in adolescence and maintains a relatively stable course throughout adulthood. However, there is growing evidence that while this may be true for childhood-onset SPD, this is not the case when onset comes later in life. While early onset SPD does appear to remain relatively consistent, research with adults shows that SPD symptomatology diminishes with age, with remission rates of

between 20% and 60%. This has led some researchers to argue that there are two distinct subgroups of SPD, one that has a childhood onset and shows temporal stability, and another that has a variable age of onset and shows greater temporal variation in symptoms (Raine, 2006).

Individuals diagnosed with SPD tend to experience higher rates of psychosis and schizophrenia than those with other mental disorders, and those with no psychiatric diagnoses. The actual rate varies widely depending on the age of onset of SPD; however, estimates tend to fall somewhere between 3% and 40% of individuals developing either schizophrenia or some form of psychosis. SPD is also highly comorbid with schizoid, paranoid, avoidant, and borderline personality disorders. Nearly half of individuals with SPD have a history of at least one depressive episode (Raine, 2006). Given this high rate of depression, suicidality should be carefully monitored among those diagnosed with SPD. It is estimated that approximately 10% of individuals with SPD attempt suicide (Sadock & Sadock, 2003).

Neuropsychology and Psychology of Schizotypal Personality Disorder

As is common with many psychological disorders, SPD is thought to be the result of an interaction between a genetic diathesis and environmental influences. In the case of SPD, it is believed that SPD is a neurodevelopmental disorder, reflecting the role of genetics, prenatal, and postnatal environmental factors on brain development (Raine, 2006). It is hypothesized that early prenatal and postnatal factors influence the development of the brain, causing disturbances in the prefrontal, temporal, and limbic circuits of the brain. These disturbances then lead to impairments in executive functions, sustained attention, working memory, and inhibitory functions. Affective disturbances are also present with impairments in social and emotional information processing. It is hypothesized that these impairments produce the recognized pattern of symptoms in SPD: cognitive-perceptual, interpersonal, and disorganized features (Raine, 2006).

The role of genetics in the development of SPD is also well documented, SPD is much more common among relatives of individuals with SPD and schizophrenia than in the relatives of people with other mental illnesses or in people without mentally ill relatives. It is also more common among adopted offspring of a schizophrenic biological parent. Individuals with SPD have high levels of comorbidity with other disorders in the schizophrenia spectrum (e.g., 36% for paranoid schizophrenia and 10% for SPD) (McGlashan et al., 2000). This high comorbidity and high rates of familial schizophrenia both point to a significant genetic loading of SPD and a genetic link to schozophrenia. Heritability of schizotypal features has been estimated at 0.61 (Torgersen et al., 2000). While there is clear evidence for a strong genetic component, it has been proposed that the late onset subtype or pseudoschizotypy (Raine, 2006) is in fact unrelated to schizophrenia, and more dependent on psychosocial adversity.

Neurodevelopmental processes have highlighted several sources of evidence relating neurodevelopmental factors to SPD. Prenatal influenza exposure in the fifth and sixth months of pregnancy and prenatal stress in the sixth month have been associated with greater schizotypy scores in adulthood. In addition, individuals with SPD have shown higher rates of minor physical anomalies that reflect fetal neural maldevelopment between the second and third trimester. Birth complications including low birth weight and obstetric complications have been associated with later development of SPD. Prenatal malnutrition, malnutrition at 3 months, smaller adult height, and being bottle-fed all show links to the development of SPD.

The neurochemistry of SPD is remarkably underresearched. While there are a small number of studies that examine the role of dopamine in SPD, there is a complete absence of research on noradrenaline, epinephrine, gamma-aminobutyric acid, and glutamate (Raine, 2006). The dopamine hypothesis of schizophrenia highlights the positive correlation between dopamine and positive symptoms of schizophrenia and decreases in dopamine with the negative symptoms of schizophrenia (Amin, Silverman, Siever, Smith, Knott, & Davis, 1999). The role of dopamine in cognitive-perceptual symptoms of SPD has also been examined. Research shows that increases in dopamine found among those with SPD accounts fully for the cognitive-perceptual symptoms of SPD (Siever, 1995). Neurochemical imaging paradigms have also offered support for this "bidirectional hypothesis" (Raine, 2006). Neurodevelopmental research indicates that adolescents with SPD exhibit a higher rate of dyskinesias (involuntary movements) of the upper body, indicating possible involvement of motor cortex, striatum, or dopamine pathways.

Functional and structural imaging studies have indicated prefrontal structural abnormalities and abnormalities in the prefrontal circuitry. Individuals with SPD appear to have similar pattern of dysfunction in brain structures as schizophrenics (e.g., superior temporal gyrus, prefrontal cortex, thalamus, septum pellucidum) and these impairments tend to fall between unaffected individuals and those with schizophrenia (Buchsbaum

et al., 2002; Siever & Davis, 2004), indicating that SPD may be a milder form of schizophrenia. Brain anatomy differences involving the presence of the cavum septum pellucidum (a cavity in the septum pellucidum that usually fuses by 6 months of age) reflects a higher prevalence among schizotypals (27.3%) and schizophrenics (35%) as compared with controls (13%).

Aside from pre- and postnatal environmental influences, psychosocial adversity is a significant risk factor for SPD. Individuals diagnosed with SPD have significantly higher rates of early childhood trauma and abuse, including physical, sexual, emotional abuse, and neglect (Raine, 2006). Previous research has indicated that childhood trauma and abuse are associated with dissociative experiences, cognitive disorganization, perceptual aberrations, and magical ideation in later life. These experiences are thought to influence the development of SPD through two ways, firstly through neurobiological changes and secondly through social learning. The neurobiological hypothesis argues that early abuse and neglect may result in structural and neurochemical changes in the brain, resulting in functional impairments that contribute to schizotypal symptoms. This is supported by research that shows that early stress and trauma can cause neurodevelopmental reorganization within the brain. The second means is through social learning, this hypothesis emphasizes the importance of these early negative experiences in depriving the child of normal experiences of trust and security which would in turn predispose the individual to a paranoid attributional style, a distrust of people, and a lack of close friends.

Evaluation

Evaluation of the disorder usually occurs when the individual is brought in contact with a provider for other reasons such as depression or anxiety. Currently, there are no laboratory tests that can be used to evaluate for SPD, but as with most other psychological disorders evaluation is completed through face-to-face assessments of current symptomology, psychological, developmental, medical, educational, and social history, and through collateral information from medical records and family report. Tools to assist in the diagnosis of SPD include the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II; First, Spitzer, Gibbon, Williams, & Benjamin, 1994) and the Schizotypal Personality Questionnaire (SPQ; Raine, 1991) which are based on the nine diagnostic criteria of the DSM-IV (APA, 1994). Both exhibit high correlations with clinically assessed diagnosis.

Treatment

Treatment research has almost entirely focused on psychopharmacological interventions, with limited research on behavioral or psychological interventions for SPD, despite evidence for the role of psychological factors in SPD development.

Pharmacological treatment studies have consistently shown the benefit of psychotropic medications when treating SPD. Of the 13 experimental trials that include pharmacological treatment of SPD, all 13 showed improvements in SPD symptomatology as a result of treatment. Typical antipsychotics (e.g., thiothixene haloperidol), atypical antipsychotics (e.g., olanzapine, risperidone) and antidepressants (e.g., fluoxetine, amoxapine) were all found to be effective in treating the symptoms of SPD (Raine, 2006).

Low-dose antipsychotics are the treatment of first choice for individuals with cognitive/perceptual symptoms; however, they are effective in a wide array of symptoms including affective symptoms. Both typical and atypical antipsychotics were effective in reducing paranoia, ideas of reference, anxiety, and social isolation and improving social functioning. Atypical antipsychotics are well tolerated by patients, however olanzapine was linked to significant weight gain. For individuals with prominent depressed mood antidepressants may be the treatment of choice. Both fluoxetine and amoxapine reduced paranoia, depression, anxiety, and obsessive–compulsive symptoms (Raine, 2006).

Research on psychosocial and behavioral treatments of SPD are extremely limited; however, they mainly consist various forms of crisis management, psychoeducation, improving self-awareness, addressing social isolation, and skills training for coping with daily problems (Sadock & Sadock, 2003).

Cross References

- ▶ Psychosis
- ▶ Psychotic Disorder

References and Readings

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

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

Amin, F., Silverman, J. M., Siever, L. J., Smith, C. J., Knott, P. J., & Davis, K. L. (1999). Genetic antecedents of dopamine dysfunction in schizophrenia. *Biological Psychiatry, 45*, 1143–1150.

Buchsbaum, M. S., Nenadic, I., Hazlett, E. A., Spiegel-Cohen, J., Fleischman, M. B., et al. (2002). Differential metabolic rates in prefrontal and temporal Brodmann areas in schizophrenia and schizotypal personality disorder. *Schizophrenia Research, 54*, 141–150.

First, M. B., Spitzer, R. L., Gibbon, M., Williams, J. B. W., & Benjamin, L. (1994). *Structured clinical interview for DSM-IV axis II personality disorders (SCID-II, Version 2.0)*. New York: NY State Psychiatric Institute.

McGlashan, T. H., Grilo, C. M., Skodol, A. E., Gunderson, J. G., Shea, M. T., et al. (2000). The collaborative longitudinal personality disorders study: Baseline axis I/II and II/II diagnostic co-occurrence. *Acta Psychiatrica Scandinavica, 102*, 256–264.

Oldham, J. M., Skodol, A. E., & Bender, D. S. (Eds.). (2005). *The american psychiatric publishing textbook of personality disorders*. Washington, DC: American Psychiatric Publishing.

Raine, A. (2006). Schizotypal personality: Neurodevelopmental and psychosocial trajectories. *Annual Review Clinical Psychology, 2*, 291–326.

Raine, A., Lencz, T., & Mednick, S. A. (1995). *Schizotypal personality disorder*. Cambridge: Cambridge University Press.

Sadock, B. J., & Sadock, V. A. (2003). *Kaplan and sadock's synopsis of psychiatry: Behavioral sciences, clinical psychiatry*. Philadelphia, PA: Lippincott Williams & Wilkins.

Siever, L. J., & Davis, K. L. (2004). The pathophysiology of schizophrenia disorders: perspectives from the spectrum. *American Journal of Psychiatry, 161*, 398–413.

Torgersen, S., Lygren, S., Oien, P. A., Skre, I., Onstad, S., et al. (2000). A twin study of personality disorders. *Comprehensive Psychiatry, 41*, 416–425.

Scholastic Aptitude Test

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Synonyms

SAT

Description

The Scholastic Aptitude Test (SAT) is a standardized test designed to measure important skills required for academic success at tertiary level, and the current version contains three main sections measuring basic critical reading, math, and writing skills, as well as open category not included in the final score. The SAT is typically taken by high school students who require entry to colleges and universities in the United States; other countries (e.g., Israel, Sweden) (e.g., Bellar, 2009; Wedman, 1994) have also adopted the SAT as entrance exam for admission to higher education institutions. Research has shown that the SAT combined with high school grade point average (GPA) provides a good indicator of success in college (Coyle & Pillow, 2008; see also http://www.colegeboard.com/about/index/html).

The SAT contains 3 hour and 45 minutes of actual timed sections, although administrators of the test allow extra time for timed breaks, administration, and completion of biographical sections. The test consists of three major sections:

- Writing skills – a 60 min long section composed of both multiple choice questions and an essay assessing the student's ability to clearly communicate ideas, identify grammatical errors, and improve sentences and paragraphs.
- Mathematics – a 70 min long section composed of both multiple choice questions and student-produced response questions aimed at assessing, among others, students' ability to deal with numbers and operations, algebra, geometry, and data analysis.
- Critical reading – a 70 min long section aimed at testing a student's ability to understand what he or she has read, as well as vocabulary and sentence completion.

There is also an "experimental" section of 25 min, which may be in any of the three major sections and is used to normalize questions for future administration of the SAT. The experimental section does not count toward the final score and can be a reading, writing, or math section. The student does not know which section is experimental although he or she does know which type of section it is as there is an extra one of that type.

Historical Background

The first College Board test was administered in 1901 in 67 locations in the United States and two in Europe focusing on English, French, German, Latin, Greek, history, mathematics, chemistry, and physics (Hubin, 1988; Korbin, 2006). This was not a multiple-choice test and evaluated students' responses in terms of "excellent," "good," "doubtful," "poor," or "very poor." A growing desire by American educators to open their universities to the best students across the country resulted in the first SAT being administered in 1926. This test was

prepared by Princeton psychologist Carl Campbell Brigham, who also developed the IQ test, and assessed sections of definitions, arithmetic, classification, artificial language, antonyms, number series, analogies, logical inference, and paragraph reading. It was administered to over 8,000 students at more than 300 test centers, and test takers were given only a little over 90 min to answer the 315 questions. The objective of the original SAT was to eliminate test bias between people from different socioeconomic backgrounds (Hubin, 1988). Brigham later criticized the standardized testing movement, and developments on the SAT was stunted until his death in 1943. In 1946, Henry Chauncey, a dean at Harvard, became the first president of the Educational Testing Service and under his skilful leadership the SAT grew rapidly (Pacenza, http://journalism.nyu.edu/pubzone/race_class/edu-matt3.htm).

Since 1926, the number of sections on the SAT has been adapted and time limit increased to allow test takers more opportunity to achieve good scores. Initially, the adaptation of sections eliminated the math section entirely focusing only on verbal ability (1928–1929). In 1930, both verbal and math sections were included and this structure has remained the same until 2004 (Korbin, 2006). The mathematics sections were also reviewed and replaced with only multiple-choice questions. In 1946, paragraph reading and "double definition" questions were eliminated and replaced with reading comprehension and sentence completions, respectively. Several other changes occurred through the years, and in 1980, the SAT was adapted to also give minorities a better chance at being accepted into a college of higher standard (Korbin, 2006). Further changes to the mathematics section in 1994 resulted in the inclusion of non-multiple-choice questions including also sections on concepts of probability, slope, and elementary statistics, counting problems, median, and mode.

Initially, the test was known as "Scholastic Aptitude Test" but this later changed to Scholastic Assessment Test in 1990. Since 1993, only the letters SAT (with the letters standing for anything) were retained and now the scoring categories are the following: Critical Reading, Mathematics, and Writing. The SAT II or New SAT (known as the SAT Reasoning Test) was implemented in 2005 and has been made marginally harder as a corrective to the rising number of perfect scores.

Over the years, research has shown considerable differences in scores based on race, income, and parental educational background. Gender differences have also been noted (Mau & Lynn, 2001), while others claimed that males have greater general mental ability on the SAT (Frey & Detterman, 2003; Jackson & Rushton, 2006). Based on this and other critique, a growing number of liberal arts colleges as well as the University of California have decided to drop the requirement of the SAT for admission. This encouraged the College Entrance Examination Board to restructure the SAT and the most recent changes took effect in 2005.

Other criticism of the SAT refers to the inconsistency of assessing essays stating that the time allowed for the test pushes schools to develop a formulaic system of writing. The Advanced Placement (AP) program was designed as an alternative entrance requirements for university admission, although this tool is questioned with regard to its ability to reliably predict early college grades or retention (Klopfenstein & Thomas, 2009). Furthermore, although test preparation for the SAT has been criticized by some as having little effect, others have embraced the opportunity to improve their scores. Despite these and other criticisms, all schools in the United States nowadays accept the SAT as entrance exam and today 2.1 million America teenagers take the SAT for placement in college or university courses.

Psychometric Data

Each section receives a score on a scale of 200–800, with a total test score of 600–2,400. The total score is calculated by adding up the scores for the ten subsections. Students receive their score reports within 3 (online reports) to 6 (mailed, paper scores) weeks after administration. Each section is graded on a scale of 200–800 and two subscores for the writing section: the essay score and the multiple-choice subscore. The average score for each section (writing, reading and math) is about 500 with the total average score about 1,500. Although the College Board dropped the Score Choice Option in 2002, it has since decided to re-implement this option but some highly selective colleges and universities have announced they will still require applicants to submit all scores (see http://www.colegeboard.com/about/index/html for more information).

Students also receive their percentile ranking indicating the percentage of other test takers with lower scores. The corresponding percentile of each scaled score varies from test to test and depends largely on the content of the exam and the caliber of students choosing to take each exam. Table 1 gives an example of the percentiles and various SAT scores for college-bound seniors in 2006.

Scholastic Aptitude Test. Table 1 Percentiles for various SAT scores for college-bound seniors

Percentile	Score, 1,600 scale (official, 2006)	Score, 2,400 scale (official, 2006)
99.93/ 99.98[a]	1,600	2,400
99+	≥1,540	≥2,290
99	≥1,480	≥2,200
98	≥1,450	≥2,240
97	≥1,420	≥2,100
88	≥1,380	≥1,900
83	≥1,280	≥1,800
78	≥1,200	≥1,770
72	≥1,150	≥1,700
61	≥1,090	≥1,600
48	≥1,010	≥1,500
36	≥950	≥1,400
15	≥810	≥1,200
4	≥670	≥1,010
1	≥520	≥790

[a]The percentile of the perfect score was 99.98 on the 2,400 scale and 99.93 on the 1,600 scale.

Clinical Uses

The SAT is the most widely used admission test among colleges and universities in the United States assessing cognitive skills required for academic success at tertiary level.

Other clinical uses are limited because of the duration of the test (length of time to complete) and the primary purpose of the SAT assessing knowledge of subjects necessary for college success and skills students learned in high school. Others, some also shorter in duration, are available to include in a test battery for assessing cognitive impairment (e.g., WRAT-4).

However, the SAT has been used occasionally by organizations to test the exceptional abilities of younger children (e.g., less than 13 years of age) in order to provide adequate mentoring programs.

Research by Fuller et al. (2002) also suggests the presence of premorbid cognitive impairment in patients with schizophrenia using data obtained from scholastic test results. Although early test scores show no significant differences, test scores drop significantly between grades 8 and 11 corresponding with poor or declining scholastic performance and may be a precursor to cognitive impairment observed during initial episodes of schizophrenia.

Cross References

- ▶ Advanced Progressive Matrices
- ▶ Auditory Verbal Learning
- ▶ Basic Achievement Skills Inventory
- ▶ Full Scale IQ
- ▶ Kaufman Brief Intelligence Test
- ▶ Otis-Lennon School Ability Test
- ▶ Peabody Individual Achievement Test, Revised
- ▶ Tests of General Educational Development
- ▶ Wechsler Adult Intelligence Scale (All Versions)
- ▶ Wechsler Individual Achievement Test
- ▶ Wechsler Test of Adult Reading
- ▶ WPPSI-III

References and Readings

"About the College Board" – The College Board's webpage provides extensive information regarding the SAT and is available at: http://www.colegeboard.com/about/index/html

"About ACT, Inc." is available at: http://www.act.org/aboutact/history.html

"About the Advanced Placement Program" is available at: http://apcentral.collegeboard.com/apc/Controller.jpf

Bellar, M. (2009). *Translating, equating and validating scholastic aptitude tests: The Israeli case.* Retrieved on June 15, 2009, from http://www.unifr.ch/ztd/ems/berichte/b2/translating.htm

Coyle, T. R., & Pillow, D. R. (2008). SAT and ACT predict college GPA after removing g. *Intelligence, 36*(6), 719–729.

Frey, M. C., & Detterman, D. K. (2003). Scholastic assessment or *g*? The relationship between the scholastic assessment test and general cognitive ability. *Psychological Science, 15*(6), 373–378.

Fuller, R., Nopoulos, P., Arndt, S., O'Leary, D., Ho, B., & Andreasen, N. C. (2002). Longitudinal assessment of premorbid cognitive functioning in patients with schizophrenia through examination of standardised scholastic test performance. *American Journal of Psychiatry, 159*, 1183–1189.

Hubin, D. R. (1988). The scholastic aptitude test: Its development and introduction, 1900–1948. Ph.D. Dissertation in American History at the University of Oregon. Available for download at http://www.uoregon.edu/~hubin/

Jackson, D. N., & Rushton, J. P. (2006). Males have greater *g*: Sex differences in general mental ability from 100,000 17- to 18-year-olds on the scholastic assessment test. *Intelligence, 34*(5), 479–486.

Klopfenstein, K., & Thomas, M. K. (2009) *The Link Between College Success, Advanced Placement Experience and College Success.* Unpublished paper, retrieved June 4, 2009 from http://www.utdallas.edu/research/tsp/pdfpapers/newpaper1b.pdf

Korbin, L. (2006). *SAT Program Handbook. A Comprehensive Guide to the SAT Program for School Counselors and Admissions Officers*, 1, 33+. Retrieved June 4, 2009, from College Board Preparation Database.

Martz, G., Magloire, K., & Silver, T. (2007). *Cracking The ACT*. The Princeton Review, ISBN 9780375765858

Mau, W-C., & Lynn, R. (2001). Gender differences on the scholastic aptitude test, the american college test and college grades. *Educational Psychology, 21*(2), 133–136.

The College Board's *Advanced Placement Report to the Nation* is available at: www.collegeboard.com/prod_downloads/about/news_info/ap/2005/ap-report-nation.pdf

Wedman, I. (1994). The Swedish scholastic aptitude test: Development, use, and research. *Educational Measurement: Issues and Practice, 13*(2), 5–11.

Schwannoma

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Definition

Typically occurring in peripheral or cranial nerves, schwannomas are tumors that arise from the Schwann cells that produce the myelin sheath around nerve cells. A schwannoma can displace or damage neurons if there is associated pressure on the neuron resulting from pressure on some other structure (e.g., bone). These types of tumors are generally benign and slow growing. They may occur in the context of neurofibromatosis, Type II.

SCL-90-R

► Symptom Checklist-90-Revised

Scorer Reliability

► Inter-rater Reliability

Scotoma

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Synonyms

Scotomata (plural)

Definition

A scotoma is a small blind spot within the visual field. This "island" of impaired vision can be detected by confrontation testing, perimetry, and scanning laser ophthalmoscope (Cheung & Legge, 2005). Causes of scotoma include age-related macular degeneration, retinal infarcts or hemorrhage, demyelinating disorders, migraine, infection, and nutritional deficiency. The position and shape of the scotoma, and whether it is monocular or binocular, helps localize the causative abnormality in the visual pathways (Blumenfeld, 2002). Various types of scotomata have been described, including central, scintillating, fortification, and altitudinal scotoma (Blumenfeld, 2002). While scotomata often go undetected due to nystagmus and spontaneous "filling in" by the visual system, they can interfere with daily activities such as reading and driving.

Cross References

► Homonymous Quadrantanopsia
► Visual Field Deficit

References and Readings

Blumenfeld, H. (2002). *Neuroanatomy through clinical cases*. Sunderland, MA: Sinauer Associates.

Cheung, S. H., & Legge, G. E. (2005). Functional and cortical adaptations to central vision loss. *Visual Neuroscience, 22*(2), 187–201.

Scotomata (plural)

► Scotoma

Screening Measures

► Neuropsychological Screening Examination

SCWT

► Stroop Color Word Test (adult)

SDH

► Subdural Hematoma

SDS

► Zung Self-Rating Depression Scale

Searchlight Hypothesis

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Synonyms

Feature integration theory; Spotlight hypothesis

Definition

The phenomena of attentional selection of one visual area at a time within a greater visual space for the purpose of identifying a target among distractors. The analogy is to a searchlight illuminating an area at dusk such that the overall landscape is not entirely dark, but that the area within the searchlight is intensified.

Current Knowledge

The searchlight hypothesis coined by Crick (1984) is an extension of the feature integration theory of Treisman and her colleagues (1982) that suggests that attention selects one area at a time within a "master map" of locations. The theory was motivated by studies documenting different detection rates for visual targets presented in a distractor array dissimilar from the target. For example, a black "A" presented within a randomly arranged set of green "X"s and brown "T"s will "pop out" at the viewer. In contrast, a green "T" among the same distractor set will take much more time. The key element is that it requires the viewer to recognize the conjunction of color and shape. Further, this process tends to increase reaction time linearly, as if the mind, to use the metaphor, was using a searchlight that moved from one visual item to the next. Crick proposed to extend this searchlight theory to incorporate a potential brain mechanism. He proposed that: (1) the searchlight is controlled by the reticular complex of the thalamus; (2) the expression of the searchlight is the production of rapid firing in a subset of active thalamic neurons; (3) the conjunctions produced by the searchlight are mediated by Malsburg synapses, especially by rapid bursts acting on them; and (4) conjunctions are expressed by cell assemblies, especially assemblies of cells in different cortical regions.

Crick's hypothesized road map for a possible brain mechanism underlying the attentional searchlight has motivated neuroscience research aimed to demonstrate his ideas. McAlonan, Cavanaugh, and Wurtz (2006) found support for Crick's theory in their study of the lateral geniculate nucleus and the thalamic reticular nucleus of awake monkeys attending to visual and auditory information. They found that TRN activity was modified by shifts of visual attention, such that relevant sensory input was selected for additional processing at the expense of irrelevant input.

Cross References

► Thalamus

References and Readings

Crick, F. (1984). Function of the thalamic reticular complex: The searchlight hypothesis. *Proceedings of the National Academy of Sciences, 81*, 4586–4590.

McAlonan, K., Cavanaugh, J., & Wurtz, R. H. (2006) Attentional modulation of thalamic reticular neurons. *Journal of Neuroscience, 26*, 4444–4450.

Treisman, A. M. (1982). Perceptual grouping and attention in visual search for features and for objects. *Journal of Experimental Psychology: Human Perception and Performance, 8*, 194–214.

Seashore Rhythm Test

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Synonyms

Rhythm test; SRT

Description

The Seashore Rhythm Test (SRT) requires examinees to discriminate between like and unlike pairs of musical beats. Examinees are required to differentiate between 30 pairs of rhythmic beats. The beats or stimuli are presented to examinees from a tape recorder. After listening to one pair at a time, examinees write S on their answer sheets if they think the two beats sounded the same or D if they think the beats were different.

Historical Background

Originally, as a subtest of the Seashore Test of Musical Talent, Ward Halstead integrated the SRT into his test battery in 1947. The test was originally thought to be sensitive to impairment in the right hemisphere; however, most studies found no difference between patients with lesions of the right or the left cerebral hemisphere (Hom & Reitan, 1990; Reitan & Wolfson, 1989).

Psychometric Data

The raw error score is translated into a rank score as shown in a table in the manual. If an examinee has difficulty controlling a pencil, the examiner may write the responses when spoken by the examinee. If examinees get ahead of, or behind, the tape, the examiner is not allowed to correct or help them; the standard procedure requires that examinees keep pace with the speed of the tape without any assistance.

Clinical Uses

The SRT has been found to be most useful as a measure of attention and concentration, as persons with severe brain injuries usually perform significantly below the levels of normal control subjects, and patients with bilateral and diffuse lesions tend to make more errors than those with lateralized lesions (Reitan & Wolfson, 1989). Normal control subjects typically make between three and five errors (Bornstein, 1985; Reitan & Wolfson, 1989). The number of errors correlates positively with the severity of the traumatic brain injury (Hom & Reitan, 1990). No sex differences have been found; however, a musical background can have a positive effect on scores. Because cognitively impaired patients with musical training can achieve scores in the normal range, their scores must be interpreted with caution.

Cross References

► Halstead–Reitan Neuropsychological Test Battery

References and Readings

Bornstein, R. A. (1985). Normative data on selected neuropsychological measures form a nonclinical sample. *Journal of Clinical Psychology, 41*, 651–659.

Hom, J., & Reitan, R. M. (1990). Generalized cognitive function after stroke. *Journal of Clinical and Experimental Neuropsychology, 12*, 644–654.

Reitan, R. M., & Wolfson, D. (1989). The seashore rhythm test and brain functions. *The Clinical Neuropsychologist, 3*, 70–78.

Second Impact Syndrome

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Short Description

Second Impact Syndrome (SIS) has been defined as the rapid cerebral edema and herniation after a second impact on the head, before the recovery from the initial blow. (Erlanger, Kutner, Barth & Barnes, 1999).

Categorization

SIS is characterized as a very rare, preventable condition that has been exhibited mostly in younger adults and adolescents, owing to the fragility of younger people's developing brains (Bowen, 2003).

Epidemiology

SIS has been primarily observed in younger people, particularly athletes, under the age of 20 years. It has been reported that those who have had any prior history of concussion have a higher chance of experiencing SIS if a second concussion occurs (Bowen, 2003). Every documented case has been of males; however, this may be due to higher male participation in more physical activities such as contact sports.

Natural History, Prognostic Factors, Outcomes

SIS is due to a second impact to the brain, chest, side, or an impact that snaps the head during the recovery period after the initial impact. The vulnerable period (e.g., minutes to weeks after initial concussion or impact) before the second impact is characterized by a decrease in cerebral blood flow and an increase in glucose (e.g., energy demand) intake, which leaves the neurovascular system incapable of fully meeting the increasing energy strains (Cobb & Battin, 2004). SIS can be prevented by allowing the cerebral cortex to fully recover before partaking in any activity that could further damage the brain. In addition, protective head gear would greatly reduce the risk of concussions and SIS (Cobb & Battin, 2004). It is often fatal due to the cerebral edema.

Neuropsychology of Second Impact Syndrome

SIS's neurophysiology has been found to be associated with increased intracranial pressure and diffuse cerebral swelling, causing herniation within the temporal lobes and eventually spreading through the cortex. Mortality has been closely related to full brain stem failure, which can occur within 2–5 min of the second impact (Proctor and Cantu, 2000).

Evaluation

SIS has been diagnosed, largely, in young athletes who have experienced a substantial concussion, and then experience a minor impact on the brain during the recovery period of the initial blow. After the second impact, usually 15 seconds to minutes after impact, the individual will collapse, lose consciousness, experience rapid eye dilation, respiratory difficulty, and lack of eye movement (Bowen, 2003). Diagnosis is performed by Computed Topography (CAT scan) and Magnetic Resonance Imaging (MRI).

Treatment

Recognizing the exhibition of SIS is the most important factor for treating SIS (Bowen, 2003). Because of the diffuse cerebral swelling that has been closely related to SIS, it is important for the SIS patients to hyperventilate to decrease intracranial pressure; in addition, administering osmotic agents has also been implemented in the treatment of SIS. However, in 50% of patients recovery or survival is not likely, and the 50% of SIS patients who do survive SIS are often permanently disabled.

Cross References

- ▶ Cerebral Edema
- ▶ Concussion

References and Readings

Bowen, A. P. (2003). Second impact syndrome: A rare catastrophic preventable complication of concussion in young athletes. *Journal of Emergency Nursing, 29*(3), 287–289.

Cobb, S., & Battin, B. (2004). Second-impact syndrome. *The Journal of School Nursing, 20*(5), 262–267.

Erlanger, D. M., Kutner, K. C., Barth, J. T., & Barnes, R. (1999). Neuropsychology of sports-related injury: Dementia pugilistica to post concussion syndrome. *The Clinical Neuropsychologist, 13*(2), 193–209.

Proctor, M. R., & Cantu, R. C. (2000). Head and neck injuries in young athletes. *Clinics in Sports Medicine, 19*(4), 693–715.

Secondary Association Cortex

- ▶ Unimodal Cortex

Secondary Brain Injury

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Synonyms

Multiple injuries

Definition

Secondary brain injury occurs after the primary mechanisms of injury have run their course (Gennarelli & Graham, 2005). For example, after a person suffers a traumatic brain injury from a motor vehicle accident, the brain may sustain further, a secondary damage that is not directly caused by the impact of the accident.

Current Knowledge

Secondary brain injuries often result from complications of the primary mechanism of injury and may occur anywhere from hours to days after the initial injury (Dawodu, 2007). Causes of secondary brain injury can include changes in cellular and chemical functions in the brain and/or damage to blood vessels and brain tissue (University of Virginia Health System, 2004). Research suggests that the initial injury can trigger a cascade of adverse events at the biochemical and microcirculatory levels within the brain resulting in decreased cerebral blood flow and oxygen supply. The occurrence of secondary insults has been shown to significantly increase morbidity and mortality (Gennarelli & Graham, 2005). However, recent advances in early medical interventions following the primary brain injury have helped to reduce the incidence of secondary brain injuries (Gennarelli & Graham, 2005).

Types

Hypoxemia – An abnormal deficiency in the concentration of oxygen in the arterial blood. This term is frequently confused with hypoxia. It is possible to have a low oxygen content (e.g., due to anemia) but a high PO_2 in the arterial blood.

Hypoxia – Results in decreased oxygen flow to brain tissue. Brain cells begin to die after several minutes without adequate oxygen supply. Hypoxia can result from effects of the primary brain injury or may be related to respiratory complications such as obstructed airway or physical injury to the lungs or chest cavity. Furthermore, transient or prolonged periods of apnea (absence of breathing) can occur after a concussive brain injury.

Hypotension – Occurs when blood pressure is reduced to $\leq$ 90/60 mmHg. As a result of the primary injury to the brain, dysregulation of autonomic processes such as control of blood pressure, heart rate, and respiration can occur (Cifu & Drake, 2006). In turn, blood pressure may drop leading to decreased oxygen supply to brain tissue.

Ischemia – Results in decreased cerebral blood flow. Ischemia can result from hypotension, damage to blood vessels, and/or increased intracranial pressure.

Swelling – Results from an increase in blood volume within the brain or an increase in water within brain tissue (Gennarelli & Graham, 2005). Swelling can lead to raised intracranial pressure which if left untreated can then cause shifting of brain tissue and compression of the brainstem.

Cross References

- ▶ Edema
- ▶ Head Injury
- ▶ Ischemia
- ▶ Loss of Consciousness
- ▶ Mild Traumatic Brain Injury
- ▶ Traumatic Brain Injury (TBI)

References and Readings

Cifu, D., & Drake, D. F. (2006). Repetitive head injury syndrome. Retrieved January 27, 2008 from http://www.emedicine.com/sports/TOPIC113.HTM

Dawodu, S. T. (2007). Traumatic brain injury: Definition, epidemiology, pathophysiology. Retrieved January 26, 2008 from http://www.emedicine.com/pmr/topic212.htm#section~pathophysiology:_secondary_injury

Dixon, T. M., & Layton, B. S. (1999). Traumatic brain injury. In M. G. Eisenberg, R. L. Glueckauf, & H. H. Zaretsky (Eds.), *Medical aspects of disability: A handbook for the rehabilitation professional* (pp.98–120). New York: Springer.

Gennarelli, T. A., & Graham, D. I. (2005). Neuropathology. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Traumatic brain injury* (pp. 27–50). Arlington, VA: American Psychiatric Publishing, Inc.

Hospital University Science Malaysia. (2000). Head injury. Retrieved January 27, 2008 from http://members.tripod.com/neurousm/education/headinjury.PDF

Medline Plus. (2006). Cerebral hypoxia. Retrieved January 26, 2008 from http://www.nlm.nih.gov/medlineplus/ency/article/001435.htm

National Institute of Neurological Disorders and Stroke. (2002). Traumatic brain injury: Hope through research. Retrieved January 26, 2008 from http://www.ninds.nih.gov/disorders/tbi/detail_tbi.htm

Singh, J., & Stock, A. (2006). Head trauma. Retrieved January 27, 2008 from http://www.emedicine.com/ped/topic929.htm

University of Virginia Health System. (2004). Acquired brain injury. Retrieved January 26, 2008 from http://www.healthsystem.virginia.edu/uvahealth/adult_pmr/braininj.cfm

Secondary Cortex

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Synonyms

Modality-specific homotypical cortex; Unimodal association cortex

Definition

Term primarily associated with Aleksandr Luria to designate the portion of the isocortex that is interposed between the primary and tertiary cortices. For most practical purposes, it is comparable to the unimodal association cortex, as defined by Mesulam.

Cross References

► Unimodal Cortex

References and Readings

Luria, A. R. (1966). *Higher cortical functions in man.* New York: Basic Books.

Mesulam, M-M (2000). Large-scale networks, association cortex, frontal syndromes, the limbic system, and hemispheric specialization. In M-M. Mesulam (Ed.), *Principles of behavioral and cognitive neurology* (pp. 1–120). New York: Oxford University Press.

Secondary Handicapping Condition

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Definition

The terms "primary" handicapping condition and "secondary" handicapping condition are commonly used in clinical and school settings to describe individuals who have been found to have one or more educational handicaps. If children have one handicapping condition (e.g., a writing learning disability), they are seen as having a primary handicapping condition. If they have more than one condition (such as a writing learning disability and a speech impairment), they are viewed as having a primary and a secondary handicapping condition. It is important to differentiate between the terms "disability" and "handicap" to best understand the current use of these terms.

Current Knowledge

These terms have changed throughout recent history. When the All Handicapped Children Act of 1975 (also known as P.L. 94-142) was passed, the terminology used was "handicap." When the law was revised later as the Individuals with Disabilities Education Act (IDEA) and most recently as the Individuals with Disabilities Education Act 2004, the term "disability" was used. This change in terminology recognizes that those with disabilities want to communicate to others that having a disability doesn't always limit what they are able to achieve. For example, an individual with a reading disability may be able to learn using modified educational materials such as a computer that projects letters into large type for the individual. Thus, the individual may need specialized educational modifications or differential treatment, but they still can comprehend the material that needs to be learned.

Deno (2002, p. 42) explains the difference as, "Handicaps are conditions that exist when successful functioning in an environment requires a level of ability beyond that possessed by the person." For example, a woman in a wheelchair would have a "handicap" when she must go to the second floor of a two-story building that only has stairs and not an elevator. A student's disability can become a handicap when the individual must use skills that they do not possess. For example, a student may read at a 6th-grade level so they would not be able to read a 10th-grade novel. In contrast, when enrolled in a 10th-grade ceramics class, the student may never be asked to use more advanced reading skills and no modifications would be necessary.

These terms are frequently used in educational settings by personnel to describe a child's functioning and later create appropriate educational programs. However, it is important to note that the terminology in the field frequently changes and different states may use different terms to describe a disability.

References and Readings

Deno, S. (2002). Problem solving as best practice. In A. Thomas & J. Grimes (Eds.), *Best practices in school psychology IV* (pp. 37–55). Bethesda, MD: NASP.

Secondary Hypothyroidism

► Hypothyroidism

Secondary Visual Cortex

► Extrastriate

Secondary-Progressive Multiple Sclerosis

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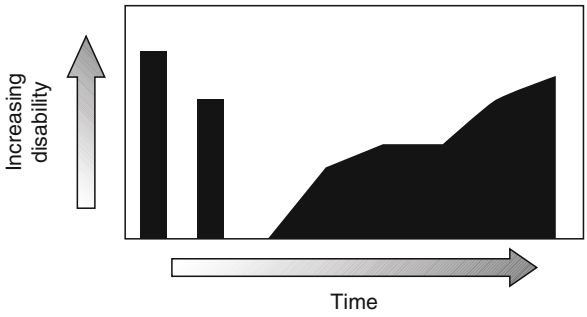
Synonyms

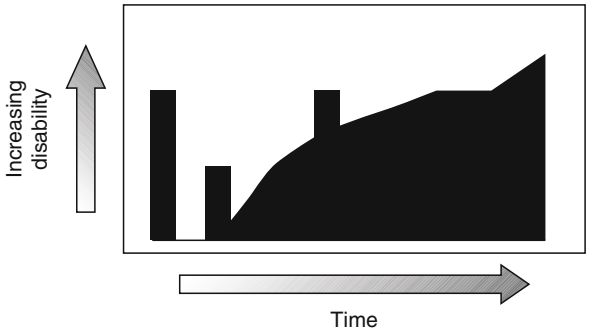
Advanced MS

Short Description or Definition

Secondary-progressive multiple sclerosis (SPMS) describes a more advanced disease stage of individuals who initially experience a relapsing–remitting course (RRMS). Typically, there are no or few discrete episodes of neurological dysfunction (i.e., relapses), and there is continued accumulation of neurological deficits resulting in increasing disability (see Figs. 1 and 2). The transition from a relapsing–remitting course to secondary progression can be subtle initially, and thus is often diagnosed in retrospect following a 6–12-month period of sustained progression. To date, no clear biological markers have been found to indicate the point at which the course of MS changes.



Secondary-Progressive Multiple Sclerosis. Figure 1
Secondary-progressive multiple sclerosis. *Note*: Disease course starts as relapsing–remitting but over time relapses no longer occur and there is a continuous worsening of symptoms and accumulation of disability. (Adapted from Lublin & Reingold (1996). Used with permission of Lippincott Williams & Wilkins.)



Secondary-Progressive Multiple Sclerosis. Figure 2
Secondary-progressive multiple sclerosis with relapses. *Note*: Relapses are superimposed upon a pattern of continuous worsening of neurological symptoms and increasing levels of disability. (Adapted from Lublin & Reingold (1996). Used with permission of Lippincott Williams & Wilkins.)

Natural History, Prognostic Factors, Outcomes

The course of multiple sclerosis is largely defined by relapses (time-limited episodes of dysfunction) and progression (worsening of symptoms over a period of at least 6 months). The former is believed to be the result of acute, focal inflammatory activity in the central nervous system, and the latter related to chronic diffuse axonal loss (Confavreux & Compston, 2005). Before the era of

immunomodulatory drugs, natural history studies have shown that half of those with an initial RRMS course develop secondary progression after approximately 10 years and that 90% show progression by 20 years after diagnosis (Weinshenker et al., 1989). This progression is most often marked by changes in ambulation, but as noted above, the onset of secondary progression can be difficult to pinpoint and is often assigned retrospectively. The strongest predictor of conversion to secondary progression is age at disease onset – the older a person is at diagnosis, the shorter the time to progression (Rovaris, Confavreux, Furlan, Kappos, Comi, & Filippi, 2006). It has also been suggested that male sex is associated with a shorter time to progression (Tremlett, Zhao, & Devonshire, 2008). Other factors that appear predictive of a greater risk for earlier progression include an incomplete recovery from the first MS attack/relapse, a shorter interval to the second attack, and a higher number of relapses early in the disease course (Rovaris et al., 2006). However, these factors are not predictive of the rate of subsequent progression once the threshold of secondary progression has been crossed. In other words, infrequent relapses early in the disease course may suggest a longer period until the onset of significant and irreversible neurological deficits, but once that threshold is reached, the rate of further progression is similar across most individuals with MS (Confavreux & Compston, 2005).

Although there are no generally accepted biological markers to indicate a shift from the RR to SP course, neuroimaging and pathology studies have highlighted important differences between the two phases of the disease. SPMS patients show fewer contrast-enhancing lesions on MRI (Wolinsky et al., 2000) and higher T1 lesion volume loads (thought to reflect axonal loss) than those with RRMS (van Walderveen et al., 2001). MR spectroscopy has allowed for the *in vivo* examination of cortical gray matter lesions, and there is evidence of a decrease in a marker of neuronal health (N-acetylaspartate, NAA) in SPMS relative to RRMS (Caramanos, DiMaio, Narayanan, Lapierre, & Arnold, 2009). Further, application of advanced MRI techniques has shown that individuals with SPMS accumulate more cortical lesions over time than those with RRMS and that this increase is associated with a decline in cognition (Roosendaal et al., 2009). Comparison of autopsy tissue suggests that RRMS is characterized by focal inflammatory demyelinating white matter lesions, whereas the SP course is marked by diffuse axonal injury, cortical demyelination, and diffuse inflammation (Kutzelnigg et al., 2005). Examination of plaques from SPMS has indicated that ongoing myelin breakdown occurs in the absence of histological signs of acute inflammation (Prineas et al., 2001). Taken together, these studies suggest that the transition from RRMS to SPMS represents the progression from the inflammatory phase to the neurodegenerative phase of the disease and that neurons not initially injured by demyelination may be affected later due to loss of input from the damaged neurons (Smith, McDonald, Miller, & Lassman, 2005).

Neuropsychology and Psychology of Secondary-Progressive MS

Studies investigating cognitive functioning in MS have generally found reduced or impaired performance relative to age-matched controls in new learning and recall, attention, processing speed, and executive functioning (Chiaravalloti & DeLuca, 2008). Individuals with a progressive course typically do not perform as well as those with a relapsing–remitting course, but there have been less consistent findings regarding cognitive abilities when individuals with SPMS and primary progressive MS (PPMS) are compared. This may be due to small sample sizes, variations in assessment instruments, or difficulty equating the groups in regards to disease characteristics. Findings have included minimal differences between the two progressive types (De Sonneville, Boringa, Reuling, Lazeron, Adèr, & Polman, 2002; Foong, Rozewicz, Chong, Thompson, Miller, & Ron, 2000; Kraus, Schütze, Brokate, Kröger, Schwendemann, & Hildebrandt, 2005; Ukkonen, Vahvelainen, Hamalainen, Dastidar, & Elvorra, 2009): relatively worse performance by SPMS patients (Comi et al., 1995; Huijbregts, Kalkers, de Sonneville, de Groot, Reuling, & Polman, 2004) and relatively better performance by SPMS patients (Wachowius, Talley, Silver, Heinze, & Sailer, 2005). Studies that have focused on specific cognitive processes rather than overall performance have shown that those with SPMS are worse than PPMS in acquisition of new material but better than PPMS at subsequent recall (Gaudino, Chiaravalloti, DeLuca, & Diamond, 2001). Additionally, although both SPMS and PPMS subjects showed intact ability but slowed processing on measures of executive functioning, this effect was more pronounced for those with SPMS (Denney, Sworowski, & Lynch, 2005).

The transition from RR to SP can represent a significant psychological challenge as the visible signs of MS become more obvious. Many individuals initially resist using aids for ambulation as they perceive this as "giving in" to the disease. Some interpret the transition to SPMS as representing failure of personal power to "fight" MS or of immunomodulatory treatment. Some individuals

may feel guilty for not initiating treatment sooner or for not remaining compliant with treatment while in the RR phase (Kalb, 2007). While disease severity (i.e., disability) is inversely correlated with quality of life ratings in MS (Beiske et al., 2007; Henricksson, Fredrikson, Masterman, & Jönsson, 2001), when the progressive courses are compared, those with SPMS score worse than PPMS on measures of psychological functioning including depression and anxiety (Vleugels et al., 1998). It is not clear if this is due to the challenge of coping with a change in disease course for individuals with SPMS. It should also be noted that direct and indirect care costs go up with increasing disability levels (Henricksson et al., 2001), and this can represent another source of significant psychosocial stress in SPMS.

Evaluation

Neurologists often use the Expanded Disability Status Scale (EDSS, Kurtzke, 1983) to quantify impairment in neurological functioning and overall level of disability. It provides a metric to track disease-related change over time and to classify people with MS by disability level. As such it has been widely used in clinical trials and in natural history studies. The EDSS ranges from 0 (normal neurological exam) to 10 (death due to MS) and is based on an evaluation of eight functional systems: pyramidal, cerebellar, brain stem, sensory, bowel and bladder, visual, cerebral, and "other." A score is assigned for the type of impairment and the degree to which the patient is aware of or impacted by it within each functional system. The EDSS rating is then based on the number of functional systems impaired and the severity of dysfunction in each. For example, an EDSS score of 2 indicates "minimal disability" in one functional system with no or very minor changes in the other systems.

There is no particular EDSS score that marks the transition from RRMS to SPMS. In RRMS, a post-relapse EDSS score might be higher than the pre-relapse score depending on how completely the relapse symptoms resolve. Thus, there will be individual variability in EDSS score at entry into secondary progression depending on how much disability was previously accumulated. Despite this variability, natural history studies often use particular EDSS benchmarks to chart progression or compare MS subtypes. The most common include time to progression to an EDSS score of 4 (severe disability in at least one functional system but able to walk without assistance at least 500 m without rest), 6 (need for unilateral support for walking and unable to walk more than 100 m without rest), and 7 (unable to walk for more than 10 m without assistance or rest; essentially wheelchair-dependent).

Although the EDSS is a widely used rating system, it has limitations (Hobart, Freeman, & Thompson, 2000; Noseworthy, 1994). First it can be somewhat cumbersome to use in regular clinical practice due to the time needed to systematically evaluate each system and assign a score. Second, inter-rater reliability can be an issue in multicenter studies. In terms of psychometric properties, the EDSS is a rank-order scale, not an interval or ratio scale. In essence, one cannot say that someone with an EDSS of 4 is twice as disabled as someone with an EDSS of 2, and nonparametric statistics must be used in the analysis of EDSS scores. Also, it has been suggested that the scale shows more sensitivity to subtle change at the lower end of the scale than at the higher end. Another limitation of the EDSS is that it does not fully assess and give proper weight to MS-related cognitive dysfunction. To that end, an alternate rating scale – the MS Functional Composite (MSFC) – was proposed as an outcome measure for clinical trials (Cutter et al., 1999). The MSFC assesses ambulation through a timed 25-foot walk, arm function with a timed 9-hole peg placement test, and cognition with the Paced Auditory Serial Addition Test. The scores for each component of the test are converted into a z-score (standardized to an MS population) and averaged to yield a composite score. As reviewed by Rudick, Cutter, and Reingold (2002), the MSFC captures change in multiple systems and shows moderate correlation with the EDSS. Additionally, the MSFC correlates better with MRI measures than the EDSS and has strong correlations with patient-reported quality of life indices. Further, the MSFC is brief, easy to administer, and more objective than EDSS ratings.

Treatment

As described above, it is believed that the transition to SPMS represents a shift in the processes that produce neurological dysfunction. This has been supported by studies that have shown the treatments approved for relapsing forms of MS are not as effective once an individual has started to show secondary progression (Giovannoni, 2004). One immunomodulatory treatment – Betaseron (interferon beta-1b) has been FDA approved for individuals with secondary-progressive MS who continue to experience relapses (see Fig. 2). While a European trial showed a significant treatment effect of delayed disability progression, in a North American trial there was no significant difference in time to confirmed progression in

EDSS score between SPMS patients on drug or placebo. However, those on treatment showed a reduced relapse rate and positive MRI changes including fewer new lesions and less accumulated burden of disease on T2-weighted scans (Panitch et al., 2004). Immunosuppressive therapy (i.e., mitoxantrone) has shown to be effective in reducing relapse rate and disability progression for rapidly deteriorating secondary-progressive MS (Perini, Calabrese, Tiberio, Ranzato, Battistin, & Gallo, 2006). Because mitoxantrone can have cardiotoxic effects, there are limits on the duration of treatment and cumulative dose. Although the efficacy of disease-modifying treatment may be limited in SPMS, symptomatic treatment for fatigue, pain, spasticity, bowel/bladder dysfunction, and mood disorders remain important components of MS care.

Cross References

- ▶ Demyelination
- ▶ Multiple Sclerosis
- ▶ Primary Progressive Multiple Sclerosis
- ▶ Relapsing–Remitting Multiple Sclerosis

References and Readings

Chiaravalloti, N. D., & DeLuca, J. (2008). Cognitive impairment in multiple sclerosis. *Lancet Neurology, 7*, 1139–1151.

Confavreux, C., & Compston, A. (2005). The natural history of multiple sclerosis. In A. Compston et al., (Eds.), *McAlpine's multiple sclerosis.* (4th ed.). Philadelphia, PA: Elsevier, Inc.

Giovannoni, G. (2004). Management of secondary-progressive multiple sclerosis. *CNS Drugs, 18*, 653–669.

Kalb, R. (2007). The emotional and psychosocial impact of multiple sclerosis relapses. *Journal of the Neurological Sciences, 256*, S29–S33.

Kurtzke, J. F. (1983). Rating neurological impairment in multiple sclerosis: An expanded disability status scale (EDSS). *Neurology, 33*, 1444–1452.

Lublin, F. D., & Reingold, S. C. for the National Multiple Sclerosis Society (USA) Advisory Committee on Clinical Trials of New Agents in Multiple Sclerosis (1996). Defining the clinical course of multiple sclerosis: Results of an international survey. *Neurology, 46*, 907–911.

Rovaris, M., Confavreux, C., Furlan, R., Kappos, L., Comi, G., & Filippi, M. (2006). Secondary progressive multiple sclerosis: Current knowledge and future challenges. *Lancet Neurology, 5*, 343–354.

Rudick, R. A., Cutter, G., & Reingold, S. (2002). The multiple sclerosis functional composite: A new clinical outcome measure for multiple sclerosis trials. *Multiple Sclerosis, 8*, 359–365.

Smith, K., McDonald, I., Miller, D., & Lassman, H. (2005). The pathophysiology of multiple sclerosis. In A. Compston et al., (Eds.), *McAlpine's multiple sclerosis.* (4th ed.). Philadelphia, PA: Elsevier, Inc.

Tremlett, H., Zhao, Y., & Devonshire, V. (2008). Natural history of secondary progressive multiple sclerosis (2008). *Multiple Sclerosis, 14*, 314–324.

Weinshenker, B. G., Bass, B., Rice, G. P., Noseworthy, J., Carriere, W., Baskerville, J., et al. (1989). The natural history of multiple sclerosis: A geographically based study. I. Clinical course and disability. *Brain, 112*, 133–146.

Additional References

Beiske, A. G., Naess, H., Aarseth, J. H., Andersen, O., Elovaara, I., Farkkila, M., et al. for the Nordic SPMS Study Group (2007). *Multiple Sclerosis, 13*, 386–392.

Caramanos, Z., DiMaio, S., Narayanan, S., Lapierre, Y., & Arnold, D. L. (2009). 1H-MRSI evidence for cortical gray matter pathology that is independent of cerebral white matter lesion load in patients with secondary progressive multiple sclerosis. *Journal of the Neurological Sciences, 282*, 72–79.

Comi, G., Filippi, M., Martinelli, V., Campi, A., Rodegher, M., Alberoni, M., et al. (1995). Brain MRI correlates of cognitive impairment in primary and secondary progressive multiple sclerosis. *Journal of the Neurological Sciences, 132*, 222–227.

Cutter, G. R., Baier, M. L., Rudick, R. A., Cookfair, D. L., Fischer, J. S., Petkau, J., et al. (1999). Development of a multiple sclerosis functional composite as a clinical trial outcome measure. *Brain, 122*, 871–882.

Denney, D. R., Sworowski, L. A., & Lynch, S. G. (2005). Cognitive impairment in subtypes of multiple sclerosis. *Archives of Clinical Neuropsychology, 20*, 967–981.

De Sonneville, L. M. J., Boringa, J. B., Reuling, I. E. W., Lazeron, R. H. C., Adèr, H. J., & Polman, C. H. (2002). Information processing characteristics in subtypes of multiple sclerosis. *Neuropsychologia, 40*, 1751–1765.

Foong, J., Rozewicz, L., Chong, W. K., Thompson, A. J., Miller, D. H., & Ron, M. A. (2000). *Journal of Neurology, 247*, 97–101.

Gaudino, E. A., Chiaravalloti, N. D., DeLuca, J., & Diamond, B. J. (2001). A comparison of memory performance in relapsing-remitting, primary progressive, and secondary progressive, multiple sclerosis. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 14*, 32–44.

Henricksson, F., Fredrikson, S., Masterman, T., & Jönsson, B. (2001). Costs, quality of life and disease severity in multiple sclerosis: A cross-sectional study in Sweden. *European Journal of Neurology, 8*, 27–35.

Hobart, J., Freeman, J., & Thompson, A. (2000). Kurtzke scales revisited: The application of psychometric methods to clinical intuition. *Brain, 123*, 1027–1040.

Huijbregts, S. C. J., Kalkers, N. F., de Sonneville, L. M. J., de Groot, V., Reuling, I. E. W., & Polman, C. H. (2004). Differences in cognitive impairment of relapsing remitting, secondary, and primary progressive MS. *Neurology, 63*, 335–339.

Kutzelnigg, A., Lucchinetti, C. F., Stadelmann, C., Bruck, W., Rauschka, H., Bergmann, M., et al. (2005). Cortical demyelination and diffuse white matter injury in multiple sclerosis. *Brain, 128*, 2705–2712.

Kraus, J. A., Schütze, C., Brokate, B., Kröger, B., Schwendemann, G., & Hildebrandt, H. (2005). Discriminant analysis of the cognitive performance profile of MS patients differentiates their clinical course. *Journal of Neurology, 252*, 808–813.

Noseworthy, J. H. (1994). Clinical scoring methods for multiple sclerosis. *Annals of Neurology, 36*, S80–S85.

Panitch, H., et al.; The North American Study Group on Interferon beta-1b in Secondary Progressive MS (2004). Interferon beta-1b in secondary progressive MS: Results from a 3-year controlled study. *Neurology, 63*, 1788–1795.

Perini, P., Calabrese, M., Tiberio, M., Ranzato, F., Battistin, L., & Gallo, P. (2006). Mitoxantrone versus cyclophosphamide in secondary-progressive multiple sclerosis. *Journal of Neurology, 253*, 1034–1040.

Prineas, J. W., Kwon, E. E., Cho, E. S., Sharer, L. R., Barnett, M. H., Oleszak, E. L., et al. (2001). Immunopathology of secondary-progressive multiple sclerosis. *Annals of Neurology, 50*, 646–657.

Roosendaal, S. D., Moraal, B., Pouwels, P. J., Vrenken, H., Castelijns, J. A., Barkhof, F., et al. (2009). Accumulation of cortical lesions in MS: relation with cognitive impairment. *Multiple Sclerosis, 15*, 708–714.

Ukkonen, M., Vahvelainen, T., Hamalainen, P., Dastidar, P., & Elvorra, I. (2009). Cognitive dysfunction in primary progressive multiple sclerosis: A neuropsychological and MRI study. *Multiple Sclerosis, 15*, 1055–1061.

Van Walderveen, M. A. A., Lycklama a Nijeholt, G. J., Ader, H. J., Jongren, P. J. H., Polman, C. H., Castelijns, J. A., et al. (2001). Hypointense lesions on T1-weighted spin-echo magnetic resonance imaging: Relation to clinical characteristics in subgroups of patients with multiple sclerosis. *Archives of Neurology, 58*, 76–81.

Vleugels, L., Pfennings, L., Pouwer, F., Cohen, L., Ketelaer, P., Polman, C., et al. (1998). Psychological functioning in primary versus secondary progressive multiple sclerosis. *British Journal of Medical Psychology, 71*, 99–106.

Wachowius, U., Talley, M., Silver, N., Heinze, H., & Sailer, M. (2005). Cognitive impairment in primary and secondary progressive multiple sclerosis. *Journal of Clinical and Experimental Neuropsychology, 27*, 65–77.

Wolinsky, J. S., Narayana, P. A., Noseworthy, J. H., Lublin, F. D., Whitaker, J. N., Linde, A., et al.; The MRI Analysis Center of the University of Texas–Houston Health Science Center, & the North American Linomide Investigators (2000). Linomide in relapsing and secondary progressive MS: Part II: MRI results. *Neurology, 54*, 1734–1741.

Section 504 of the Rehabilitation Act of 1973

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Synonyms

Rehabilitation Act of 1973

Definition

Section 504 of the 1973 Rehabilitation Act or, more formally, the Rehabilitation Act of 1973 is American legislation that guarantees certain rights to people with disabilities. Section 504 is widely recognized as the first civil-rights statute for workers with disabilities. Section 504 of the Rehabilitation Act of 1973 is a national law that protects individuals from discrimination based on their disability. This section forbids organizations and employers from excluding or denying individuals with disabilities an equal opportunity to receive program benefits and services. It defines the rights of individuals with disabilities to participate in, and have access to, program benefits and services.

Section 504 protects individuals with disabilities. Under this law, individuals with disabilities are defined as persons with a physical or mental impairment that substantially limits one or more major life activities. People who have a history of, or who are regarded as having a physical or mental impairment that substantially limits one or more major life activities, are also covered. Major life activities include caring for one's self, walking, seeing, hearing, speaking, breathing, working, performing manual tasks, and learning. Some examples of impairments that may substantially limit major life activities, even with the help of medication or aids/devices, are AIDS, alcoholism, blindness or visual impairment, cancer, deafness or hearing impairment, diabetes, drug addiction, heart disease, and mental illness (http://www.hhs.gov/ocr/504.html).

Historical Background

Section 504 is widely recognized as the first civil-rights statute for persons with disabilities. It took effect in May 1977. Section 504 was somewhat controversial, because it afforded people with disabilities many rights similar to those for other minority groups in the Civil Rights Act of 1964. At this time, the rights afforded from this act extend to those, even in the public sector.

Current Knowledge

Section 504 of the 1973 Rehabilitation Act or, more formally, the Rehabilitation Act of 1973 is American legislation that guarantees certain rights to people with disabilities. Section 504 is widely recognized as the first civil-rights statute for workers with disabilities. Section 504 of the Rehabilitation Act of 1973 is a national law that protects individuals from discrimination based on their disability. Section 504 forbids organizations and employers from excluding or denying individuals with disabilities an equal opportunity to receive program benefits and services. It defines the rights of individuals with disabilities to participate in, and have access to, program benefits and services.

Future Directions

Section 504 protects individuals with disabilities. Under this law, individuals with disabilities are defined as persons with a physical or mental impairment that substantially limits one or more major life activities. People who have a history of, or who are regarded as having a physical or mental impairment that substantially limits one or more major life activities, are also covered. Major life activities include caring for one's self, walking, seeing, hearing, speaking, breathing, working, performing manual tasks, and learning. Some examples of impairments that may substantially limit major life activities, even with the help of medication or aids/devices, are AIDS, alcoholism, blindness or visual impairment, cancer, deafness or hearing impairment, diabetes, drug addiction, heart disease, and mental illness (http://www.hhs.gov/ocr/504.html).

Cross References

► Americans with Disabilities Act (ADA)

References and Readings

Boundy, K. (2006, January–February). Examining the 2004 amendments to the individuals with disabilities education act: What advocates for students and parents need to know. *Clearinghouse Review Journal of Poverty Law and Policy, 39*, 550–578.

Demers, S. T., & Nellis, L. (2006). Assessing eligibility for and appropriateness of special education services. In S. Sparts & G. Koocher (Eds.), *Forensic mental health assessment of children and adolescents.* New York: Oxford University Press.

Imber, S. C., & Radcliff, D. (2003). Independent educational evaluations under IDEA'97: It's a testy matter. *Exceptional Children, 70*, 27–44.

Wills, K. E. (2008). A pediatric neuropsychologist's lessons from "Independent Educational Evaluations": Respect parents, listen to teachers, do your homework, but think for yourself. In R. Heilbronner (Ed.), *Neuropsychology in the courtroom: Expert analysis of reports and testimony.* New York: Guilford.

Wills, K. E., & Sweet, J. J. (2006). Neuropsychological considerations in forensic child assessment. In S. Sparts & G. Koocher (Eds.), *Forensic mental health assessment of children and adolescents.* New York: Oxford University Press.

Secular IQ Gains

► Flynn Effect

Sedative Hypnotic Drugs

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Definition

Medications that are classified as sedative hypnotic medications are used clinically to cause sedation by the relief of anxiety or to promote sleep. The major classes of sedative hypnotic medications include barbiturates, benzodiazepines, and other drugs that do not fit into either of the other categories.

Current Knowledge

Mechanism of Action

The *benzodiazepines* are generally classified into three groups: short acting, intermediate acting, and long acting. The duration of action plays a major role in determining its clinical use. The molecular site of action for the drug is at the $GABA_A$ (gamma-aminobutyric acid) receptor in the central nervous system (CNS). An increase in the effect of GABA results in general suppression of the CNS when it binds with a benzodiazepine.

Regarding use, medications such as diazepam, clonazepam, and lorazepam have been useful in the treatment of generalized seizures. Some of the benzodiazepines cause anesthesia without loss of consciousness and are used during medical procedures like colonoscopy or endoscopy procedures. Midazolam is also used before general anesthesia.

The *barbiturates* may also be divided into groups based on their duration. Similar to the benzodiazepine, the barbiturate binds at a site on the $GABA_A$ receptor. At high doses, the barbiturate may act as a direct agonist at receptor and produce significant CNS depression.

The barbiturate is a nonselective CNS depressant. The indications can include sedation, anesthesia, and anticonvulsant actions. Barbiturates are used less frequently in health care today, as they have been replaced by much safer medications.

Other Sedative Hypnotics

These medications do not fit into either of the above categories. These medications also bind to the benzodiazepine receptor at the $GABA_A$ receptor, but their chemical

Sedative Hypnotic Drugs. Table 1 Common sedative hypnotic medications listed by major class

Benzodiazepines	Barbiturates	NBRA (nonbenzodiazepine benzodiazepine receptor agonists)
Alprazolam (Xana'x) also classified as an anxiolytic	Amobarbital (Amytal)	Buspirone (Buspar) also classified as an anxiolytic
Chlordiazepoxide (Librium)	Butabarbital (Butisol)	Chloral Hydrate (Somnote) replaced with safer medications
Clonazepam (Klonopin)	Mephobarbital (Mebaral)	Eszoplicone (Lunesta)
Clorazepate (Tranxene)	Pentobarbital (Nembutal)	Ramelteon (Rozerem)
Diazepam (Valium)	Phenobarbital (Luminol)	Zaleplon (Sonata)
Flurazepam (Dalmane)	Secobarbital (Seconal)	Zolpidem (Ambien)
Lorazepam (Ativan) also classified as an anxiolytic		
Midazolam (Versad)		
Oxazepam (Serax)		
Quazepam (Doral)		
Temazepam (Restoril)		
Triazolam (Halcion)		

structure, duration of action, and adverse effects are different. The medications are often used for their hypnotic effects in short-term relief of insomnia. The medications are generally recommended for short-term relief of insomnia (10 days or less). The exception is Eszoplicone, which can be used for more long-term sleep disorders.

Contraindications/Drug interactions

In general, patients who have sleep apnea syndrome, severe respiratory insufficiency, and acute narrow angle glaucoma. They should not be prescribed during pregnancy. The additive CNS effects of other drugs should be considered when prescribing sedative hypnotic medication. Medications potentially causing additive effects include other medications in the sedative hypnotic class, phenothiazines, antidepressants, scopolamine, haloperidol, and clozapine.

Adverse Events

A major adverse affect of most of the sedative hypnotic medications is that of physical and psychological tolerance (reduced excitability of the neurons) and potential drug abuse. When short- and intermediate-acting benzodiazepines are abruptly discontinued after physical tolerance, the person may experience a rebound effect characterized by anxiety or it may result in a seizure. Most of these medications are controlled substances.

Related to toxicity, the medications can produce coma and death with an overdose, particularly with barbiturates. The benzodiazepines and NBRAs will most likely produce anesthesia at high doses, rather than coma and death. In mild cases, the patient may experience drowsiness, mental confusion, lethargy, ataxia, and hypotension.

The patient should be instructed not to drink alcohol and take sedative hypnotic medications as this can lead to death.

Side Effects

Paradoxical reactions can occur with benzodiazepines including mania, irritability, restlessness, agitation, aggression, rage, or hallucinations.

Cross References

- ▶ Barbiturates
- ▶ Benzodiazepines
- ▶ Psychopharmacology

References and Readings

Cranwell-Bruce, L. (2007). Hypnotic sedative drugs. *MEDSURG Nursing, 16*(3), 198–200.

Jaffe, J., Bloor, R., Crome, I., Carr, M., Alam, F., Simmons, A., et al. (2004). A postmarketing study of relative abuse liability of hypnotic sedative drugs. *Addiction, 99*(2), 165–173.

Oray, N., Hocaoglu, N., Oray, D., Demir, O., Atilla, R., & Tuncok, Y. (2008). Sedative-hypnotic medication exposures and poisonings in Izmir, Turkey. *Basic & Clinical Pharmacology & Toxicology, 103*(4), 380–385.

Schuckit, M., Smith, T., Kramer, J., Danko, G., & Volpe, F. (2002). The prevalence and clinical course of sedative-hypnotic abuse and dependence in a large cohort. *American Journal of Drug & Alcohol Abuse, 28*(1), 73.

Seguin–Goddard Formboard

► Tactual Performance Test

Seizure

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Definition

An epileptic seizure is "a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain" (Fisher et al., 2005). Neurons of the cerebral cortex generate seizures, although they originate in the thalamocortical system. Certain brain regions, including the temporal lobes, have a higher susceptibility to seizures. Seizures may be spontaneous or associated with a precipitating event such as sleep deprivation or alcohol consumption. They occur one or more times per day in some patients, but at much longer intervals in others.

A recent definition of epilepsy describes it as a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures and the occurrence of at least one epileptic seizure (Fisher et al., 2005). Well-established risk factors for seizures include moderate–severe head trauma, central nervous system infection, Alzheimer's disease, stroke, febrile seizures, cerebral palsy, and mental retardation. In symptomatic epilepsies, seizures are the consequence of a known or presumed lesion or other physical or metabolic etiology. In the idiopathic epilepsies, seizures are not associated with a brain lesion and tend to be self-limited or respond readily to medications, and genetic factors are important (Engel & Pedley, 2008).

There are many types of epileptic seizures. The *ictus* or *ictal period* refers to the seizure itself. A *prodrome* is a pre-ictal phenomenon that heralds the onset of a seizure. It is usually a sensation or change in behavior that is of variable duration and difficult to define. An *aura* is a subjective ictal phenomenon that may precede an observable seizure. When occurring in isolation, it constitutes a simple partial seizure (see below). The *postictal period* reflects temporary nervous system dysfunction that follows some seizures. The *interictal period* is the interval between seizures.

Ictal signs and symptoms are referred to as seizure semiology. Epileptic seizures have a broad range of semiologic features, depending on the location of onset in the brain and the propagation pattern (i.e., the cortical and subcortical neuronal networks activated), the age of the patient, sleep–wake cycle, medications, etc. By definition, a seizure must be accompanied by at least one change in sensory, motor, or automatic function or emotion, memory, cognition, behavior, or consciousness. The seizure may include positive behavioral signs and/or negative features with loss of function.

Paroxysmal discharges occur when the threshold of the neuronal membranes is reduced beyond the capability of intrinsic stabilizing mechanisms to prevent firing. The electroencephalogram (EEG) is based on volume conduction of ionic currents generated by nerve cells through the extracellular space and discharges appear on scalp EEG recording as spikes, slow waves, and spike-wave potentials (Browne & Holmes, 2008). The scalp EEG can appear normal during simple partial and subclinical seizures, but an abnormal electrical discharge is assumed to be present in the brain and measurable under ideal circumstances in the case of a true epileptic seizure. Invasive EEG recordings are sometimes necessary to reveal precise ictal onset zone localization. In some patients, a degree of abnormal neuronal activity is viewable on the scalp EEG even interictally and the interictal EEG abnormality may be more readily apparent after a period of sleep deprivation.

Historical Background

Hippocrates provided a description of different seizure types and localized seizures to the brain, 2,500 years ago. But, ancient attempts to classify seizures by etiology and pathology generally had serious shortcomings. For example, a Babylonian text emphasized demonic possession and unfortunately this conception of seizures remained common in Western writing through the Middle Ages. Progress in the understanding of seizures accelerated in the nineteenth century, a period when epilepsy started to

become a domain of the neurologist. The advent of the human EEG in the 1920s and later twentieth century progress in simultaneous video-EEG recordings and use of intracranial electrodes were among the key events in modern epileptology.

The terminology used to describe and classify seizures continues to evolve and no classification system satisfies all epileptologists. An early nineteenth century French school divided seizures into two categories: grand mal events (now more commonly known as generalized tonic-clonic seizures) and all other seizures, which were labeled petit mal or the minor evil (Niedermeyer, 1990). The term petit mal became synonymous with absence seizures and other distinctions were made. On the basis of the work of committees led by the French neurologist Henri Gastaut in the 1960s, the International League Against Epilepsy (ILAE) published a classification of seizures in 1981 that continues to be most widely accepted. It is a product of consensus expert opinion and emphasizes seizure semiology, as well as EEG findings, making it pragmatic for clinical use.

In brief, the 1981 ILAE taxonomic framework categorizes seizures as partial, generalized, or unclassified. The latter category covers, for example, some seizures occurring in infants. The initial clinical and EEG changes of partial seizures are consistent with a focal neuronal abnormality in one or both of the hemispheres. Partial seizures are further broadly divided into two types. In simple partial seizures (SPS), consciousness is preserved, whereas complex partial seizures (CPS) are characterized by altered consciousness. SPS symptom(s) may be motoric, somatosensory, autonomic, or psychic (i.e., marked by a disturbance in higher cortical function such as an illusion). CPS may be associated with automatisms, which are more or less coordinated, repetitive motor activities of the mouth, face, or extremities for which the patient usually is amnestic afterward. SPS may evolve into CPS and either of these types may evolve into a secondarily generalized tonic-clonic seizure. Generalized seizures represent a widespread, bilateral neuronal discharge. This category is heterogeneous and includes convulsive and nonconvulsive seizures. The generalized seizure subtypes include tonic-clonic, tonic, clonic, atonic, myoclonic, and absence. The absence seizure category has its own subtypes. Combinations of generalized seizure types can occur in the same patient.

Diagnosis of a specific seizure type has etiologic, therapeutic, and prognostic implications (Tuxhorn & Kotagal, 2008), although additional diagnosis at the epilepsy syndrome level can be more fruitful than seizure classification alone (Wyllie, 1997). It appears that some seizure types, especially if prolonged or frequent, can damage the brain. There is an increased mortality risk in patients with epilepsy, but it occurs mainly in those with symptomatic seizures due to brain tumors or cerebrovascular disease, for example (Engel & Pedley, 2008).

Successful treatment of seizures in the modern era began with the discovery of the anticonvulsant property of potassium bromide in the mid-nineteenth century. Phenobarbital was introduced early in the twentieth century, followed by phenytoin a few decades later, and then the benzodiazepines, carbamazepine, and sodium valproate in the 1960s and 1970s. By the end of the twentieth century, the number of antiepilepsy drug options had expanded further. Different medications often are used to treat generalized versus partial seizures.

Current Knowledge

The evaluation of a patient referred because of seizures requires gathering a thorough medical history, a physical exam, EEG, brain imaging, metabolic and toxic screens and, in some cases, EKG, spinal tap, or genetic testing. Paroxysmal episodes with a neurologic, cardiologic, metabolic, or psychological basis may mimic epileptic seizures and epilepsy, but these have no epileptiform electrical pattern in the brain. In particular, it has been estimated that about 20% of patients who are referred for evaluation at epilepsy specialty clinics do not have epilepsy but rather have nonepileptic seizures attributable to psychological factors. However, it also should be noted that some patients have both epileptic and nonepileptic seizures.

It has been estimated that about 5% of the population suffers at least one unprovoked seizure at some point in life and the figure may be as high as 10% when childhood febrile seizures are included (Engel & Pedley, 2008; Niedermeyer, 1990). Many individuals who suffer one seizure never experience another. Factors increasing the likelihood of recurrence include a prior neurological insult, a diagnosis of a partial seizure type, and an abnormal EEG. Approximately 3% of the population will experience recurrent seizures at some point during life (Browne & Holmes, 2008). Modern population-based studies in industrialized countries suggest that about two thirds of these patients will eventually become seizure-free with antiepilepsy drugs. Many in whom complete control of seizures is achieved subsequently are able to discontinue medications and maintain long-term remission. However, certain seizures, especially partial seizures, are resistant to medications. Fortunately, in some cases intractable seizures are surgically remediable.

Future Directions

Breakthroughs in the neurosciences and genomics will necessitate revisions to seizure classification. The ILAE currently is at work on a revision of the 1981 seizure taxonomy, with the aim of specifying seizure types via a greater emphasis on pathophysiologic mechanisms and neuronal substrates, as well as response to antiepileptic drugs, ictal EEG patterns, propagation patterns, and the association of seizure types with epilepsy syndromes (Engel, 2006).

Cross References

- ▶ Absence Epilepsy
- ▶ Anticonvulsants
- ▶ Aura
- ▶ Complex Partial Seizures with Automatisms
- ▶ Electroencephalography
- ▶ Epilepsy
- ▶ Grand Mal Seizure
- ▶ Partial Seizure (Simple)
- ▶ Psychomotor Epilepsy
- ▶ Status Epilepticus
- ▶ Temporal Lobe Epilepsy

References and Readings

Blume, W. T., Luders, H. O., Mizrahi, E., Tassinari, C., van Emde Boas, W., & Engel, J. Jr. (2001). Glossary of descriptive terminology for ictal semiology: Report of the ILAE task force on classification and terminology. *Epilepsia, 42*, 1212–1218.

Browne, T. R., & Holmes, G. L. (2008). *Handbook of epilepsy* (4th ed.). Philadelphia: Lippincott, Williams & Wilkins.

Commission on Classification and Terminology of the International League Against. Epilepsy (1981). Proposal for revised clinical and electroencephalographic classification of epileptic seizures. *Epilepsia, 22*, 489–501.

Engel, J. Jr. (2006). Report of the ILAE classification core group. *Epilepsia, 47*, 1558–1568.

Engel, J. Jr., & Pedley, T. A. (Eds.). (2008). *Epilepsy: A comprehensive textbook* (2nd ed.). Philadelphia: Lippincott, Williams & Wilkins.

Fisher, R. S., van Emde Boas, W., Blume, W., Elger, C., Genton, P., Lee, P., et al. (2005). Epileptic seizures and epilepsy: Definitions proposed by the International League Against Epilepsy (ILAE) and the International Epilepsy Bureau (IEB). *Epilepsia, 46*, 470–472.

Laidlaw, J., Richens, A., & Oxley, J. (Eds.). (1988). *A textbook of epilepsy* (3rd ed.). Edinburgh: Churchill Livingstone.

Niedermeyer, E. (1990). *The epilepsies: Diagnosis and management.* Baltimore, MD: Urban & Schwarzenberg.

Tuxhorn, I., & Kotagal, P. (2008). Classification. *Seminars in Neurology, 28*, 277–288.

Wyllie, E. (Ed.). (1997). *The treatment of epilepsy: Principles and practice* (2nd ed.). Baltimore, MD: Williams & Wilkins.

Seizure Disorder

▶ Epilepsy

Selection of Jurors

▶ Voire Dire

Selective Attention Models

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Definition

At nearly every waking moment, our brains are bombarded with far more sensory information than can be processed at one time. It is necessary, therefore, that some information is selected for processing at the expense of other information. This is the fundamental notion of selective attention. Selective attention models are formalisms that attempt to describe and understand the mechanisms by which behaviorally relevant sensory information is preferentially processed over less-relevant information. Several questions have been central to attempts to understand the process of selective attention. At what stage of processing does information get selected? What is the mechanism for giving priority to some signals over others? What is the fate of unattended stimuli? Are unattended items actively suppressed?

Historical Background

The intuitive concept of selective attention has a long history. In the nineteenth century, William James in his influential work, *The Principles of Psychology*, wrote "Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. . .. It implies withdrawal from some things in order to deal effectively with others. . .." In the 1950s, controlled experiments began

investigating selective listening in which subjects monitored and repeated speech presented to one ear while irrelevant speech was simultaneously presented to the other ear. In these so-called shadowing studies, the research participants were found to be largely unaware of any content in the unattended ear. Nonetheless, it was also found that some types of highly relevant information (e.g., a subject's own name) were able to be identified from the unattended ear.

Models were developed that attempted to capture and describe the mechanisms of selection. Early models were largely qualitative in nature – so-called flowchart or box-and-arrow models – in which information was seen as flowing from early stages of sensory or perceptual processing to later, higher-level process such as working memory or motor output. Later models have become more explicitly quantitative, which allows them to both model existing experimental data as well as to make explicit testable predictions. Although selective attention has been studied and models developed relating to virtually all sensory modalities, including interactions between sensory modalities, a majority of work on selective attention has focused on selective attention in the visual domain.

A fundamental classification is often made for selective attention models regarding the "locus of selection." In *early-selection* models, unattended information is filtered out or attenuated at early stages of processing. In *late-selection* models, by contrast, perceptual information is processed in parallel to a point of semantic identification at which point it competes for access to awareness, memory, or motor output. Hybrid models have also been proposed in which the locus of selection can vary depending on task demands such as perceptual or working memory load.

The first formal model of selective attention was proposed by Donald Broadbent in 1958 and is often referred to as the Filter Model of selective attention. Influenced by recent developments in communication theory, Broadbent proposed a model in which the physical aspects sensory information are initially registered in short-term storage buffers where they decay very rapidly. At this very early stage of processing, attention acted to select a single channel of information for further processing, where a channel was defined by information that shared specific physical properties such as color, spatial location, etc. In response to findings that subjects were able to report some types of especially salient information outside of the attended channel, Triesman proposed another early-selection model in which attention acted to reduce or attenuate non-attended information. Information for which strong learned associations existed might thus retain sufficient levels of activation to receive further processing.

In contrast with these early-selection models, theorists such as Deutsch and Deutsch, Duncan, and others proposed late-selection models in which all available sensory information could be processed in parallel up to the point of semantic identification, at which point attention was necessary to select the behaviorally relevant items for awareness and action. Evidence for late-selection models comes largely from interference effects from unattended stimuli. In the classic laboratory example known as the Stroop task, subjects must attend to and report the ink color of a word while at the same time ignoring the meaning of the word. Subjects are slower to respond and less accurate when the printed word is itself the name of a different color than the ink in which it is printed, for example, the word "blue" printed in red ink, compared to when the word spells a noncolor word or spells the same color as the ink. In flanker tasks, subjects must make a judgment about and respond to a centrally displayed target item which is flanked on either side by other items that are irrelevant to the task. Responses are slower and less accurate when the flanking items are associated with a response that is incompatible with the response required for the target item compared to when the flanking items are associated with a compatible response.

Selective attention may be stimulus-driven, that is, by external or exogenous events such as sudden movement of a visual item or the abrupt onset of a sound. In other cases, selective attention is said to be internally driven, that is, endogenous or voluntary. In exogenous cueing experiments, subjects may fix their eyes on the center of a computer display and detect or identify target stimuli displayed in their peripheral vision. Subjects will respond more rapidly to the targets if a cue stimulus is flashed at the same location (valid cues) just prior to the target's arrival and more slowly if the cue is flashed elsewhere (invalid cues). Interestingly, if the duration between an exogenous cue and the target exceeds more than a few hundred milliseconds, subjects will actually be slower to respond to validly cued stimuli than to invalidly cued stimuli – a phenomenon known as inhibition of return. This may reflect a bias to explore novel areas of the visual scene.

What is selected by attention? In models of visual selective attention, a distinction is often drawn between those models that suggest attention operates primarily in the spatial domain, such that preferential processing is allocated to all items at an attended location, and models in which attention selects objects. Attention may be directed to particular regions in the visual field but it is typically the case that such regions will contain specific objects. Indeed,

there is evidence that, at least in some cases, objects may be the unit of selection in attention. For example, attention to one part of an object will tend to facilitate processing of items or features located at other places on that same object compared to items located on other objects.

What is the function of attention? Anne Treisman and her colleagues have proposed an influential model called Feature Integration Theory, which suggests that attention is necessary to bind the various features of items in the world into a coherent perceptual representation. Visual objects in particular can be said to be composed of many different features – for example, size, color, shape, movement, etc. In Feature Integration Theory, features are registered in parallel in various spatial feature maps. Attention operates through a master map of space that binds together multiple features from the same location across these various maps. One line of evidence for Feature Integration Theory comes in the form of "illusory conjunctions" in which under heavy attentional demand observers may mistakenly report the presence of a target formed by a conjunction of features (e.g., a red X) when the individual features were present but associated with separate objects (e.g., a blue X and a red T).

One function of attention may be to bias the competitive interactions in the brain resulting from the simultaneous processing of multiple stimuli. Biased competition theory proposes that different stimuli compete with each other for neural representation. Attention serves to bias that competition in favor of the behaviorally relevant items. For example, imagine a cortical neuron that responds preferentially (i.e., more strongly) to stimulus A compared to stimulus B. When both stimuli are present in the absence of attention, the neuron will exhibit an intermediate response as a result of competition. However, when attention is directed to stimulus A then A "wins" the competition and the activity of that neuron will increase and resemble the activity level seen when stimulus A is presented alone. Correspondingly, when attention is directed to stimulus B then B "wins" and the activity of the neuron will decrease and appear similar to what would be seen if stimulus B was presented in isolation.

Current Knowledge

What is the brain basis of selective attention? Attention acts to modulate the processing of sensory information in the brain. In the visual domain, there is a mapping of regions of the visual field to specific regions of visual cortex. It is known that attention to any particular region of visual space increases neural processing in those distinct regions of the brain that correspond to that region of space. These changes in neural processing may be seen as early as primary visual cortex, which has sometimes been taken for *prima facie* evidence for early selection. Neuroimaging and direct recordings from the brain have shown that the size of attention effects increases from early to higher-order visual areas. In addition to increases in neural activity for attended regions of space, it also appears to be the case that there is active suppression of neural activity corresponding to unattended regions, particularly when those regions may contain distracting information. Further, certain regions of the brain appear to respond selectively to the presence of particular types of visual objects such as faces. When attention is directed to these objects (e.g., when one is attending to a face in a cluttered visual scene), increases in neural activity are seen in these specialized areas.

When visual stimuli are presented to a subject, electrical potentials (EEG) may be recorded from the scalp related to the processing of those stimuli by the visual system in the brain. Attention appears to alter the size of the EEG waveforms that are recorded but not to alter the timing or number of elements in the early portions of these waveforms, suggesting that attention is acting to alter the strength of the signal but not generally to change the speed of processing or to add fundamentally new stages of processing.

A distinction can be made between the sites of attentional modulation of sensory activity and the sources of that modulation. It is presumed that attentional control signals originate in higher-order brain areas where they exert a modulatory influence on sensory regions through feedback connections. Evidence for the role of specific brain regions in attentional control in humans comes from neuroimaging work and from patients with localized brain lesions. Among the areas that appear to be critical are the superior parietal lobe, the temporal–parietal junction, and the frontal eye fields.

The most common disorders of attention resulting from damage to the brain are *extinction* and *neglect*. Both extinction and neglect are commonly associated with unilateral lesions, especially in the right parietal lobe and other regions as well. These are deficits of attentional orienting in which the deficit is expressed as an impaired ability to orient attention to the contralesional side of space. Patients suffering from extinction typically are able to detect and orient to stimuli on either side of space but are impaired in their ability to detect stimuli contralateral to their lesion when stimuli are presented simultaneously to both sides of space. A similar but more

severe disorder is unilateral neglect. Neglect patients may be profoundly impaired in their ability to attend to items on the contralesional side of space despite having intact sensation. Such patients may dress only half of their bodies, eat food from only one-half of their plates, and fail to recognize or respond to individuals approaching them from the side opposite their lesion.

Cross References

- ▶ Attention
- ▶ Attention Network Test (ANT)
- ▶ Disengagement of Attention
- ▶ Extinction
- ▶ Focused Attention
- ▶ Hemi-Neglect
- ▶ Sustained Attention

References and Readings

Desimone, R., & Duncan, J. (1995). Neural mechanisms of selective attention. *Annual Review of Neuroscience, 18*, 193–222.

James, W. (1980). *The principles of psychology.* (Vol. 1). New York: Henry-Holt & Co.

Johnson, A., & Proctor, R. W. (2004). *Attention: Theory and practice.* Thousand Oaks, CA: Sage.

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13*, 25–42.

Selective Incomplete White Matter Infarction

▶ Small Vessel Ischemic Disease

Selective Reminding Test

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Synonyms

Verbal selective reminding test (VSRT)

Description

The Buschke Selective Reminding Test (SRT) is a test designed to measure verbal learning and memory through the use of a list-learning procedure over multiple trials. This paradigm is believed to separate verbal memory into distinct processes. The adult SRT involves reading the subject a list of 12 unrelated words and then having the subject immediately recall as many of these 12 words as possible. Every trial after the first involves selectively presenting only those words in which the subject did not recall on the immediately preceding trial. The selective reminding trials proceed in this manner until the subject is able to correctly recall all 12 words on three consecutive trials, or until 12 trials have been completed. Through assessing the recall of items that are not presented on a given trial, this test is believed to distinguish between retrieval from long-term storage (LTS) and short-term recall (STR). In theory, as the subject requires less reminding across trials, the short-term memory component of the SRT decreases, and the long-term memory component is more taxed (Hannay & Levine, 1985). More specifically, words not recalled on two consecutive trials are believed to be recalled from short-term memory. Words that have been recalled on two consecutive trials, but presented only on the first of the two trials, are believed to be recalled from LTS, and scored as long-term retrieval (LTR). The words recalled from LTS are further broken down into those recalled on every subsequent trial (termed consistent long-term retrieval, CLTR) and those recalled inconsistently (random long-term retrieval, RLTR). A sum recall scores on each trial is also calculated, which includes the sum of STR and LTR. The number of reminders provided by the examiner prior to the subsequent recall attempt is scored as 12 (or total number of words in list) minus Sum Recall of the previous trial.

Hannay and Levin (1985) created several alternate forms of the SRT. Furthermore, they extended Buschke's (1973) procedure by adding three additional trials. In their SRT, after the last selective reminding trial, a cued-recall trial is presented in which the first two to three letters of each word is presented on an index card, and the subject is asked to recall the corresponding word on the list. Following the cued-recall trial, a multiple-choice recognition trial is presented in which the subject is presented with a series of 12 index cards, each of which is composed of a list word, a synonym, a homonym, and one unrelated distractor word. The subject must select the list word from each card. A delayed-recall trial is presented without any forewarning, 30 min after the multiple-choice trial is

completed in which the subject is asked to recall the words from the list without any further cueing.

Currently, several different forms of the test are available (Buschke, 1973; Coen, Kinsella, Lambe, Kenny, & Darragh, 1990; Deptula et al., 1990; Dikmen, Heaton, Grant, & Temkin, 1999; Hannay & Levin, 1985). The test has also been developed in Spanish (Campo & Morales, 2004; Campo, Morales, & Juan-Malpartida, 2000) and Hebrew (Gigi, Schnaider-Beeri, Davidson, & Prohovnik, 1999).

Short adult versions of the SRT also exist. A six-trial, ten-item form for elderly individuals was created and normed by Wiederholt et al. (1993). Larrabee, Trahan, and Levin (2000) provided normative data of a six-trial administration of Form I of the SRT (Hannay & Levin, 1985) on 267 normal adults ranging from 18 to 91 years of age. The most recent version of the SRT is designed to specifically assess learning and its relationship with retrieval, called the open trial-SRT (OT-SRT) (Chiaravalloti, Balzano, Moore, & DeLuca, 2009).

Alternate child versions of the test also exist. Two alternate 12-word lists were developed by Clodfelter, Dickson, Newton Wilkes, and Johnson (1987) for children aged 9–12 years old. In this child version, the test ends when the child recalls all 12 words on two consecutive trials, or up to a total of eight selective recall trials. Clodfelter et al. (1987) provided normative data based on 58 children aged 9–12. Expanded norms using List 1 based on 475 school children aged 9–15 years have been provided (Miller, Murphy, Paniak, Spackman, & LaBonte, 1996, Unpublished data) (see Strauss, Sherman, & Spreen, 2006 for these norms). Three alternate eight word lists were also created and normed on 30 children ages 5 through 8 years (Morgan, 1982). This version ends when the child has correctly recalled all eight words on two consecutive trials, or up to a total of six selective recall trials.

In addition, the Test of Memory and Learning (TOMAL; Reynolds & Bigler, 1994) includes a word selective reminding (WSR) test in which a list of words is read, and trials continue until mastery is achieved or eight trials are completed. Normative data is available for ages 5 through 19:11. The TOMAL, second edition (TOMAL-2; Reynolds & Voress, 2007) contains expanded norms for ages 5 through 59:11 on the WSR. Similarly, the Children's Memory Scale (CMS; Cohen, 1997) also uses a selective reminding procedure for their Word Lists test, normed on children ages 5 through 16.

The adult version of the SRT requires an administration time of approximately 30 min, whereas the children's version (or adult short version) takes approximately 10 min.

Historical Background

Buschke devised the SRT in 1973. As described above, alternative and modified adult and child versions of the test were created throughout the 1980s. Alternate language versions were developed later in Spanish (Campo et al., 2000) and Hebrew (Gigi et al., 1999). No manuals for the various SRT tests exist, but administrative information and forms are available (Strauss et al., 2006).

Psychometric Data

Alternate form reliability ranges from 0.48 to 0.85. Information on test-retest reliability is not available. The SRT has been found to correlate modestly with other measures of verbal learning and memory (e.g., ► California Verbal Learning Test (CVLT), ► Rey Auditory Verbal Learning Test (RAVLT), and ► Wechsler Memory Scale (WMS)). An absence of practice effects has been shown for individuals suffering from neurological disorders. However, practice effects have been found for normal individuals (Strauss et al., 2006).

Clinical Uses

The SRT is one of the most widely used tests of verbal learning and memory. The unique selective reminding procedure allows for the analysis and distinction of retrieval from LTS and STR. It further distinguishes two aspects of long-term memory retrieval, RLTR and CLTR.

The SRT has been shown to be sensitive to normal age-declines in memory. The validity of the SRT has also been demonstrated with a wide variety of clinical populations, including those with closed-head injury, dementia of the Alzheimer type, multiple sclerosis, epilepsy, and individuals with mood or thought disorders (see Strauss et al., 2006 for details).

Cross References

► California Verbal Learning Test (California Verbal Learning Test - II)
► Children's Memory Scale
► Learning
► Memory
► Retrieval, Retrieval Techniques
► Rey Auditory Verbal Learning Test, Rey AVLT

► Short-Term memory
► Wechsler Memory Scale All Versions
► Word Memory Test

References and Readings

Buschke, H. (1973). Selective reminding for analysis of memory and learning. *Journal of Verbal Learning and Verbal Behavior, 12*, 543–550.

Campo, P., & Morales, M. (2004). Normative data and reliability for a Spanish version of the verbal selective reminding test. *Journal of Clinical and Experimental Neuropsychology, 25*, 991–999.

Campo, P., Morales, M., & Juan-Malpartida, M. (2000). Development of two Spanish versions of the verbal selective reminding test. *Journal of Clinical and Experimental Neuropsychology, 22*, 279–285.

Chiaravalloti, N. D., Balzano, J., Moore, N. B., & DeLuca, J. (2009). The open-trial selective reminding test (OT-SRT) as a tool for the assessment of learning and memory. *The Clinical Neuropsychologist, 23*, 234–251.

Clodfelter, C. J., Dickson, A. L., Newton Wilkes, C., & Johnson, R. B. (1987). Alternate forms of selective reminding for children. *The Clinical Neuropsychologist, 1*, 243–249.

Coen, R. F., Kinsella, A., Lambe, R., Kenny, M., & Darragh, A. (1990). Creating equivalent word lists for the Buschke selective reminding test in young and elderly subjects. *Psychological Reports, 3*, 1287–1295.

Cohen, M. J. (1997). *Children's memory scale.*San Antonio, TX: The Psychological Corporation.

Deptula, D., Singh, R., Goldsmith, S., Block, R., Bagne, C. A., & Pomara, N. (1990). Equivalence of five forms of the selective reminding test in young and elderly subjects. *Psychological Reports, 3*, 1287–1295.

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.

Gigi, A., Schnaider-Beeri, M., Davidson, M., & Prohovnik, I. (1999). Validation of a Hebrew selective reminding test. *Israel Journal of Psychiatry and Related Sciences, 36*, 11–17.

Hannay, J. H., & Levin, H. S. (1985). Selective reminding test: An examination of the equivalence of four forms. *Journal of Clinical and Experimental Neuropsychology, 7*, 251–263.

Larrabee, G. J., Trahan, D. E., & Levin, H. S. (2000). Normative data for a six-trial version of the verbal selective reminding test. *The Clinical Neuropsychologist, 14*, 110–118.

Morgan, S. F. (1982). Measuring long-term memory, storage and retrieval in children. *Journal of Clinical Neuropsychology, 4*, 77–85.

Reynolds, C. R., & Bigler, E. D. (1994). *Manual for the test of memory and learning.* Austin, TX: PRO-ED.

Reynolds, C. R., & Voress, J. K. (2007). *Test of memory and learning, second edition. Examiner's manual.* Austin, TX: PRO-ED.

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

Wiederholt, W. C., Cahn, D., Butters, N. M., Salmon, D. P., Kritz-Silverstein, D., & Barrett-Connor, D. (1993). Effects of age, gender, and education on selected neuropsychological tests in an elderly community cohort. *Journal of the American Geriatrics Society, 41*, 639–647.

Selective Serotonin Reuptake Inhibitors

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Synonyms

SSRIs

Definition

Selective serotonin reuptake inhibitors (SSRIs) are a type of psychotropic medication used primarily for the treatment of clinical depression. It inhibits the reuptake of extracellular serotonin, allowing more of the neurotransmitter substance to remain in the synaptic cleft to stimulate postsynaptic receptors. Common side effects of SSRIs include nausea and sexual dysfunction. A topic of much controversy is a possible increase in risk of suicidal ideation and behavior among pediatric and young adult patients (See Current Knowledge; Iversen, Iversen, Roth, & Bloom, 2009).

SSRIs are also used to treat other conditions. Reports suggest some efficacy for the treatment of Generalized Anxiety Disorder, Panic Disorder and Obsessive Compulsive Disorder (Bourin & Lambert, 2002), eating disorders (Powers & Bruty, 2009), ejaculatory disorders, and when taken in combination with tricyclic antidepressants, fibromyalgia (Stone, Viera, & Parman, 2003).

Current Knowledge

The efficacy of SSRIs in treating clinical depression is approximately 60–70%. Recent studies suggest treatment response is greater (compared to placebo controls) among patients with more severe symptoms (Kirsch, Deacon, Huedo-Medina, Scoboria, Moore, & Johnson, 2008; Fournier et al., 2010) and those who are carriers of the long (L) allele of the serotonin transporter gene (5-HTTLPR; Horstmann & Binder, 2009).

A modest increase in risk of suicidal ideation and behavior (suicidality) has been observed with SSRIs and other antidepressants among pediatric and young adult patients, leading to several U.S. Food and Drug

Administration (FDA) advisories on the use of these compounds. In a meta-analysis of 20 clinical trials, the relative risk for suicidality among pediatric patients treated with SSRIs was approximately 1.6 times that of patients not receiving treatment (Hammad, Laughren, & Racoosin, 2006). Some have hypothesized that the modest increase in risk may occur in a subset of high risk patients with agitated major depression or unrecognized bipolar disorder (Rihmer & Akiskal, 2006). More research is needed to clarify issues of safety and efficacy of these compounds.

Cross References

► Antidepressants
► Anxiolytics
► Serotonin

References and Readings

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

Fournier, J. C., DeRubeis, R. J., Hollon, S. D., Dimidjian, S., Amsterdam, J. D., Shelton, R. C., et al. (2010). Antidepressant drug effects and depression severity: A patient-level meta-analysis. *Journal of the American Medical Association, 303*, 47–53.

Hammad, T. A., Laughren, T., & Racoosin, J. (2006). Suicidality in pediatric patients treated with antidepressant drugs. *Archives of General Psychiatry, 63*, 332–339.

Horstmann, S., & Binder, E. B. (2009). Pharmacogenomics of antidepressant drugs. *Pharmacology and Therapeutics, 124*, 57–73.

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.

Kirsch, I., Deacon, B. J., Huedo-Medina, T. B., Scoboria, A., Moore, T. J., & Johnson, B. T. (2008). Initial severity and antidepressant benefits: A meta-analysis of data submitted to the food and drug administration. *PlOS Medicine, 5*, e45.

Powers, P. S., & Bruty, H. (2009). Pharmacoptherapy for eating disorders and obesity. *Child Adolescent Psychiatric Clinics of North America, 18*, 175–187.

Rihmer, Z., & Akiskal, H. (2006). Do antidepressants t(h)reat(en) depressives? Toward a clinical judicious formulation of the antidepressant-suicidality FDA advisory in light of declining national suicide statistics from many countries. *Journal of Affective Disorders, 94*, 3–13.

Stone, K. J., Viera, A. J., & Parman, C. L. (2003). Off-label applications for SSRIs. *American Family Physician, 68*, 498–504.

SSRIs

► Selective Serotonin Reuptake Inhibitors

Selegiline

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Generic Name

Selegiline

Brand Name

EMSAM (transdermal), eldepryl

Class

Monoamine Oxidase inhibitor (MAOI)

Proposed Mechanism(s) of Action

Irreversibly inhibits monoamine oxidase from breaking down any of the catecholamines, thus boosting their presynaptic release; transdermal selegiline selectively inhibits MAO-B in the intestine and liver, thus reducing the chances for dietary side effects.

Indication

Major depression; Parkinson's disease.

Off Label Use

Treatment-resistant depression, panic disorder, social anxiety disorder, treatment-resistant anxiety disorders, and Alzheimer's disease.

Side Effects

Serious

Theoretically, could cause hypertensive crisis if given with certain foods or drugs. May also theoretically cause

serotonin syndrome if given with other serotonergic agents. At high does could cause seizure.

Common

Local site reactions for the patch or the adhesive. Insomnia, headache, diarrhea, dry mouth, nausea, dizziness, abdominal pain, confusion, hallucinations, and vivid dreams.

References and Readings

Physicians' Desk Reference (62nd ed.). (2007). Montvale, NJ: Thomson PDR.

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

Additional Information

Drug Interaction Effects: http://www.drugs.com/drug_interactions.html
Drug Molecule Images: http://www.worldofmolecules.com/drugs/
Free Drug Online and PDA Software: www.epocrates.com
Gene-Based Estimate of Drug interactions: http://mhc.daytondcs.com:8080/cgi bin/ddiD4?ver=4&task=getDrugList
Pill Identification: http://www.drugs.com/pill_identification.html

Self-Awareness

► Anosognosia

Self-Control

► Self-Regulation

Self-Incrimination

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Synonyms

Compulsory self-incrimination; Pleading the fifth

Definition

Self-incrimination is the act of accusing oneself of a crime for which a person can then be prosecuted. The act of self-incrimination may happen either directly or indirectly. Oftentimes, a person directly incriminates when they disclose information that is self-incriminating during an interrogation and under pressure from another. Individuals have a tendency to indirectly self-incriminate when they provide information without the pressure from another individual and by nature is done so voluntarily.

References and Readings

Levy, L. W. (1986). *Origins of the fifth amendment* (reprint ed.). New York: Macmillan.

Self-Management

► Self-Regulation

Self-Rating Depression Scale

► Zung Self-Rating Depression Scale

Self-Regulation

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Synonyms

Behavioral control; Behavioral regulation; Effortful control; Self-control; Self-management

Definition

Self-regulation is a global term that has multiple subconstructs and is often used interchangeably with terms such as behavioral control, behavioral regulation, self-control, self-management, effortful control, and self-regulated

learning (Boekaerts et al., 2000; Bronson, 2000; Eisenberg et al., 2001; Post et al., 2006). Baumeister and Vohs (2004) provided the most global definition for self-regulation. They defined self-regulation as a person's ability to modulate, activate, and depress cognitive, behavioral and emotional responses to a variety of stimuli. Given this definition of self-regulation, research regarding inhibition, motivation, compliance, modulating emotion, impulsivity, hyperactivity, and attention all involve components that comprise self-regulation.

Cross References

- ▶ Attention
- ▶ Attention Deficit, Hyperactivity Disorder
- ▶ Behavior Rating Inventory for Executive Functions
- ▶ Disinhibition
- ▶ Executive Functioning
- ▶ Impulsivity
- ▶ NEPSY-II
- ▶ Posttraumatic Stress Disorder

References and Readings

Barkley, R. A. (2001). The executive functions and self-regulation: An evolutionary neuropsychological perspective. *Neuropsychology Reviews, 11*, 1–29.

Baumeister, R. F., & Vohs, K. D. (Eds.). (2004). *Handbook of self-regulation.* New York: Guilford.

Lewis, M. D., & Todd, R. M. (2007). The self-regulating brain: Cortical-subcortical feedback and the development of intelligent action. *Cognitive Development, 22*, 406–430.

Sheese, B. A., Rothbart, M. K., Posner, M. I., White, L. K., & Fraundorf, S. H. (2008). Executive attention and self-regulation in infancy. *Infant Behavior & Development, 3*, 501–510.

Self-Report Measures

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Definition

A common form of data collection in clinical research and practice is administration of a **self-report measure** in which an individual is asked to provide information about their subjective experiences and behaviors. Although less common, an individual's caregiver may also be asked to complete self-report measures on that individual's behalf, such as when a caregiver for a patient with dementia completes an activities of daily living questionnaire.

Current Knowledge

Self-report measures are usually completed in a paper-and-pencil format, although computer administration is utilized with increasing frequency. The scope of self-report measures may vary from assessment of a broad range of symptoms (e.g., childhood behavior disorders) to targeted assessment of specific symptoms (e.g., obsessive compulsive disorder). The selection of an appropriate self-report measure is dependent on the goal of the clinician's assessment and is aided by consideration of the reliability and validity of the measure (Kenny, Alvarez, Donohue, & Winick, 2008).

There are several types of self-report measures (Field & Hole, 2003). Problem behavior checklists are efficient tools that have the ability to survey a large variety of issues that an individual may experience. Daily diaries, self-monitoring records, and logs are also useful instruments that will enable the clinician or researcher to gain a wealth of data on an individual's subjective experiences. One of the more common self-report measures is a yes/no scale in which an individual is asked questions that elicit either a "yes" or "no" response. The limited, forced choice format of the yes/no scale, however, may not adequately capture variability in the human experience. Another frequently used self-report measure is the Likert-type scale. In this scale, an individual is asked to read a statement and indicate their level of agreement with that statement from a range of forced choices. Typical Likert-type scales contain categorical choices from three to seven (e.g., "not at all true," "somewhat true," "very true"), although more choices are possible. Likert-type scales have the advantage of capturing a broader range of responses than a yes/no scale. A final type of self-report measure is the visual analogue scale (VAS). Although there is a lot of variability in the depiction of the VAS, this measure is often illustrated as a line with a minimum of numerical markers that range from lowest to highest value. For instance, an individual may be presented with the question, "How happy are you right now?" and then asked to indicate their response by putting an "X" on the VAS line, which contains a

marker at one of the lines that denotes "0 = not at all happy" and a marker at the far end of the line that denotes "100 = very happy." The score is determined by measuring the distance from the start of the scale to the position of the "X" that the respondent has indicated.

The use of self-report measures is accompanied by relative advantages and disadvantages (Jex, 2002). An advantage of self-report measures is that they are generally cost effective and easily understood by the respondent and easily administered by the clinician or researcher. Self-report measures are also valuable tools that provide the respondent with a format in which to express their perceptions of their experiences, reflections on their internal or emotional states, or their view of others. One of the primary disadvantages of self-report measures, however, is that they assume that information provided by an individual is valid (Jex, 2002). That is, they assume that the respondent understands and is able to characterize the information that is being assessed, and that the respondent is reporting information accurately. Individuals may not respond truthfully to self-report measures for reasons that are both deliberate (e.g., secondary gain, poor motivation) and not deliberate (e.g., social desirability). Regardless of the source, however, it is advisable for the clinician or researcher to be aware of the threats to validity for self-report measurement and to take steps to avoid or mitigate these potential confounders. For instance, the researcher may wish to consider administration of both subjective self-report measures and objective measures. It would also be preferable to use self-report instruments, which have response bias scales embedded in the test.

Cross References

► Response Bias

References and Readings

Field, A., & Hole, G. (2003). *How to design and report experiments.* London: Sage.

Jex, S. M. (2002). *Organizational psychology: A scientist-practitioner approach.* New York: Wiley.

Kenny, M. C., Alvarez, K., Donohue, B. C., Winick, C. B. (2008). Overview of behavioral assessment with adults. In M. Hersen & J. Rosqvist (Eds.), *Handbook of psychological assessment, case conceptualization, and treatment* (Vol. 1). New York: Wiley.

Semantic Cue

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Definition

A semantic cue is a prompt that contains semantic information, and is given to facilitate word retrieval. Semantic information is knowledge that is related to the meaning of the word. This may include a formal description or definition (e.g., "Something that contains coffee" for *cup*), grammatical information (e.g., word class, conventional usage), word/phrase associations (e.g., "I spilled the water out of mine this morning" for *cup*), sentence completion (e.g., "Here is my coffee. . ." [cup]), and perceptual information (e.g., gesturing the object's use). A semantic cue may be considered weak or strong depending on how much semantic information is provided. Semantic cues are distinguished from phonemic cues, which provide information about the sounds of the word (e.g., /k/ as a cue for *cup*).

The individual's response to semantic cues may be included in the assessment of naming ability, either informally or as part of a standardized naming test (e.g., see ► Boston Naming Test). Semantic Cueing Treatment (SCT) is one therapeutic use of semantic cues that enhances word retrieval in patients with aphasia.

Cross References

► Anomia
► Aphasia
► Boston Naming Test
► Naming
► Phonemic Cue
► Semantic Paraphasia
► Speech-Language Therapy
► Word Retrieval

References and Readings

Chapey, R. (2008). *Language intervention strategies in aphasia and related neurogenic communication disorders.* Baltimore, MD: Lippincott, Williams & Wilkins.

Wambaugh, J., Cameron, R., Kalinyak-Fliszr, M., Nessler, C., & Wright, M. (2004). Retrieval of action names in aphasia: Effects of two cueing treatments. *Aphasiology, 18*(11), 979–1004.

Semantic Dementia

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Synonyms

Frontotemporal lobar degenerations; Primary progressive aphasia; Progressive fluent aphasia

Short Description

Semantic dementia (SD) is one of three clinical syndromes collectively associated with frontotemporal lobar degeneration (FTLD) (Neary et al., 1998). It typically begins in the presenium, and the initial presentation is characterized by severe naming and word comprehension deficits, as well as deficits in object recognition (i.e., associative agnosia). Other cognitive domains remain relatively preserved early in the disease course. The term semantic dementia has also been used to refer to a subtype of primary progressive aphasia, to describe patients with progressive fluent aphasia and verbal comprehension deficits, even in the absence of associative agnosia (Mesulam, 2003).

Categorization

The diagnostic features of SD outlined by Neary et al. (1998) are given below.
Core diagnostic features:

1. Fluent, empty speech; little information conveyed, use of broad generic terms
2. Loss of word meaning including both single-word comprehension and naming (i.e., two-way naming deficit)
3. Semantic paraphasias, often consisting of superordinate category substitutions (e.g., animal for camel), or category errors (e.g., dog for elephant)
4. Prosopagnosia: impaired recognition of familiar faces
5. Associative agnosia: impairment of object recognition in any sensory modality
6. Preserved single-word repetition, reading, perceptual matching, and copying

Supporting diagnostic features:

1. Press of speech; speaking without interruption
2. Idiosyncratic word usage (e.g., calling any small object a "container")
3. Absence of phonemic paraphasias in speech
4. Surface dyslexia/dysgraphia (Surface dyslexia refers to a type of reading impairment that is characterized by the overuse of phonological principles, especially for low-frequency words (Wilson et al., 2009). For example, "plaid" may pronounced as "played." Reading and spelling of orthographically regular words and pronounceable nonwords are preserved.)
5. Preserved calculation
6. Loss of sympathy and empathy
7. Preoccupation with narrowed interests
8. Abnormal preoccupation with money or financial economy (e.g., hoarding money, miserliness)

Neuropathology and Genetics

Similar to the other subtypes of FTLD, Alzheimer's disease pathology is not often associated with the clinical syndrome of SD. In a series of 18 postmortem cases, the majority of SD subjects were found to have the pathology of frontotemporal dementia with ubiquitinated inclusions, with Pick disease being the next most common pathological finding (Davies et al., 2005). Genetically, SD is the subtype of FTLD least likely to show a family history of dementia (17%) or an autosomal dominant pattern (1.9%) (Goldman et al., 2004).

Epidemiology

In a study of 353 patients from three different neurology clinics, SD was found to represent the smallest subgroup among the FTLD disorders; SD = 19%, frontotemporal dementia = 56%, and progressive nonfluent aphasia = 25% (Miller, 2007). The age of onset was 59 in SD, similar to the FTD group. Unlike FTD, which is more prevalent in male subjects, the male to female ratio in SD is more likely to be equal (Rogers, Ivanoiu, Patterson, & Hodges, 2006).

Natural History, Prognostic Factors, Outcomes

In SD, the dominant presenting feature is the decline of expressive vocabulary, often related to proper nouns.

A decline in word comprehension is also common. However, because SD includes nonverbal semantic deficits, it is also common to see deficits in the recognition of faces and facial emotions. Behavioral and personality changes in SD typically are not very prominent in the early stages of the disease. However, as the disease progresses to the frontal lobes, behavioral and personality changes have been found to emerge, and the changes have been shown to be similar to those of FTD. For example, apathy and changes in food preferences (Snowden, Neary, Mann, Goulding, & Testa, 1992), stereotypic behavior, changes in eating preference, disinhibition, and reduced social awareness have all been reported in SD (Bozeat, Gregory, Ralph, & Hodges, 2000).

Median survival from onset in SD has been found to be approximately 12 years, similar to Alzheimer's disease (AD), and significantly longer than the FTD subtype of FTLD (8.7 years) (Roberson et al., 2005). Unlike FTD, SD is rarely found to be associated with amyotrophic lateral sclerosis (ALS).

Neuropsychology and Psychology of SD

Given that there are multimodal semantic deficits spanning verbal and nonverbal domains, characterizing cognitive deficits in SD can be challenging. Standardized aphasia batteries, such as the Boston diagnostic aphasia examination (BDAE; Goodglass & Kaplan, 1983) or the Western aphasia battery (WAB; Kertesz, 1982), are useful in systematically documenting early language impairments, in particular the presence of a two-way naming deficit. The Boston naming test (Kaplan, Goodglass, & Weintraub, 1983) is a picture naming task that is often used to determine the presence of semantic errors (e.g., "mouse" for a picture of a "camel"). The pyramids and palm trees test (Howard & Patterson, 1992) can be administered for more detailed assessment of semantic knowledge. In this test, the patient is asked to match a target word or picture to one of two choices based on semantic principles. The stimuli can be verbal (either written or spoken) or nonverbal (pictorial), which allows for assessment of semantic knowledge through multiple modalities.

Evaluation

In addition to detailed neuropsychological evaluation, neuroimaging (e.g., MRI, SPECT, PET) can be helpful in establishing the diagnosis. SD typically involves more circumscribed atrophy involving the anterior temporal lobes, whereas both the frontal and anterior lobes are atrophied in FTD. In contrast to AD, posterior cortical regions, including the parietal, posterior temporal, and occipital regions are typically spared in SD (Rosen et al., 2002).

Treatment

There are currently no medications that are specifically approved for treating any of the subtypes of FTLD, including SD. The majority of clinical trials in the past have focused on symptomatic treatment of behavioral issues. Selective serotonin reuptake inhibitors are typically used in the early stages for the management of behavioral symptoms (Boxer & Boeve, 2007). Refractory behavioral symptoms may require the use of atypical antipsychotic agents, although potential negative side effects may limit the efficacy of these types of medications.

Patients with SD may benefit from speech-language treatment early in the course of the disease. The types of interventions used are based on the specific areas of language impairment and are primarily aimed at reducing the rate of decline in functional communication skills (Thompson & Johnson, 2005).

Cross References

- ▶ Frontotemporal Lobar Degenerations
- ▶ Progressive Aphasia

References and Readings

Boxer, A. L., & Boeve, B. F. (2007). Frontotemporal dementia treatment: Current symptomatic therapies and implications of recent genetic, biochemical, and neuroimaging studies. *Alzheimer Disease & Associated Disorders, 21*(4), S79–S87.

Bozeat, S., Gregory, C. A., Ralph, M. A., & Hodges, J. R. (2000). Which neuropsychiatric and behavioural features distinguish frontal and temporal variants of frontotemporal dementia from Alzheimer's disease? *Journal of Neurology, Neurosurgery & Psychiatry, 69*(2), 178–186.

Davies, R. R., Hodges, J. R., Kril, J. J., Patterson, K., Halliday, G. M., & Xuereb, J. H. (2005). The pathological basis of semantic dementia. *Brain, 128*(Pt 9), 1984–1995.

Goldman, J. S., Miller, B. L., Safar, J., de Tourreil, S., Martindale, J. L., Prusiner, S. B., et al. (2004). When sporadic disease is not sporadic: The potential for genetic etiology. *Archives of Neurology, 61*(2), 213–216.

Goodglass, H., & Kaplan, E. F. (1983). *The Boston diagnostic aphasia examination.* Philadelphia: Lea and Febiger.

Howard, D., & Patterson, K. (1992). *Pyramids and palm trees: A test of symantic access from pictures and words.* Bury St. Edmonds, Suffolk, UK: Thames Valley Test Company.

Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *The Boston naming test.* Philadelphia: Lea and Febiger.
Kertesz, A. (1982). *Western aphasia battery (WAB).* San Antonio, Texas: The Psychological Corporation.
Mesulam, M.-M. (2003). Primary progressive aphasia- A language-based dementia. *New England Journal of Medicine, 349*(16), 1535–1542.
Miller, B. L. (2007). Frontotemporal dementia and semantic dementia: Anatomic variations on the same disease or distinctive entities? *Alzheimer Disease & Associated Disorders, 21*(4), S19–S22.
Neary, D., Snowden, J. S., Gustafson, L., Passant, U., Stuss, D., Black, S., et al. (1998). Frontotemporal lobar degeneration: A consensus on clinical diagnostic criteria [see comments]. *Neurology, 51*(6), 1546–1554.
Roberson, E. D., Hesse, J. H., Rose, K. D., Slama, H., Johnson, J. K., Yaffe, K., et al. (2005). Frontotemporal dementia progresses to death faster than Alzheimer disease. *Neurology, 65*(5), 719–725.
Rogers, T. T., Ivanoiu, A., Patterson, K., & Hodges, J. R. (2006). Semantic memory in Alzheimer's disease and the frontotemporal dementias: A longitudinal study of 236 patients. *Neuropsychology, 20*(3), 319–335.
Rosen, H. J., Gorno-Tempini, M. L., Goldman, W. P., Perry, R. J., Schuff, N., Weiner, M., et al. (2002). Patterns of brain atrophy in frontotemporal dementia and semantic dementia. *Neurology, 58*(2), 198–208.
Snowden, J. S., Neary, D., Mann, D. M., Goulding, P. J., & Testa, H. J. (1992). Progressive language disorder due to lobar atrophy. *Annals of Neurology, 31*(2), 174–183.
Thompson, C., & Johnson, N. (2005). Rehabilitation of language deficits. In D. Koltai & K. Welsh-Bohmer (Eds.), *Geriatric neuropsychology: Assessment and intervention.* New York: Guilford Press.
Wilson, S. M., Brambati, S. M., Henry, R. G., Handwerker, D. A., Agosta, F., Miller, B. L., et al. (2009). The neural basis of surface dyslexia in semantic dementia. *Brain, 132*(Pt 1), 71–86.

Semantic Errors

► Semantic Paraphasia

Semantic Fluency

► Verbal Fluency

Semantic Memory

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Synonyms

Conceptual knowledge; Knowledge system

Definition

Semantic memory encompasses one's general world knowledge. It also refers to a memory system that stores, organizes, and manipulates information pertaining to the meaning of words, concepts, and their associations. This organized knowledge system enables people to make judgments about the properties and functions of items, such as whether a hammer is a living or nonliving thing, can be categorized as a tool, has a handle, or is larger than a screwdriver. Semantic memory is almost always contrasted with episodic memory. Whereas factual information within semantic memory is context free (e.g., knowing that the weather in Acapulco is typically hot, Thai food is spicy, the chemical symbol for potassium is K), episodic memory is contextually detailed (e.g., recollections of getting sunburned in Acapulco, eating a spicy meal at a Thai restaurant, studying the periodic table to learn chemical symbols). Thus, semantic memory is encyclopedic, whereas episodic memory has an experiential or autobiographical referent and can be dated absolutely (in terms of clocks or calendars) or relatively (in terms of before or after some other event).

Historical Background

Quillian (1968) introduced the term *semantic memory* in a book chapter of the same name to refer to a hierarchical network model of semantic knowledge. The model consists of a set of propositions or concepts that are represented as nodes, which are organized hierarchically according to the semantic relations between them. The taxonomic levels within the network range from basic categories (e.g., animals) to intermediate categories (birds, mammals) to specific categories (canary, dog), which themselves can be subdivided one or more times (spaniel, poodle, or Cocker spaniel; English Springer spaniel, toy poodle). Features characterizing one level (e.g., animals breathe and eat) apply to all members of lower levels, which is an economical way of storing information without any repetition for subordinate category members. Problems with the hierarchic model, such as the inability to account for exceptions (e.g., penguins are birds that do not fly, bats are mammals that do) and typicality effects (faster classification of typical than atypical category members) led to other models of semantic memory, including feature comparison models (Smith, Shoben, & Rips, 1974), spreading activation models (Collins & Loftus, 1975), and connectionist models

(McClelland & Rumelhart, 1985). For example, in connectionist theories of semantic processing, activation in one node spreads to conceptually interconnected nodes within a network. The stronger the conceptual connection between nodes, the more readily the activation spreads between them.

Although the term *semantic memory* was published in 1968 by Quillian, most psychologists associate the term with a book chapter by Endel Tulving in a 1972 book on the organization of human memory. In that initial chapter and a subsequent book, Tulving (1972, 1983) distinguished semantic from episodic memory in terms of the characteristics of their operations, the kind of information they process and represent, their underlying neural substrates, and their developmental trajectories. The episodic/semantic distinction supported the more general idea that memory is not a unitary function. This idea in turn gave rise to a theory of multiple (five) memory systems: semantic memory, episodic memory, working memory, the perceptual representation system, and procedural memory (Schacter & Tulving, 1994). These distinctions were based on (1) experimental studies showing that healthy populations perform differently on different types of memory tasks and (2) neuropsychological studies showing selectively preserved and impaired performances on different types of memory tasks following brain damage. More recently, neuroimaging studies have validated the concept of dissociable memory systems, but they have also revealed complex interactions among them.

Current Knowledge

Personal Versus General Semantic Memory

Current research typically distinguishes between two major subdivisions within semantic memory. *General semantic memory* refers to conceptual, factual, and world knowledge. Examples of general semantic memory include factual knowledge (e.g., the University of Michigan is in Ann Arbor; Gerald Ford played football there). *Personal semantics* includes knowledge about past and current personal information, such as one's age, address, marital status, occupation, education, friends and family, etc. Examples of personal semantics include the knowledge of where, when, and what one studied in school as well as the highest degree obtained. In contrast to personal and general semantic memory, specific recollections of one's own graduation or of watching a documentary about Gerald Ford are episodic memories.

Semantic Versus Episodic Autobiographical Memory

As noted, autobiographical knowledge is contained within one's personal semantics. Such conceptual knowledge about one's own past derives from accumulated experiences that may occur over many years. In this way, events that initially constitute episodic memories become "semanticized" over time. For example, a person may know that his or her family went out for ice cream every Sunday evening in the summer, which is a "gist" memory within one's personal semantics. Nevertheless, the person may specifically recall a particular incident from this habitual activity, which would constitute an episodic memory.

Assessment of Semantic Memory

Semantic memory is commonly assessed in standard neuropsychological evaluations by verbal fluency with category cues, such as supermarket items or animals, confrontation naming with the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 2001), and the Vocabulary subtest of the WAIS-III (Wechsler, 1997). The Pyramids and Palm Trees test (Howard & Patterson, 1992) is a nonverbal test of semantic knowledge used primarily by speech-language pathologists or for specialized assessments of semantic memory. In this test, patients view a picture or a word of a target (e.g., pyramid) and must select a conceptually related item from among two alternatives (e.g., a pine tree or a palm tree). The Cambridge semantic memory battery (Hodges & Patterson, 1995) was developed to assess consistency of deficits across multiple tasks involving a central semantic knowledge system. Target items representing living and man-made things are repeatedly tested in different ways throughout the battery, which includes category fluency, picture naming, word-picture matching, sorting of pictures and words at different levels of specificity, and generation of word definitions.

Semantic Memory Deficits in Dementia

Semantic memory deficits are common in several dementia disorders. They are the defining feature, however, of semantic dementia, a variant of frontal–temporal dementia

associated with atrophy of the anterior and inferolateral temporal lobes. The breakdown of conceptual knowledge in semantic dementia is pervasive and results in severe anomia as well as impairments in single-word comprehension, verbal fluency (category fluency worser than letter fluency), vocabulary, and general knowledge. Deficits are observed using different materials (words, pictures, or real objects), presentation modalities (visual, auditory, or tactile), test formats (receptive or expressive), and response modalities (oral or pointing). One of the most striking features of semantic dementia is its selectivity; despite the severe impairment in semantic memory, there is relative sparing of other aspects of language (including syntax and phonology), perceptual skills, nonverbal problem solving, autobiographical memory, and episodic memory. Notably, patients are able to use familiar objects appropriately early in the disorder (especially in their own home) despite being unable to name them. The pattern of deficits observed in this population supports the theory of a central (amodal) semantic knowledge system. Although the anterior temporal lobes are affected bilaterally in semantic dementia, the pathology is typically asymmetric, with corresponding lateralization of the semantic deficits observed. Those with greater left-side involvement show particularly marked anomia and comprehension impairments, whereas predominant right-sided pathology is nearly always associated with deficits in person knowledge.

Impaired semantic knowledge also typifies Alzheimer's disease. These deficits tend to occur early in the course of the disease, in concert with pathologic changes in temporal–parietal cortex. For example, individuals with Alzheimer's disease may identify a picture of a hippopotamus as an "animal." This type of error reflects a loss of knowledge about the specific features that characterize a unique category exemplar despite retention of the superordinate concept. Similarly, Alzheimer's disease patients typically show a disproportionate impairment on semantic relative to phonemic verbal fluency tasks, as noted for patients with semantic dementia. That is, they have greater difficulty generating items belonging to a particular category, such as animals or supermarket items, than generating words beginning with a particular letter, such as "F." Taken together, impaired confrontation naming and disproportionately impaired semantic fluency support the notion of a breakdown of semantic knowledge in Alzheimer's disease.

Semantic memory impairments are also observed among individuals with Lewy body dementia. In contrast to individuals with semantic dementia or Alzheimer's disease, those with Lewy body dementia have greater difficulty with accessing meaning from pictures than words and are equally impaired on letter and category fluency. Thus, semantic memory deficits in this disorder are not selective and are attributed to a combination of semantic and visuoperceptual impairments. Similarly, semantic memory is often impaired in the various frontal–temporal dementias (progressive nonfluent aphasia, frontal variant frontal–temporal dementia, and posterior cortical atrophy). However, in these disorders, deficits in semantic memory are much milder than and secondary to deficits in other cognitive difficulties, such as verbal production, executive functions, and visual processing, respectively.

Neuroimaging of Semantic Memory

Studies of patients with selective brain damage and selective behavioral deficits implicate different neuroanatomic substrates in episodic and semantic memory. For example, individuals with amnesia (severely impaired episodic memory) due to hippocampal damage typically have intact semantic memory. Semantic dementia patients with atrophy of the anterior and inferolateral temporal lobes show the opposite pattern of abilities. Taken together, these observations support a double dissociation between the hippocampus and anterior, lateral temporal lobe for episodic and semantic memory, respectively. Recently, the focus of semantic memory research has transitioned from behavioral studies to neuroimaging studies. Such research suggests that semantic (gist) memory becomes established in the neocortex over time.

Just as memory has been subdivided into multiple memory systems, neuroimaging studies suggest that semantic knowledge itself is not a unitary system. For example, visual semantic knowledge can be subdivided into types of visual information such as color, form, motion, and size. Each type of information is associated with activation in different brain regions: retrieval of knowledge of color and form associated with activation of left or bilateral ventral temporal cortex; knowledge of motion with activation of left lateral temporal cortex; and knowledge of size with parietal cortex activation (Thompson-Schill, 2003). Nonperceptual (verbal) conceptual knowledge is associated with activation of anterior temporal cortex, and manipulation knowledge is associated with activation of premotor cortex. Similarly, functional neuroimaging studies reveal highly specific activations corresponding to various task features including modality

specificity, attribute specificity, and category specificity (e.g., medial–occipital–cortical activation in response to animals versus lateral–temporal–premotor activation for tools). Moreover, activation shifts anteriorly during semantic retrieval relative to perception, which suggests a distinction between semantic processing (semantic retrieval) and sensorimotor processing (perception).

Despite the findings of highly specific activations corresponding to various task features, there is some evidence to implicate left prefrontal cortex and/or ventral temporal cortex (either left or bilateral) in a general-purpose semantic system involved in the storage or retrieval of semantic knowledge. Such a system is not amodal; instead, it appears to be a highly distributed system or network of semantic representations whose interactions result in conceptual knowledge spanning different attributes, categories, and modalities. Notably, the left-inferior frontal gyrus appears to be involved in a general-purpose selection mechanism that is useful but not necessary for semantic retrieval.

Future Directions

A number of controversies in the semantic memory research literature remain to be resolved. One of the biggest controversies pits two theories against each other. Adherents of the amodal view of semantic memory hold that there is a central semantic knowledge system that integrates diverse semantic representations to form a coherent conceptual knowledge base. This view is supported by behavioral findings involving patients with semantic dementia. The contrasting theory, supported by anatomic distinctions revealed by recent neuroimaging findings, maintains that semantic representations are distributed across a widely dispersed semantic network involving a number of cortical brain regions. Coherent conceptual knowledge results from extensive interactions among these semantic representations.

Most studies of category specificity to date have been verbal. Thus, future studies are needed to determine the degree to which category-specific activations truly reflect the organization of semantic rather than lexical representations. This question is part of the broader question involving the relationship between semantic knowledge and language. Similarly, neuroimaging studies of semantic memory consistently reveal left-hemisphere activation. Thus, future studies are needed to explore the role of the right hemisphere in semantic processing.

Cross References

- ▶ Alzheimer's Disease
- ▶ Autobiographical Memory
- ▶ Dementia with Lewy Bodies
- ▶ Episodic Memory
- ▶ Semantic Dementia
- ▶ Semantic Fluency

References and Readings

Balota, D. A., & Coane, J. H. (2008). Semantic memory. In J. H. Byrne (Ed.), *Learning and memory: A comprehensive reference* (Vol. 2, pp. 511–534). Amsterdam: Elsevier.

Chang, T. M. (1986). Semantic memory: Facts and models. *Psychological Bulletin, 99*, 199–220.

Collins, A. M., & Loftus, E. F. (1975). A spreading-activation theory of semantic processing. *Psychological Review, 82*, 407–428.

Hodges, J. R., & Patterson, K. (1995). Is semantic memory consistently impaired early in the course of Alzheimer's disease? Neuroanatomical and diagnostic implications. *Neuropsychologia, 33*(4), 441–459.

Hodges, J. R., & Patterson, K. (2007). Semantic dementia: A unique clinicopathological syndrome. *Lancet Neurology, 6*, 1004–1014.

Howard, D., & Patterson, K. (1992). *Pyramids and palm trees.* Bury St. Edmunds, England: Thames Valley Test Company.

Kaplan, E. F., Goodglass, H., & Weintraub, S. (2001). *The Boston naming test* (2nd ed.). Philadelphia: Lippincott Williams & Wilkins.

McClelland, J. L., & Rumelhart, D. E. (1985). Distributed memory and the representation of general and specific information. *Journal of Experimental Psychology: General, 114*, 159–188.

Ober, B. A., & Shenaut, G. K. (2006). Semantic memory. In M. J. Traxler & M. A. Gernsbacher (Eds.), *Handbook of psycholinguistics* (2nd ed., pp. 403–453). Amsterdam: Elsevier.

Quillian, M. R. (1968). Semantic memory. In M. Minsky (Ed.), *Semantic information processing* (pp. 216–270). Cambridge, MA: MIT Press.

Rogers, T. T., Ivanoiu, A., Patterson, K., & Hodges, J. R. (2006). Semantic memory in Alzheimer's disease and the frontotemporal dementias: A longitudinal study of 236 patients. *Neuropsychology, 20*, 319–335.

Schacter, D. L., & Tulving, E. (Eds.). (1994). *Memory systems.* Cambridge, MA: MIT Press.

Smith, E. E., Shoben, E. J., & Rips, L. J. (1974). Structure and process in semantic memory: A featural model for semantic decision. *Psychological Review, 81*, 214–241.

Thompson-Schill, S. L. (2003). Neuroimaging studies of semantic memory: Inferring "how" from "where." *Neuropsychologia, 41*, 280–292.

Tulving, E. (1972). Semantic memory. In E. Tulving & W. Donaldson (Eds.), *Organization of memory.* New York: Academic Press.

Tulving, E. (1983). *Elements of episodic memory.* Oxford, United Kingdom: Clarendon Press.

Wechsler, D. (1997). *WAIS-III.* San Antonio, TX: The Psychological Corporation.

Westmacott, R., Black, S. E., Freedman, M., & Moscovitch, M. (2003). The contribution of autobiographical significance to semantic memory: Evidence from Alzheimer's disease, semantic dementia, and amnesia. *Neuropsychologia, 42*, 25–48.

Semantic Paraphasia

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Synonyms

Meaning-based lexical errors; Semantic errors; Semantic word substitutions; Word-level errors

Definition

The substitution of one full word for another on the basis of a meaning relation between the two.

Historical Background

Semantic word errors have been recorded in the study of aphasia practically since its first case descriptions several hundred years ago. All the literature in the history of the study of aphasia makes this observation, although the linguistic characterizations were scant. Johann Gesner in 1770 is credited with the first in-depth description of semantic paraphasias in fluent aphasia on the basis of association psychological theory (Benton, 2000).

Current Knowledge

Linguistic Structure

To begin with, we must outline the general typology of word connectivity, or how they are associated one with the other. The principal tenets of word relatedness stem from the centuries-old "association psychology," which has its prodromes in the writings of Aristotle (Buckingham, 2002). Words are associated in several ways; they may share features of meaning. In this case, the metric is *similarity*. Words may also appear in a spatial contiguity or in a functional continuity. That is words may appear in close order often enough to become connected ("pins and needles"; "bread and butter") or more often they may function together in propositions. Noun arguments may occur together around certain verbs, such as "paper" and "scissors" with the verb "to cut." Equally, a noun may occur with high frequency around a specific verb, such as "song" with verb "sing," or in this case with the necessary agent, the singer. The important characteristic of this type of semantic relation is referred to by Aristotle and down through the centuries as *contiguity*. Along these two lines of association, we fit metaphor to similarity and metonymy to contiguity. When we say metaphorically that the Tennessee Walker is the Rolls Royce of horses, then we point to the similarities between that kind of horse and that kind of car. The many forms of metonymic associations between words have to do with the context of togetherness. Here, a word like "president" associates with "white house," or "popcorn" with "bag," and in fact one can refer to popcorn in saying "I ate the whole bag," not unlike "I drank the whole bottle." As Aristotle points out, contiguous associative linkages are much more frequent in language than are similarity linkages. We will limit our discussion here to these two sorts of semantic associations.

Another way in which we see connectivity among words is through hierarchical architectures of words referred to as *hyponymy*. Here, we deal with an organization system of word categorization, where similarity is displayed in hierarchical fashion (this sentence seems to me to be redundant). For example, living things subsume plants and animals, animal subsumes a division of [+/− human], and so on. Food subcategorizes into several subtypes, one of which would be fruit, and within fruit would be exemplars such as apple, orange, grape, and banana. Again, here the metric would be one of shared features of meaning, and a grouping of this kind (the types of fruits) is referred to as *co-hyponymic*; those four words are co-hyponyms, and in many cases, such co-hyponyms are learned early by children and are often referred to as *basic level* word. Often, a *prototype* will be established for the category, in this case that of fruit. Many semantic paraphasias involve substitutions among co-hyponyms.

One very interesting and important observation from the literature of semantic substitution errors is that rarely if ever do we see a one word substitution where for a co-hyponym the speaker substitutes the higher category word. Only when a speaker offers a definition of a hyponym not retrievable do we observe the hypernymic word. This is referred to as "the problem of the hypernym." It is rare for the word "animal" to be substituted for "horse" as a one word error.

Neuropsychological Structure

Now, how do the above word associations reveal themselves in semantic paraphasia? The literature breaks down

neuropsychologically into models of function only and models that try to map the language functions to brain regions; with language, the brain regions are most often in the dominant left hemisphere, further narrowed to the perisylvian regions in the left.

In a major study of semantic paraphasia (1990), A. Caramazza and A. Hillis present a paradigm analysis in terms of a functional model, where a disrupted element results from a "functional lesion" (1990, p. 98). It is still the case that many experimental psychologists work at a remove from the physical systems and in so doing do not flinch from the notion of a functional lesion. Present day connectionist modeling does precisely this. In the Caramazza and Hillis (1990) paper, a major type of patient with semantic paraphasias was the one who when searching for word form representation for spoken output produced many meaning-based errors, but nevertheless understood nonverbally all meaning relation. Errors in naming were numerous, but the patients easily displayed by pointing that they knew associations between printed words and pictures. The point here is that underlying meaning structural representation does not *cause* the error. Rather, the semantic paraphasias come about by the nature of the lexical search process itself.

A crucial aspect of searching the lexicon for a word is the manipulation of meaning properties that are located elsewhere. Accordingly, it appears that meaning is distributed through language and is used or accessed at different input/output modalities. The patients in the 1990 (p. 102) study made either "semantic coordinate errors" (= similarity metric) and what they called "associated names" (= contiguity metric). Rarely in reality do we observe patients who exclusively make one or the other type of semantic paraphasia, although there is some discussion of this distinction still (Buckingham, to appear). The lexical system model in Caramazza and Hillis (1990) had two output buffers, phonological and orthographic and both could malfunction in a modular sense with no disruption in the lexical/semantic system "above" them. The output problems were instantiated by semantic paraphasia of different types, including the provision of definitional responses that the authors called "circumlocution." These patients, however, had no disruption at all in the semantic component of this model. The model however had, in other studies, described patients who had overarching semantic problems as well. Those patients produced semantic paraphasias of all types but failed in the nonverbal modalities as well, having trouble as well with word-picture matching verification tasks that did not require speech or written output.

Neuroanatomical Structure

Nineteen years later, a follow-up study by Cloutman et al. (2009) inserted the phrase "in the brain" into the 1990 title, and thus mapped that earlier functional model as best they could to various left posterior brain regions, mostly in the temporal lobe. Phonemic paraphasias, both "coordinate" (similarity) and "associative" (contiguity) were recorded for a large series of patients with stroke, while undergoing perfusion-weighted imaging. Correlations of location of tissue hypoperfusion with behavioral responses of verbal output naming as well as nonverbal word-picture matching verification were calculated. Three left posterior regions were distinguished for different arrays of oral naming *with* semantic errors in nonverbal comprehension and *without* any disruption of nonverbal comprehension at all. We recall that earlier purely functional studies such as that of Caramazza and Hillis (1990) distinguished two such groupings.

First observed were the patients with hypoperfusion in the region of BA 22, or Wernicke's area in left superior temporal lobe, occasionally including the supramarginal and angular gyri, BA 40 and 39, respectively. Patients with cortical dysfunction here produced semantic paraphasias of all types in oral naming and had severe semantic disruption on nonverbal comprehension testing. The second cortical grouping had hypoperfusion in and around BA 37, which comprises the fusiform (spindle-like) gyrus in posterior/middle temporal lobe, extending to anterior occipital regions. Here, the finding was that patients produced numerous semantic paraphasias, again on the bases of similarity and contiguity. Patients in this cortical hypoperfusion category had no semantic errors in nonverbal comprehension tests of word-picture verification matching.

Rather than an overall semantic level involvement of the Wernicke group, the fusiform gyrus group produced semantic paraphasias as a result of impaired access to lexical representations for oral output exclusively. Patients in this group mirrored the two studied in Caramazza and Hillis (1990), where the functional disruption was confined to the "phonological output lexicon." At times, orthographic output will show semantic paraphasia, and at times with certain acquired dyslexias, deep dyslexic reading errors show semantic paraphasic production. Most of these studies focus upon spoken output on naming testing. In general, these patients produce their semantic paraphasias while being in the "tip-of-the-tongue" state; they "know" the meaning, but their search computations often produce semantic paraphasia.

The third grouping comprises the left inferior temporal lobe, BA 21, and the anterior temporal region, BA 38,

at the temporal pole. This group, similar to the Wernicke's patients, produces semantic paraphasias and errors in nonverbal comprehension. This group differs, however, from patients with hypoperfusion in BA 22, because these basal temporal lobe regions are necessary for semantic memory. Tissue dysfunction here causes impoverishment of semantic features themselves, thereby compromising a most important content of semantic representation. Although this inferior/anterior temporal lobe grouping is unilateral and does not display agnosia, these patients will confuse the verb "to milk" with its typical argument "cow," and speak of milking a horse, etc. Bilateral tissue dysfunction in these regions is often what is involved in semantic dementia, where more bizarre semantic output errors are seen.

Future Directions

Semantic paraphasia is therefore shown to result from distinct lesion arrays in the left temporal lobe, and is correlated with different connectivities to semantic representation. Wernicke's aphasics are claimed to have phonemic paraphasia, because BA 22 serves as a major linkage system between lexical production in distinct modalities. Fusiform gyrus hypoperfusion simply causes semantic paraphasia as part and parcel word access computational derailment. The phonemic paraphasia that stems from tissue dysfunction in left anterior temporal cortex is a result of direct disintegration of elements of semantic memory itself, which is not unreasonable due to the proximity of amygdaloid structures subjacent to BA 38. Future research will do doubt clarify many of the complexities in the neurolinguistics of semantic paraphasic production in brain damage.

In summary, the definition of semantic paraphasia must include three architectures: the linguistic structure of word association, the functional account of the neuropsychological input-output modality behaviors and, as we have now seen, a closer and more meaningful description of the anatomical regions that, when lesioned, lead to different correlations between oral output naming and semantic disruption.

Cross References

► Literal Paraphasia
► Semantic Dementia
► Semantic Fluency
► Semantic Memory
► Semantic Pragmatic Disorder (SPD)

References and Readings

Benton, A. (2000). *Exploring the history of neuropsychology: Selected papers* (pp. 175–182). Oxford, UK: Oxford University Press.

Buckingham, H. (2002). The roots and amalgams of connectionism. In R. G. Daniloff (ed.), *Connectionist approaches to clinical problems in speech and language* (pp. 265–311). Mahwah, NJ: Lawrence Erlbaum Associates.

Buckingham, H. (2010). Aristotle's functional association psychology. The syntagmatic and the paradigmatic axes in the neurolinguistics of Roman Jakobson and Alexander Luria: An anatomical and functional quagmire. A response to Alfredo Ardila's reinterpretations and reclassifications. *Aphasiology, 24*(3), 395–403.

Caramazza, A., & Hillis, A. (1990). Where do semantic errors come from? *Cortex, 26*(1), 95–122.

Cloutman, L., et al. (2009). Where (in the brain) do semantic errors come from? *Cortex, 45*(5), 641–649.

Semantic Pragmatic Deficit Disorder (SPDD)

► Semantic Pragmatic Disorder (SPD)

Semantic Pragmatic Disorder (SPD)

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Synonyms

Pragmatic language impairment (PLI); Semantic pragmatic deficit disorder (SPDD); Semantic pragmatic language disorder (SPLD)

Short Description or Definition

Semantic pragmatic disorder (SPD) also known as pragmatic language impairment (PLI) and semantic pragmatic language disorder (SPLD) is a developmental disorder that involves impairment in a person's ability to use language effectively during social interactions (Bates, 1976). Individuals with SPD lack the knowledge of *when* to say *what* and *how much* to *whom* (Hymes, 1971).

Categorization

Some professionals believe that SPD is best understood as a specific language impairment (SLI) while others view it as a *communication impairment* possibly related to Autism Spectrum Disorders.

Epidemiology

Determining the incidence of SPD is complicated by the above-noted lack of consensus about whether SPD is a distinct diagnosis or is a component of another diagnostic category (i.e., autism spectrum disorder) (Prutting & Kirchner, 1987). In addition, that most assessment of SPD is nonstandardized and observational (Philosky & Hepburn, 2010) contributes to the difficulty in determining true incidence rates. Since the rates of autism spectrum disorders have risen dramatically over the past several decades, an increased rate of SPD, or at least an increased awareness of pragmatic language issues, seems likely.

Natural History, Prognostic Factors, and Outcomes

Semantic pragmatic deficit syndrome was first introduced as a language disorder by Rapin and Allen in 1983 and was subsequently renamed SPD by Bishop and Rosenblum (1987). While some view SPD as an SLI, others have suggested that these difficulties constitute a possible subtype of autism spectrum disorders (e.g., Boucher, 1998). It remains in dispute whether SPD is a separate clinical category as proposed by Botting (1998), an area of language deficit secondary to SLI, an overlapping characteristic of autism spectrum disorders (Bishop, 2000, p.111), or a separate PLI (Bishop, 2000; Botting & Conti-Ramsden, 1999, 2003).

Neuropsychology and Psychology of SPD

A number of functional impairments are associated with SPD including awareness of, comprehension of, and appropriate application of nonverbal communication (e.g., eye contact, body language, functional gestures, facial expressions, tone of voice, etc.). In addition, comprehension and application of conversational routines and skills are typically impaired (e.g., using greetings and closings, maintaining and shifting eye contact in conversation, initiating conversations, topic maintenance, ability to role play, turn-taking during play or in conversation, or using words for a variety of pragmatic functions such as labeling, requesting, answering, and gaining attention). Code switching or changing register as a function of communication audience can be problematic. Comprehension and application of abstract thought (e.g., slang terminology and humor) are also often affected.

With respect to the neural correlates of SPD, the underlying assumption is that the neural correlates associated with normal language development are involved. Comprehensive summaries of such correlates can be found elsewhere (e.g. Bates, Thal, Finaly, & Clancy, 2003).

In children with autism spectrum disorders, recent data on the neural correlates of pragmatic language comprehension have implicated increased activation in the right inferior frontal gyrus and decreased activation in the right ventral medial prefrontal cortex including the right anterior cingulate cortex (Tesink et al., 2009).

Evaluation

Like other aspects of language impairment, SPD is evaluated by speech/language pathologists and psychologists using standardized assessment measures. However, given the complexity of pragmatic language behaviors, assessment of pragmatics can be difficult, leaving many clinicians to rely on nonstandardized, observational methods. This can hamper identification of problems and create challenges in determining service eligibility (American Speech-Language-Hearing Association, 2006; Olswang, Coggins, & Timler, 2001; Young, Diehl, Morris, Hyman, & Bennetto, 2005). Often informal measures collected by experienced clinicians such as language samples, observations, structured play interactions, parent and teacher interviews, and rating scales assist in making the diagnosis of SPD. Below are some examples of standardized assessment tools frequently utilized when providers are concerned about the possible presence of semantic pragmatic language issues.

Test of Pragmatic Language (TOPL). The TOPL provides formal assessment of the social areas of language, providing an in-depth screening of the effectiveness and appropriateness of a student's pragmatic (social) language skills. It is designed for children 5–13 years of age. This instrument is comprised of tasks read by the examiner and answered by the child; administration typically takes between 30 and 45 min. It generates four scores: raw scores, percentiles, quotients, and age equivalents. Diana Phelps-Terasaki & Trisha Phelps-Gunn (1992). Austin, TX: Pro-Ed.

The Clinical Evaluation of Language Fundamentals – Fourth Edition (CELF-4): As part of the CELF-4, the pragmatics profile is a supplementary criterion referenced checklist. This checklist can provide additional information about the student's overall pragmatic development compared to typical skills used in social and school interactions. The evaluator can then elicit further information from the informant (usually a parent or teacher) familiar with the student's social behaviors and classroom interaction skills (Semel, Wiig, & Secord, 2004). Clinical Evaluation of Language Fundamentals-4. Orlando, FL: Harcourt Assessment.

Children's Communication Checklist – 2 U.S. Edition was developed as a parent or caregiver rating scale. It is comprised of 70 questions and is designed for children ages 4–16 years, 11 months of age. It is a screening measure that can be used to determine the need for further assessment of speech, syntax, semantics, coherence, pragmatic initiation, scripted language, context, nonverbal communication, social relations, and interests. Children's Communication Checklist – 2 Bishop, D., (2006). Children's Communication Checklist-2. Orlando, FL: Harcourt Assessment.

Treatment

No one treatment is known to be effective for all individuals demonstrating SPD. Current treatment practices include various forms of social skills training including individual one-on-one therapy and group therapies encompassing discussions, role playing, and problem-solving situations. These interventions target the comprehension of social situations as well as the application of effective language skills within the social situation. In addition, treatments can include peer modeling within the classroom or treatment session, parent education, and involvement. More recent treatment strategies involve video modeling to facilitate skill development. These types of therapies are often provided by speech/language pathologists (SLP), psychologists, licensed counselors, and can be implemented by an individual therapist or counselor or by an interdisciplinary team.

Cross References

- ▶ Asperger's Disorder
- ▶ Autistic Disorder
- ▶ Pervasive Developmental Disorder NOS
- ▶ Social Skills Training
- ▶ Speech-Language Therapy
- ▶ Williams Syndrome

References and Readings

American Speech-Language-Hearing Association. (2006). Guidelines for speech-language pathologists in diagnosis, assessment, and treatment of autism spectrum disorders across the life span. Available from www.asha.org /policy

Bates, E. (1976). *Language and context: The acquisition of pragmatics.* New York: Academic Press.

Bates, E., Thal, D., Finaly, B., & Clancy, B. (2003). Early language development and its neural correlates. In S. Segalowitz (Ed.), *Handbook of neuropsychology* (2nd ed., Vol. 8(Part 2)). Amsterdam: Elsevier.

Bishop, D. V. M. (2000). Pragmatic language impairment: A correlate of SLI, a distinct subgroup or part of the autistic continuum? In D. V. M. Bishop & L. B. Leonard (Eds.), *Speech and language impairments in children: Causes, characteristics, intervention and outcome* (pp. 99–113). Hove: Psychology Press.

Bishop, D. V. M., & Rosenblum, L. (1987). Classification of childhood language disorders. *Clinics in Developmental Medicine, No. 101/102.* London: Mac Keith Press.

Botting, N. (1998). Semantic pragmatic disorder (SPD) as a distinct diagnostic entity: Making sense of the boundaries. *International Journal of Language and Communication Disorders, 33*(1), 87–90.

Botting, N., & Conti-Ramsden, G. (1999). Pragmatic language impairment without autism. *Autism, 3*, 371–396.

Botting, N., & Conti-Ramsden, G. (2003). Autism, primary pragmatic difficulties and specific language impairment: Can we distinguish them using psycholinguistic markers? *Developmental Medicine and Child Neurology, 45*, 515–524.

Boucher, J. (1998). SPD as a distinct diagnostic entity: Logical considerations and directions for future research. *International Journal of Language and Communication disorders, 33*(1), 71–108.

Hymes, D. (1971). On communicative competence. In J. Pride & J. Holmes (Eds.), *Socilinguistics* (pp. 269–293). Baltimore: Penguin.

Kirchner, D., & Prutting, C. (1987). A clinical appraisal of the pragmatic aspect of language. *Journal of Speechand Hearing Disorders, 52*, 105–119.

Rapin, I., & Allen, D. (1983). Developmental language disorders: Nosologic considerations. In U. Kirk (Ed.), *Neuropsychology of language, reading, and spelling* (pp. 155–184). New York: Academic Press.

Tseink, C. M. J. Y., Buitelaar, J. K., Petersson, K. M., van der Gaag, R. J., Kan, C. C., Tendolkar, I. et al. (2009). Neural correlates of pragmatic language comprehension in autism spectrum disorders. *Brain, 132*, 1941–1952.

Semantic Pragmatic Language Disorder (SPLD)

- ▶ Semantic Pragmatic Disorder (SPD)

Semantic Word Substitutions

► Semantic Paraphasia

Semantics

► Vocabulary

Semi-Structured Clinical Interview

► Structured Clinical Interview For DSM-IV (SCID-I/SCID-II)

Senile Amyloid Plaques

► Senile Plaques

Senile Dementia

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Synonyms

Alzheimer's disease; Senility

Short Description or Definition

Dementia is an acquired syndrome consisting of impairment in multiple areas of cognition such as memory, perception, language, executive function, calculation ability, praxis, semantic knowledge, and personality or social behavior (Mendez & Cummings, 2003). Although most definitions recognize memory impairment as the cardinal feature, they are unsuitable for dementing illnesses where other symptoms predominate. To address this concern, many classification systems attempt to provide criteria for specific types of dementia such as Alzheimer's disease, frontotemporal dementia, and others. To meet criteria for dementia, the impairments must not occur exclusively in the context of a delirium, and must represent a decline from previously higher levels of functioning. Additionally, the impairments must be of sufficient severity to cause impairment in social or occupational functioning. Senile dementia generally refers to an onset age greater than or equal to 65. In the late nineteenth and early twentieth centuries, senile dementia was thought to result from atherosclerosis of the brain blood vessels.

Historical Background

Pre-senile Versus Senile Dementia

Senile dementia was already a recognized clinical condition when, in 1906, Alois Alzheimer presented a case study of dementia with *pre-senile* onset, the well-known case of Aguste D. In 1901, the patient was admitted to the hospital. Her symptoms consisted of delusions, memory impairment, apathy, and impairment in performing common tasks of daily living (e.g., cooking). She had difficulty finding objects, no longer understood the concept of money, and became increasingly suspicious of others. Her course was rapid, marked at times by nonsensical speech and periods of agitation and aggression. She was treated with sedatives and frequent therapeutic baths. At the end of her illness, she was bedridden, exhibiting severe deterioration in all cognitive spheres. She died in 1906 after a 5-year course. At autopsy, Alzheimer described generalized cerebral atrophy, atherosclerosis of the cerebral arteries, and neuronal alterations in the cortex that are now recognized as neurofibrillarly tangles and senile plaques (Lage, 2006).

In the 1910 edition of his textbook of psychiatry, Emil Kraepelin distinguished pre-senile dementia from senile dementia, designating the former as Alzheimer's disease (AD). The age (65) to distinguish pre-senile dementia from senile forms was not based on any specific medical criteria, but presumably was selected in accordance to the age of qualification for old age insurance in Germany (Bick, 1994). Although there were occasional reports of Alzheimer's-like brain changes in *senile* dementia, the majority of physicians restricted their search for cases of AD to dementias below age 65 (Lage, 2006). Thus, AD was regarded as the underlying cause of pre-senile dementia, and senile dementia was regarded as a separate disorder.

Similar neuropathological changes link AD and senile dementia

In 1968, Martin Roth, Garry Blessed, and Bernard Tomlinson published a seminal paper reporting that the neuropathological features of senile dementia were practically identical to those of AD, specifically, the presence of senile plaques and neurofibrillary tangles in the brain (Lage, 2006). Thus, the distinction between senile dementia and AD was blurred, and AD was recognized as one of the primary causes of senile dementia.

Current Knowledge

Presently, the term *senile dementia* no longer retains its previous meaning of a dementia caused by atherosclerosis of brain blood vessels. More often than not, it refers to an age of dementia onset in late life, on or after age 65. It does not convey a specific illness. However, as a result of history, many consider the term synonymous with late-onset AD. Because it lacks specificity, the term "senile dementia" is rarely used as a sole diagnosis, unless to designate a dementia of unknown cause (e.g., senile dementia, not otherwise specified). A designation of late- versus early-onset dementia is useful as specific genetic mutations have been associated with early onset forms, such as familial AD.

References and Readings

Bick, K. L. (1994). The early story of Alzheimer's disease. In R. D. Terry, R. Katzman, & K. L. Bick (Eds.), *Alzheimer disease* (pp. 1–8). New York: Raven Press.

Lage, J. M. M. (2006). 100 Years of Alzheimer's disease (1906–2006). *Journal of Alzheimer's Disease, 9*, 15–26.

Mendez, M. F., & Cummings, J. L. (2003). *Dementia: A clinical approach* (3rd ed.). Philadelphia: Butterworth.

Senile Dementia of Lewy Type

► Dementia with Lewy Bodies

Senile Dementia of the Alzheimer's Type

► Alzheimer's Dementia

Senile Plaques

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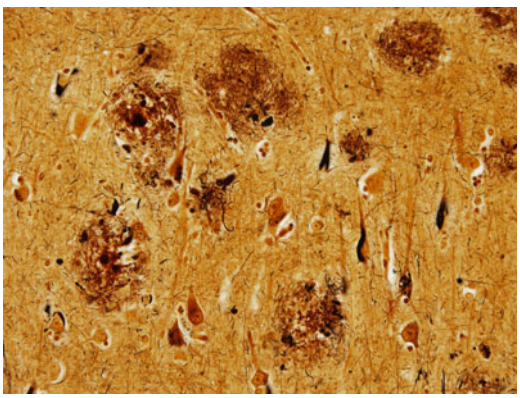
Synonyms

Amyloid plaques; Neuritic plaques; Senile amyloid plaques

Definition

Senile plaques are one of the neuropathological hallmarks of Alzheimer's disease (AD) and are found in the extracellular space between neurons in the brain. Fully formed senile plaques are characterized by a central beta-amyloid core and surrounded by elements of degenerating neurons (dystrophic neurites). Diffuse plaques, by contrast, are widely distributed in the brain, but lack the surrounding neurites. Senile plaque counts, along with neurobrillary tangles, form the basis by which a pathological diagnosis of AD is made. Senile plaques also occur in the brains of elderly individuals without dementia, but are generally of insufficient quantity to meet criteria for a pathological diagnosis of AD (Grabowski & Damasio, 2004; Morris & Nagy, 2004). Figure 1 displays senile plaques in brain.



Senile Plaques. Figure 1 Senile plaques stained with a modified Bielschowsky silver stain (Photo courtesy of Steven S. Chin, M.D., Ph.D., University of Utah Health Sciences Center)

Cross References

- ▶ Alzheimer's Dementia
- ▶ Neurofibrillary Tangles
- ▶ Senile Dementia

References and Readings

Grabowski, T. J., & Damasio, A. R. (2004). Definition, clinical features and neuroanatomical basis of dementia. In M. M. Esiri, V. M.-Y. Lee, & J. Q. Trojanowski (Eds.), *The neuropathology of dementia* (2nd ed., pp. 1–33). Cambridge: Cambridge University Press.

Morris, J. H., & Nagy, Z. (2004). Alzheimer's disease. In M. M. Esiri, V. M.-Y. Lee, & J. Q. Trojanowski (Eds.), *The neuropathology of dementia* (2nd ed., pp. 161–206). Cambridge: Cambridge University Press.

Senility

- ▶ Dementia
- ▶ Senile Dementia

Sensitive Periods

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Synonyms

Critical periods

Definition

The term *sensitive period* refers to the notion that the effects of environmental stimuli on the developing organism are stronger during certain periods of development. The traditional term, "critical period," was based on the assumption that these developmental periods were innate and had a fixed onset and termination. However, more recent research has suggested that they are influenced by environmental factors and are flexible in terms of their beginning and end; thus, the term "sensitive periods" is more commonly preferred (Michel & Taylor, 2005).

Additional distinctions have been made between the terms "sensitive" and "critical" period. Fox (1970) suggested that the term "critical period" be used when a specific environmental stimulus is needed to cue normal development, whereas the term "sensitive period" be used to describe when the development is more easily influenced by a negative environmental event. The relative vulnerability of the developing fetus to teratogens during the first trimester is an example of a sensitive period (Spreen, Risser, & Edgell, 1995). Knudsen (2004) posited that "critical" periods are a specific class of sensitive periods that involve permanent changes in functional brain development.

Historical Background

The concept of a sensitive period for development began with the work of early researchers studying the development of social bonds among animals and humans (Michel & Tyler, 2005). Konrad Lorenz discovered that laboratory-hatched geese would "imprint" on the first moving object they perceived during a specific period of time shortly after birth. Harry Harlow studied the development of social behaviors in rhesus monkeys and found that the negative effects of social deprivation on later social behaviors are especially apparent during the first 3 months of life. Bowlby extended this early work on nonhuman animals and theorized that the period of time from 6 months to 3 years of age is a crucial period for the development of the attachment between the human baby and mother that serves as a basis for the development of later social relationships. Eric Lenneberg studied the development of language in children and proposed that the critical period for language development extended from birth to approximately age 12. Later work following the language development of a girl held in isolation across this age span suggested that this long time frame is more flexible and more consistent with the notion of sensitive periods (Spreen et al., 1995).

Current Knowledge

Various theories have been offered regarding the specific mechanisms underlying sensitive periods in functional brain development. Specifically, Johnson (2005), Thomas

and Johnson (2008) described three competing models. From a *maturational* perspective, functional development occurs through the maturation of specific brain regions that are innately involved with specific functions. Thus, the physical maturation of specific brain regions guides sensitive periods. This maturational perspective fits more closely with the traditional concept of "critical periods" (Stiles, 2008). From a *skill learning* perspective, similar cortical areas are activated during skill learning in both infancy and adulthood, but these areas become less plastic after the skill has been learned. As a result, the sensitive period ends after the specific skill has been mastered. Johnson has proposed an alternative viewpoint, *interactive specialization*, which posits that the interactions between different brain regions are important for functional brain development. Under this theory, the interactions between components of the cerebral network result in more highly specialized brain regions and this specialization results in the termination of a sensitive period.

Although sensitive periods are measured in terms of observable behaviors, recent work has concluded that the effects of environmental stimuli during sensitive periods results in actual changes in neuronal circuitry, brain architecture, and neurochemistry (Knudsen, 2005). Related research has explored the possibility of "reopening" critical periods to possibly increase the effectiveness of rehabilitation after brain injury. This work has suggested some limited potential to increase plasticity after sensitive periods have purportedly ended (Knudsen, 2005).

Cross References

► Plasticity

References and Readings

Fox, M. W. (1970). Overview and critique of stages and periods in canine development. *Developmental Psychobiology, 4*, 37.

Johnson, M. H. (2005). Sensitive periods in functional brain development: Problems and prospects. *Developmental Psychobiology, 46*, 287–292.

Knudsen, E. I. (2004). Sensitive periods in the development of the brain and behaviour. *Journal of Cognitive Neuroscience, 16*, 1412–1425.

Michel, F. T., & Tyler, A. N. (2005). Critical period: A history of the transition from questions of when, to what, to how. *Developmental Psychobiology, 46*, 156–162.

Spreen, O., Risser, A. H., & Edgell, D. (1995). Critical periods, plasticity, and recovery of function. In *Developmental neuropsychology* (pp. 139–155). New York: Oxford University Press.

Stiles, J. (2008). *The fundamentals of brain development* (pp. 313–358). Cambridge, MA: Harvard University Press.

Thomas, M. S. C., & Johnson, M. H. (2008). New advances in understanding sensitive periods in brain development. *Current Directions in Psychological Science, 17*, 1–5.

Sensitivity

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Definition

A clinical outcome statistic concerned with evaluating the accuracy of a test/measure to predict what the test/measure purports to predict. Sensitivity refers to the *true positive* rate of a test/measure. It answers the question, knowing that a person belongs to a certain group (e.g., patients exhibiting poor effort), what percentage of these people will be correctly identified as belonging to that group by a particular test/measure (e.g., ► Test of Memory Malingering [TOMM]). For example, a sensitivity of 0.89 indicates that 89% of the people in the poor effort group were correctly identified by the TOMM. Sensitivity is calculated by dividing the number of people accurately identified by the TOMM as providing poor effort (i.e., Test Positive, Target Behavior Present) by the number of people who belong in the poor effort group (Total Behavior Present).

Cross References

► Negative Predictive Power
► Outcome, Outcome Measurement
► Positive Predictive Power
► Receiver-Operating Characteristics
► Specificity

References and Readings

Retzlaff, P. D., & Gibertini, M. (1994). Neuropsychometric issues and problems. In R. D. Vanderploeg (Ed.), *Clinician's guide to neuropsychological assessment* (pp. 195–209). Hillsdale: Erlbaum.

Slick, D. J. (2006). Psychometrics in neuropsychological assessment. In E. Strauss, E. M. S. Sherman, & O. Spreen (Eds.), *A compendium of neuropsychological tests: Administration, norms, and commentary* (pp. 3–43). New York: Oxford University Press.

Sensorimotor Assessment

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Synonyms

Motor examination; Sensory examination

Definition

Sensory Examination

The examination of the sensory system consists of testing multiple sensory modalities. These consist of light touch, pain, proprioception, two-point discrimination, vibration, temperature, and stereognosis. The patient's presentation and complaints should guide which modalities are tested as well as which parts of the body are tested.

The testing should follow a dermatomal distribution and the patient should have their eyes closed to make testing more reliable.

To test light-touch sensation, a wisp of cotton wool should be made to touch the surface of the skin and the patient should respond when the stimulus is felt.

Pain sensation is assessed through the use of a safety pin which has both sharp and dull edges. The sharp and dull edges are placed on the skin in a random order and the patient should report whether the stimulus is felt as "sharp," "dull," or "unable to tell." This modality assesses not only an acknowledgment of feeling the stimulus, but also the painful quality of the stimulus.

Proprioception is a sense of both position and movement of the body and its limb. It is independent of vision and is obtained from sensory nerve terminals in muscles and tendons as well as the joint capsule. To test proprioception, the examiner should grip the sides of a particular joint (so as not to let the pressure on the joint influence the patient's perception of movement) and then move the joint either up or down. The patient should then be asked to report the direction of movement. It is best to start distally and move proximally until correct responses are obtained. For example, if the responses are inaccurate at the great toe, testing should be performed at the ankle, then at the knee, and so forth.

To test two-point discrimination, two stimuli are placed on the skin with equal, gentle pressure. For this purpose, one may use a pair of compasses with gradations in centimeters indicating separation of the tips, or calipers. The patient should report when the stimulus is felt, as well as whether one or two stimuli are felt. As a general rule, on the finger tips, one should be able to detect a separation of approximately 3 mm, while on the palm it is 1 cm, and on the sole of the foot 3 cm.

A 128-Hz tuning fork is used to test the vibration sense. The vibrating tuning fork is placed on the distal pad or the joint of the finger or toe and the patient is asked to report whether the vibration is felt. As with proprioceptive testing, it is best to start distally and work proximally.

To test temperature sensation, it is necessary to have one object which is cold and another which is warm. Two metal test tubes are commonly used, one filled with ice chips and one filled with hot water. The two test tubes are then placed on the skin in random order on comparable parts of the two sides of the body. The patient should be asked to distinguish between hot and cold.

Stereognosis is the appreciation of the form of an object by means of touch. To test this sensation, the examiner will use items of differing size and shape such as keys, coins, etc. The objects are placed in the patient's hands and they are asked to identify the object.

Motor examination

The examination of the motor system consists of evaluating the appearance of muscles as well as muscle tone and power.

Muscle bulk should be assessed and areas of atrophy or wasting noted. The muscle should also be visually examined for spontaneous contractions or fasciculations. Muscles may hypertrophy secondary to fat or connective tissue. It is important to compare muscles from side to side in order to note the subtle differences.

For a screening assessment of tone, the examiner should examine flexion and extension at the elbow, pronation, and supination of the forearm and flexion and extension at the knee. Tone may be increased, decreased, or normal. Testing should be done at a variety of speeds in order to distinguish spasticity, which is dependent on velocity, from rigidity, which is independent of velocity. Both spasticity and rigidity are examples of increased tone or hypertonia.

Strength is tested by having the patient resist your force as you attempt to move the body part against the direction of pull of the muscle you are evaluating. This is graded on a graded scale of 0–5, with "0" representing no visible contraction and "5" being normal.

0. No movement
1. Palpable movement or visible contraction
2. Active movement through full range of motion with gravity eliminated
3. Active movement through full range of motion against gravity
4. Active movement against moderate resistance through full range of motion
5. Normal strength based on age, sex, and body habitus

The muscles that are tested depend on the patient's presentation; however, a screening muscle strength exam includes muscles representative of spinal cord levels C5 through T1, and L2-S1. These are C5-elbow flexion, C6-wrist extension, C7-elbow extension, C8-finger flexion, T1- finger abduction, L2- hip flexion, L3- knee extension, L4-ankle dorsiflexion, L5-great toe extension, and S1-ankle plantarflexion.

More extensive muscle strength testing should be done if weakness is detected, and as always, the exam should be done bilaterally to detect the subtle differences.

Cross References

► Neurologic Examination
► Physiatric Assessment

References and Readings

Epstein, O., Perkin, G. D., de Bono, D. P., & Cookson, J. M. D. (Eds.), (1997). The nervous system. In *Clinical examination* (2nd ed., pp. 351–353). London: Mosby.

Image of Dermatomes: http://www.nim.nih.gov/midlineplus/ency/image/1806.htm

Nolan, M. F. (1983). Limits of two-point discrimination ability in the lower limb in young adult men and women. *Physical Therapy, 63*, 1424.

Nolan, M. F. (1985). Quantitative measure of cutaneous sensation: Two-point discrimination values for the face and trunk. *Physical Therapy 65*, 181.

Nolan, M. G. (1982). Two-point discrimination assessment in the upper limb in young adult men and women. *Physical Therapy, 62* (7), 965–9.

O'Sullivan, S. B., & Schmidt, T. J. (1988). Motor control assessment. In *Physical rehabilitation: Assessment and treatment* (2nd ed., p 140). Philadelphia: F.A. Davis Co.

O'Sullivan, S. B., & Schmidt, T. J. (Eds.), (1988). Sensory assessment. In *Physical rehabilitation: Assessment and treatment* (2nd ed., pp. 79–93). Philadelphia: F.A. Davis Co.

Swenson, R. (2008). Motor system examination. http://www.dartmouth.edu/~dons/part1/chapter_10.html. Accessed 30 August, 2007.

Sensory

► Afferent

Sensory Aphasia

► Fluent Aphasia
► Wernicke's Aphasia

Sensory Ataxia

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Definition

Sensory ataxia is a problem of incoordination resulting from a disruption of somatosensory feedback.

Current Knowledge

Sensory ataxia is both a sign and a symptom in neurology. Although one may not always be consciously aware of it, motor activities – especially those involving the coordination of either upper or lower extremities – rely heavily on sensory input, especially kinesthetic and proprioceptive feedback. While visual feedback (looking at the limbs) during movements can be of some assistance, vision alone is inadequate in many situations. Thus, sensory ataxia is the result of disruption of neuronal pathways that interfere with proprioception, particularly lesions involving peripheral nerves, dorsal nerve roots, posterior (dorsal) columns, or the medial lemniscus. Cortical (parietal) lesions may produce similar deficits, but less commonly. Sensory ataxia is distinguished from other types of ataxia by marked worsening of coordination when the eyes are closed. Patients with this condition often walk with a

broad-based gait and stand with their legs apart. They may complain that they find it difficult or near impossible to walk in a darkened room or losing their balance when closing their eyes in the shower or removing clothes over their head. A routine test for this condition is to ask the patient to stand with their feet together and then continue to do so after closing their eyes. Marked difficulty in doing so (a "positive Romberg") is suggestive of loss of proprioceptive feedback. Asking a patient to flex their arms at their elbows and rapidly rotate their closed fists around each other (with their eyes closed) may demonstrate upper limb ataxia (fists may keep bumping into one another).

Cross References

► Ataxia
► Proprioception

References and Readings

Mendoza, J. E., & Foundas, A. L. (2008). The somatosensory systems. *Clinical neuroanatomy: A behavioral approach*(pp. 23–47). New York: Springer.

Sobue, G. (1998). Acute and chronic sensory neuronopathy. In N. Latov, J. H. Wokke, & J. J. Kelly (Eds.), *Immunological and infectious diseases of the peripheral nerves* (p. 129). New York: Cambridge University Press.

Sensory Examination

► Sensorimotor Assessment

Sensory Extinction

► Extinction

Sensory Inhibition

► Lateral Inhibition

Sensory Integration-C/APD (SI-C/APD)

► Central Auditory Processing Disorder

Sensory Nerve Roots

► Dorsal Nerve Roots

Sensory Perceptual Exam

► Reitan–Klove Sensory Perceptual Examination

Sentence Completion

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Synonyms

Garden-path sentences; Hayling sentence completion task; Incomplete sentences blank

Definition

Sentence completion tests require an individual to provide the final word to a series of sentences. In personality assessments, these are used to examine for themes in personality, psychopathology, and aptitude. Sentence completion tests used in research, however, often require individuals to suppress their initial response while providing either a given response or a nonsense response as a measure of inhibition.

Current Knowledge

Sentence completion tests have long been used in personality assessment to examine personality traits, to give insight into psychiatric functioning, to establish rapport, or in research. Holaday and colleagues (Holaday, Smith, & Sherry, 2000) provide a summary and review of the literature regarding uses of sentence completion measures for personality assessment.

One of the earliest measures was the incomplete sentences blank (Rotter, 1951), which has subsequently been

updated (Rotter, Lah, & Raffert, 1992). The measure was developed to screen high school and college students for emotional difficulties. Respondents complete 40 sentence stems; responses are scored as positive, neutral, or negative, and a composite score is calculated by summing all item scores. Low scores indicate positive adjustment, while high scores are believed to reflect psychosocial conflict. This measure correlates reasonably well with other measures of personality (e.g., MMPI-A; Weiss, Toolis, & Cerankosky, 2008). It is among the most widely used measures for personality assessment (Holaday et al., 2000), yet there is limited empirical evidence of its applicability with neuropsychological populations. Respondents reported using the incomplete sentences blank with children (18 percent of respondents), adolescents (32 percent), and adults (47 percent); all well higher than any other sentence completion test included in the survey or submitted by respondents. The authors did not speculate as to why the Rotter test was the most used nor did they discuss why the Rotter test was popular with children and adults when it was developed for adolescents and college age students. While use of sentence completion tests as a personality measure may be relevant to the field of neuropsychology, specific sentence completion tests have also been utilized in more traditional neuropsychological assessments and research.

Garden-Path Sentences

The Garden-Path Sentences Test (Hartman & Hasher, 1991) was developed to examine inhibitory efficiency. In this test, individuals are initially asked to complete a series of 28 sentences, and are then provided with different final words to half of the sentences. They subsequently complete a second series of sentences, containing both the original 28 sentences and a number of new sentences. For 14 of the original 28 sentences, the participants are asked to inhibit their original response and provide the word given to them by the examiner. Participant responses are examined on the original 28 sentences, focusing specifically the 14 sentences for which a different word ending was provided. From these responses, participant inhibitory control is computed. Research has demonstrated that this test measures inhibitory efficiency as opposed to simply measuring "access" to relevant as opposed to nonrelevant information (May et al., 1999), and differentiates well between those with inhibitory control and those with poor inhibitory control (Hasher, Quig, & May, 1997) or with induced impairments (e.g., by sleep deprivation, May & Hasher, 1998).

Hayling Sentence Completion Test

The Hayling Sentence Completion Test (Burgess & Shallice, 1996) consists of 30 sentence stems that the participant is required to complete with the first word that comes to their mind. Their responses are logged, and the same sentence stems are again provided in the second condition, in which the participant is to provide a word that does not make sense in the sentence stem. In other words, they are asked to inhibit the prepotent (initial) response and provide a novel word. Response latency for each trial is recorded, and error scores are also computed by raters for the second set of sentences. Errors are rated either a 3 (direct completion that makes sense), 1 (completion that is not common but makes sense), or 0 (correct nonsense completion). The sentences used in the Hayling test were taken from the sentence context study completed by Bloom and Fischler (1980); the test is available from Thames Valley Test (Burgess & Shallice, 1997).

The Hayling Sentence Completion Test has been used in numerous studies of executive functioning, including those examining aging effects (e.g., de Frias, Dixon, & Strauss, 2006), schizophrenia (e.g., Turnbull, Evans, Kemish, Park, & Bowman, 2006), Alzheimer's disease (e.g., Belleville, Chertkow, & Gauthier, 2007), alcoholism (Noel, Bechara, Dan, Hanak, & Verbanck, 2007), brain damage (e.g., Draper & Ponsford, 2008), PTSD (Koso & Hansen, 2006), and attention-deficit/hyperactivity disorder (ADHD) (Clark, Prior, & Kinsella, 2000). All of the aforementioned studies have shown the Hayling Sentence Completion Test to reliably differentiate between individuals with impaired executive functioning (inhibition) and those without. Imaging tests have shown that completion of the second condition of the Hayling test is associated with increased activation in left frontal regions (Nathaniel-James, Fletcher, & Frith, 1997). In addition, this measure has demonstrated a moderate correlation with other ratings of disability in a study of ecological validity (Odhuba, van den Broek, & Johns, 2005).

Cross References

- ▶ Attention
- ▶ Disinhibition
- ▶ Executive Functioning
- ▶ Inhibition

References and Readings

Belleville, S., Chertkow, H., & Gauthier, S. (2007). Working memory and control of attention in persons with Alzheimer's disease and mild cognitive impairment. *Neuropsychology, 21*(4), 458–469.

Bloom, P. A., & Fischler, I. (1980). Completion norms for 329 sentence contexts. *Memory & Cognition, 8*(6), 631–642.

Burgess, P. W., & Shallice, T. (1996). Response suppression, initiation, and strategy use following frontal lobe lesions. *Neuropsychologia, 34*(4), 263–273.

Burgess, P. W., & Shallice, T. (1997). *The hayling and brixton tests (Two tests of dysexecutive syndrome)*. Bury St. Edmonds, England: Thames Valley Test.

Clark, C., Prior, M., & Kinsella, G. J. (2000). Do executive function deficits differentiate between adolescents with ADHD and oppositional defiant/conduct disorder? A neuropsychological study using the Six Elements Test and Hayling Sentence Completion Test. *Journal of Abnormal Child Psychology, 28*(5), 403–414.

de Frias, C. M., Dixon, R. A., & Strauss, E. (2006). Structure of four executive functioning tests in healthy older adults. *Neuropsychology, 20*(2), 206–214.

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

Hartman, M., & Hasher, L. (1991). Aging and suppression: Memory for previously relevant information. *Psychology & Aging, 6*, 587–594.

Hasher, L., Quig, M. B., & May, C. P. (1997). Inhibitory control over no-longer relevant information: Adult age differences. *Memory & Cognition, 25*(3), 286–295.

Holaday, M., Smith, D. A., & Sherry, A. (2000). Sentence completion tests: A review of the literature and results of a survey of members of the Society for Personality Assessment. *Journal of Personality Assessment, 74*(3), 371–383.

Koso, M., & Hansen, S. (2006). Executive function and memory in posttraumatic stress disorder: A study of the Bosnian war veterans. *European Psychiatry, 21*, 167–173.

May, C. P., & Hasher, L. (1998). Synchrony effects in inhibitory control over thought and action. *Journal of Experimental Psychology, 24*(2), 363–379.

May, C. P., Hasher, L., Zacks, R. T., & Multhaup, K. S. (1999). Inhibition in the processing of Garden-Path Sentences. *Psychology and Aging, 14*(2), 304–323.

Nathaniel-James, D. A., Fletcher, P., & Firth, C. D. (1997). The functional anatomy of verbal initiation and suppression using the Hayling test. *Neuropsychologia, 35*(4), 559–566.

Noel, X., Bechara, A., Dan, B., Hanak, C., & Verbanck, P. (2007). Response inhibition deficit is involved in poor decision making under risk in nonamnesic individual with alcoholism. *Neuropsychology, 21*(6), 778–786.

Odhuba, R. A., van den Broek, M. D., & Johns, L. C. (2005). Ecological validity of measures of executive functioning. *British Journal of Clinical Psychology, 44*, 269–278.

Rotter, J. B. (1951). Word Association and sentence completion methods. In A. I. Ravin & M. R. Haworth (Eds.), *An introduction to projection techniques* (pp. 279–310). New York: Prentice Hall.

Rotter, J. B., Lah, M. I., & Rafferty, J. E. (1992). *The Rotter Incomplete Sentences Blank*, (2nd ed. manual). New York: Psychological Corporation.

Turnbull, O. H., Evans, C. E. Y., Kemish, K., Park, S., & Bowman, C. H. (2006). A novel set-shifting modification of the Iowa Gambling Task: Flexible emotion-based learning in schizophrenia. *Neuropsychology, 20*(3), 290–298.

Weiss, R., Toolis, E. E., & Cerankosky, B. C. (2008). Construct validity of the Rotter Incomplete Sentences Blank with clinic-referred and nonreferred adolescents. *Journal of Personality Assessment, 90*(6), 564–573.

Septum Pellucidum

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Definition

Multi-layered membrane extending downward from the corpus callosum, separating the two lateral ventricles. Its anterior extension terminates in the basomedial frontal cortex in the region of the septal nuclei. More posteriorly, its base follows the dorsal surface of the fornix. Occasionally a fluid-filled opening or slit is found within the anterior wall of this membrane known as a *cavum septum pellucidum*. Although this latter condition can be found in a normal population, it has been noted to occur with increased frequency in professional boxers (presumably as a result of repeated head trauma), schizophrenia, and other neurological disorders.

Sequential Processing

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Synonyms

Successive processing

Definition

Sequential processing refers to the mental process of integrating and understanding stimuli in a particular, serial order. Both the perception of stimuli in sequence and the

subsequent production of information in a specific arrangement fall under successive processing. The consecutive organization of stimuli is based upon the lack of relationship between separate stimuli, but the interdependence of each stimulus is unidirectional toward the preceding stimuli in the sequence, thus forming a chain-like progression. Thus, information can only be comprehended in a temporal, sequential manner, with each piece being dependent on the preceding element.

Current Knowledge

Successive processing is used to accomplish tasks where the focus is on the sequential or temporal order of information. Therefore, it is essential to early reading development, including phonological skills and decoding. Readers need to be able to identify and sound out letters in succession in order to determine the words in a text. In addition, the comprehension of syntax in a written language depends on the efficiency of sequential processing. These skills are fundamental to future success as a reader.

Sequential processing is often measured by recollection or completion of information in a temporal order to generate an accurate response. Instruments may include auditory, visual, or kinesthetic stimuli or even a combination of stimuli when assessing sequential processing. Examples include subtests that require the replication or repetition of numbers (Digit Span-Forward from the Wechsler Intelligence Scale for Children, Fourth Edition), words (Sentence Repetition from the Cognitive Assessment System), hand movements (Hand Movements from the Kaufman Assessment Battery for Children, Second Edition), or pictures (Symbolic Memory from the Universal Nonverbal Intelligence Test) in a predetermined sequence. It is also important to note that the vast majority of tools used to measure sequential processing also incorporate a strong memory component.

In Luria's theory, successive processing is one of two complementary information processing strategies comprising the second functional unit (occipital, parietal, and temporal lobes of the brain; Das, Naglieri, & Kirby, 1994). Sequential processing is predominantly accommodated by the left hemisphere, and specifically, in the frontotemporal region (Reynolds & French, 2005).

Cross References

► Cognitive Assessment System
► Simultaneous Processing

References and Readings

Horton, Jr., & A. M. Jr., & Wedding, D. (Eds.). (2006). *The neuropsychology handbook.* New York: Springer.

Reynolds, C. R., & French, C. L. (2005). The brain as a dynamic organ of information processing and learning. In R. C. D'Amato, E. Fletcher-Janzen, & C. R. Reynolds (Eds.), *Handbook of school neuropsychology* (pp. 86–119). Hoboken, NJ: Wiley.

Serial 3s

► Serial Subtractions

Serial 7s

► Serial Subtractions

Serial Digit Learning

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Synonyms

Digit supraspan

Description

Serial Digit Learning (Schinka, 1974) is a supraspan learning task that involves repeated presentation of a sequence of either eight or nine digits. There are three alternate forms for each length. As a general rule, the nine-digit sequences (SD9) are administered to persons under 65 years of age who have 12 or more years of education, whereas the eight-digit sequences (SD8) are administered to persons who are over 65 years of age or have fewer than 12 years of education. Examinees are given up to 12 trials to achieve the criterion of two consecutive correct repetitions. The sequences are presented verbally at the rate of one digit per second. The test can be administered in 5–10 min.

There is more than one scoring method for Serial Digit Learning. Originally, each trial was scored as correct or incorrect. The current method allows two points for correct response and one point for "near miss" responses (i.e., omission, addition, or substitution of one digit, or reversal of two adjacent digits). Raw scores can be converted to percentiles. Norms are available for ages 16 through 74. The stimuli, norms, and interpretive information are provided in Benton, Sivan, Hamsher, Varney, and Spreen (1994).

Current Knowledge

Performance on Serial Digit Learning is significantly affected by age and education but not gender or ethnic background (Hamsher, Benton, & Digre, 1980). Specifically, groups of persons between 44 and 64 years of age perform at a higher level than those between 65 and 74 years, and persons with 12 or more years of education consistently perform better than those with less than 12 years of education. Serial Digit Learning has consistently been found to be more sensitive than Digit Span for discriminating persons with brain injury or disease from health controls. The test tends to be more sensitive to left hemisphere than right hemisphere dysfunction. Factor analysis (Larrabee & Curtiss, 1995) found Serial Digit Learning to load primarily on a factor measuring attention/immediate memory and information processing and secondarily on a verbal learning factor.

References and Readings

Benton, A. L., Sivan, A. B., Hamsher, K., Varney, N. R., & Spreen, O. (1994). *Contributions to neuropsychological assessment* (2nd ed.). New York: Oxford University Press.

Hamsher, K., Benton, A. L., & Digre, K. (1980). Serial digit learning: Normative and clinical aspects. *Journal of Clinical Neuropsychology, 2*, 39–50.

Larrabee, G., & Curtiss, G. (1995). Construct validity of various verbal and visual memory tests. *Journal of Clinical and Experimental Neuropsychology, 17*, 536–547.

Schinka, J. A. (1974). *Performances of brain damaged patients on tests of short-term and long-term verbal memory.* Ph.D. dissertation, University of Iowa.

Serial Position Curve

► Serial Position Effect

Serial Position Effect

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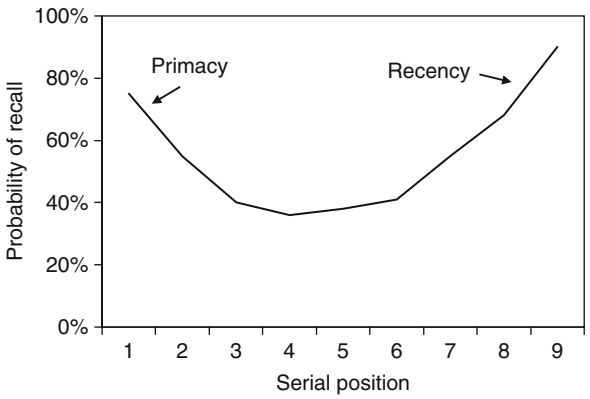
Synonyms

Serial position curve

Definition

The serial position effect refers to the finding that, on list-learning tasks, the probability of retrieving an item is dependent on the item's position in the study list. That is, items are more likely to be retrieved if they were initially presented at the beginning (i.e., the primacy effect) or the end of the list (i.e., the recency effect), relative to items presented in the middle. For example, immediately after presentation of a nine-item word list, individuals with normal memory ability might recall about 70% of the first three words, 60% of the last three words, but only 40% of the middle three words. This effect is illustrated by the serial position curve (Fig. 1). In addition to accuracy of retrieval, the serial position effect is also reflected in speed of retrieval: response times are faster when recognizing items presented at the beginning or the end of a list relative to items presented in the middle of the list.



Serial Position Effect. Figure 1 The serial position curve, showing increased probability of recall for items originally presented at the beginning and end of a list

Historical Background

Serial position effects were first reported in 1878 by physicist Francis Nipher. This initial work did not receive widespread attention, however, and it was several decades before similar effects were reported in the experimental psychology literature, by E. A. Kirkpatrick in 1894 and Hermann Ebbinghaus in 1902. Early research on the serial position effect was conducted using serial learning paradigms, in which participants were instructed to recall items in the same order of presentation. Later work showed similar findings using other paradigms such as free recall (regardless of order) and recognition.

Current Knowledge

Theoretical Interpretations

A number of theories have been offered to explain the serial position effect. One interpretation is that the primacy and recency portions of the effect are both due to the same underlying phenomenon. For example, initial and final list items are temporally and/or spatially distinctive, and this characteristic may render these items more memorable than the remaining items on the list.

There is evidence, however, that primacy and recency effects can be dissociated, both in patient groups and by experimental manipulations, and this suggests that distinct processes may be occurring. The dual-store interpretation of the serial position effect takes these findings into account. According to this view, memory for initial items reflects the operation of long-term or secondary memory, whereas memory for final items reflects the operation of short-term or primary memory. In other words, when immediate memory for a list is tested, items presented at the beginning of the list are no longer within one's short-term or working memory span, whereas items presented at the end of the list are. As such, the primacy and recency effects would be characterized by features of long-term and short-term memory, respectively.

The primacy effect, specifically, is thought to be related to the increased opportunity for rehearsal of initial list items, which enhances later retrieval of those items from long-term stores. Consistent with this, the primacy effect can be reduced or eliminated when rehearsal is prevented, for example, by presenting longer lists, decreasing the study time allowed for each item, or introducing a co-occurring secondary task. The recency effect, on the other hand, is thought to reflect a tendency to use a passive retrieval style of recalling more recently presented items first, and likely reflects the fact that retrieval of items from short-term stores tends to be easier than retrieval from long-term stores. The recency effect can be reduced or eliminated by introducing a distracter-filled delay between study and test, and the effect is less prominent at delayed recall than at immediate recall.

Relation to the Brain and Memory Disorders

Neuroimaging studies show that many of the same brain regions are involved during processing of items from the primacy and recency portions of the serial position curve. Brain areas showing increased activation include primary sensory, parietal, and frontal brain regions, and likely reflect processes such as subvocal rehearsal of items, increased attention, and executive control of memory processes that are similar for retrieval of both initial and final list items. In contrast, medial temporal brain regions are generally more active during retrieval of initial list items than final list items, likely reflecting the unique use of long-term memory processes during retrieval of items from the primacy portion of the serial position curve.

Consistent with neuroimaging findings, the primacy effect is generally reduced in memory disorders associated with medial temporal-lobe dysfunction, such as amnesia due to medial-temporal damage and early Alzheimer's disease. The primacy effect is also reduced in disorders with prominent frontal-subcortical involvement, such as schizophrenia and focal frontal-lobe lesions. The recency effect, on the other hand, is normal in most groups with focal or mild memory disorders, but is impaired in disorders with more generalized brain involvement, including moderate Alzheimer's disease, moderate vascular dementia, and traumatic brain injury. In general, the recency effect is more robust than the primacy effect, and is less likely to be disrupted by brain damage.

Cross References

▶ Primacy Effect
▶ Recency Effect

References and Readings

Gazzaniga, M. S., Ivry, R. B., & Mangun, G. R. (2002). *Cognitive neuroscience: The biology of the mind.* New York: Norton.

Tulving, E., & Craik, F. I. M. (Eds.). (2000). *The Oxford handbook of memory.* New York: Oxford University Press.

Serial Processing

► Serial/Sequential Processing

Serial Recall

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Synonyms

Digit span

Definition

A serial recall task requires participants to recall a list of items in a specific order, usually the order in which they were presented. Used in the digit span task, the serial recall test is probably the most widely-used short-term memory test in neuropsychology and psychology in general.

Current Knowledge

As participants must remember both the items and their order, accuracy is lower in serial recall than in free recall. Plotting the accuracy of items as a function of their position in the list yields the serial position curve. Typically, early list items are best recalled (primacy effect), followed by the most recent items (recency effect), with poor recall of center items. Theorists have advanced a variety of models of serial recall. According to chaining models, a person remembers a list in its correct order by forming links between adjacent items. Strength models assume that the strength of successive items decreases because of limits in attention or in rehearsal capacity, or because of interference from subsequent items. Distinctiveness models assume that early and late items are more distinctive, and thus more memorable, because they have fewer neighbors than items in the center of the list. Most models converge on the view that serial recall is not a pure storage task, but that cognitive control processes contribute to serial recall.

Accuracy in the serial recall task depends on a range of presentation and test parameters, as well as on population characteristics (Neath & Suprenant, 2003). List length is a key determinant of accuracy. People are usually able to recall lists of up to seven items after a single presentation. This limit is referred to as the memory span. Accuracy decreases for longer lists. Recall order also affects accuracy. Specifically, the requirement to recall the list in backward order lowers recall accuracy. Backward recall is thought to involve executive processes (Baddeley, 2001) to a greater extent than forward recall. The modality of stimuli influences accuracy, with better recall for auditory than visual stimuli. Any deviations from these typical parametric effects are used as a diagnostic for neuropsychological conditions (e.g., Lezak, 1995). Accuracy of serial recall depends on the age of the participants. Compared to younger participants, older participants typically score proportionally less well in serial recall than in free recall.

Based on neuropsychological deficits, neuroimaging, and ERP studies, several neural substrates of serial recall have been identified. For example, patients with damage to the inferior parietal cortex exhibit reduced accuracy in serial recall (Shallice, 1988). Studies using event-related neural imaging suggest that the left parietal cortex is implicated in the representation of order information. According to Jonides, Lacey, & Nee (2005), the brain structures that support storage and rehearsal in short-term memory are those that mediate perceptual processing. Thus, storage involves the posterior temporal cortex and the parietal cortex. Brain areas engaged in rehearsal are those that control attention to external stimuli. Depending on the modality of the stimuli, these areas include the posterior parietal and temporal cortices and the extrastriate cortex.

Cross References

► Free Recall
► Memory
► Memory Assessment Scales
► Memory Impairment
► Short-Term Memory
► Span Tests
► Verbal IQ
► Wechsler Adult Intelligence Scale (All Versions)
► Working Memory

References and Readings

Baddeley, A. (2001). Is working memory still working? *American Psychologist, 56*, 851–864.

Jonides, J., Lacey, S. C., & Nee, D. (2005). Processes of working memory in mind and brain. *Current Directions in Psychological Science, 14*, 2–5.
Lezak, M. (1995). *Neuropsychological assessment.* New York: Oxford University Press.
Neath, I., & Suprenant, A. (2003). *Human memory.* Belmont, CA: Thomson.
Shallice, T. (1988). *From neuropsychology to mental structure.* New York: Cambridge University Press.

Serial Recitation

► Serial Sevens

Serial Sevens

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Synonyms

Serial recitation

Description

Serial sevens is a measure of auditory attention/concentration, mental tracking, and computation that requires the examinee, starting at 100, to repeatedly subtract or add seven. The task originated with Kraepelin in 1899 (translated in 1990), who stated that either version could satisfactorily measure attentional disturbances, although he had the foresight to note that demographic factors (i.e., education, social class) should be considered during interpretation. Standardization of the task dates back to a 14-item version in 1942 (Hayman), which Luria (1966) interpreted as a measure of frontal lobe functioning. The subtraction version is the most commonly administered version, likely because of historical reasons (Kraeplein used subtraction in his example) and/or because it is perceived as more difficult than serial additions. Although the task is still used informally and qualitatively for rapid cognitive screening, it is best known to contemporary clinicians in a five-item subtraction form as part of the Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975). The task has also been included in the Montreal Test of Cognitive Assessment (Nasreddine et al., 2005) and the MMSE-2 (Folstein, Folstein, White, & Messer, 2010).

Current Knowledge

Women tend to make slightly more mistakes on serial subtracting sevens than men (Tombaugh & McIntyre, 1992), consistent with literature showing that men tend to outperform women on arithmetical tasks (Kaufman, McLean, & Reynolds, 1991). As with its MMSE counterpart (i.e., spelling "world" backwards), factor analytic studies determined that the task loads on a working memory and concentration factor (Banos & Franklin, 2002; Jones & Gallo, 2000). However, some authors have argued that serial sevens should not be interpreted as a pure attention/concentration task because of the relatively heavy influence of arithmetic skills (Karzmark, 2000; Manning, 1983).

Future Directions

More research is needed on the utility of the addition version of serial sevens, such as equivalency to the subtraction version, factor analytic characteristics, and the degree to which it is affected by demographic variables.

Cross References

► Mini-Mental State Exam
► Serial Subtractions

References and Readings

Banos, J. H., & Franklin, L. M. (2002). Factor structure of the mini-mental state examination in adult psychiatric inpatients. *Psychological Assessment, 14*, 397–400.

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*, 189–198.

Folstein, M. F., Folstein, S. E., White, T., & Messer, M. A. (2010). *Mini-mental state examination-2nd edition: User's manual.* Lutz, FL: Psychological Assessment Resources.

Hayman, M. (1942). Two minute clinical test for measurement of intellectual impairment in psychiatric disorders. *Archives of Neurology and Psychiatry, 47*, 454–464.

Jones, R. N., & Gallo, J. J. (2000). Dimensions of the mini-mental state examination among community dwelling older adults. *Psychological Medicine, 30*, 605–618.

Karzmark, P. (2000). Validity of the serial seven procedure. *International Journal of Geriatric Psychiatry, 15*, 677–679.

Kaufman, A. S., McLean, J. E., & Reynolds, C. R. (1991). Analysis of WAIS-R factor patterns by sex and race. *Journal of Clinical Psychology, 47*, 548–557.

Kraepelin, E. (1990). *Psychiatry: A textbook for students and physicians* (H. Metoui, Trans.). Canton, MA: Science History Publications.

Luria, A. R. (1966). *Higher cortical functions in man.* New York: Basic Books.

Manning, R. T. (1983). Serial sevens test. *Archives of Internal Medicine, 143*, 612.

Nasreddine, Z. S., Phillips, N. A., Bedirian, V., Charbonneau, S., Whitehead, V., Collin, I., et al. (2005). The Montreal Cognitive Assessment, MoCA: A brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society, 53*, 695–699.

Tombaugh, T. N., & McIntyre, N. J. (1992). The mini-mental state examination: A comprehensive review. *Journal of the American Geriatrics Society, 40*, 922–935.

Serial Subtractions

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Synonyms

Serial 3s; Serial 7s

Description

Attention and its many facets is a dimension of inquiry within any neuropsychological assessment, even when conducted in a relatively cursory fashion such as when a "bed-side" examination is performed. Unlike other neuropsychological dimensions, there is neither a single robust measure that can fully assess this dimension nor is there a unitary brain region that governs the complex network of skills, which fall under the rubric of attention. One facet of attention is referred to as mental tracking, or the ability to sustain focus while performing a cognitive operation over repeated trials. The Serial Subtractions task is one measure that assesses mental tracking capacity.

Current knowledge

The general strategy employed with serial subtractions involves requesting the examinee to "subtract 7 from 100" (Serial 7s) and, when they have completed this task, to "now subtract seven from ninety-three and to keep going until you can't go any further." The clinician records the examinee's responses, including errors, and should also make note of any other indications of difficulty, including confusion. A simpler version has also been used, which requires the examinee to subtract 3s from 50 (Serial 3s). Conversely, Serial 13s has also been described in the literature (Shum et al., 1990) and tends to be reserved for more intellectually capable examinees.

The Serial 7's task is a component of the Mini-Mental State Examination (MMSE). Only the first five responses are necessary, with "normal" performance requiring five serial subtractions. Spelling "WORLD" backward is alternatively used as a measure of concentration, but research suggests that Serial 7s and backwards spelling of "WORLD" are not commensurate tasks, with the latter consistently performed more successfully (Tombaugh & McIntyre, 1992). Serial subtraction tasks are also components of other neuropsychological measures, such as the Luria Nebraska Neuropsychological Battery.

Serial subtractions are not widely used by psychologists, which may be why validation research on these tasks has been limited, with a dearth of normative data available. Lezak, Howieson, and Loring (2004, p. 361) provided an interpretive table that refers to Sequential Operations Series, which includes normative data that can be useful for interpreting performance on the Serial 7 and Serial 3 tasks.

Difficulty on serial subtraction tasks may be related to a combination of weaknesses within skill areas, including sustained attention, task persistence, or mental calculations. Karzmark (2000) found that calculation skill was at least as important as concentration in predicting Serial Subtraction performance and suggested that the

method should be cautiously used as a measure of concentration.

Future Directions

Additional validation studies would be useful looking at the different aspects of task performance, including both error commission and speed of completion.

Cross References

► Serial Sevens

References and Readings

Karzmark, P. (2000). Validity of the serial seven procedure. *International Journal of Geriatric Psychiatry, 15*(8), 677–679.

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

Shum, D. H. K., McFarland, K. A., & Bain, J. D. (1990). Construct validity of eight tests of attention: Comparison of normal and closed head injured samples. *The Clinical Neuropsychologist, 4*, 151–162.

Smith, A. (1967). The serial sevens subtraction test. *Archives of Neurology, 17*, 18–80.

Tombaugh, T. N., & McIntyre, N. J. (1992). The mini-mental status examination: A comprehensive review. *Journal of American Geriatric Society, 40*, 922–935.

Serial/Sequential Processing

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Synonyms

Serial processing

Definition

A theoretical characterization of the processing stages for a given set of operations that is organized serially, in which one stage always precedes or follows another, from the inception to the completion of the task.

Historical Background

Early information processing models of the mind were viewed through the analogy of a computer. Computers were serial, and thus computational theories of the mind were overwhelmingly serial. Sternberg (1966) described the scanning of short-term memory as a serial process, such that the longer the set of items to be considered in a memory set the longer the reaction time. He observed that there was a linear increase in the reaction time as memory set increased, thus supporting the serial processing view that there is one comparison for each item in the set, the mean time for each comparison is constant, and so reaction time increases linearly with set size. Welford (1952) argued that there is a bottleneck in the processing system that makes it difficult, if not impossible (this is still open to debate) for two responses to two different stimuli to be made at the same time. In other words, there may be stages of processing in which processes can co-occur in parallel; however, the "bottleneck" stage is necessarily serial. Evidence of this comes from studies of the psychological refractory period. In these studies, there are two stimuli (e.g., two lights) and two responses (e.g., two buttons) and the participant must respond as quickly as possible to each. It has been found that when the two stimuli are presented closely together there is a slowing of response to the second stimulus.

Current Knowledge

Today, researchers have incorporated an understanding of the abundantly observed parallel systems of the brain into their theoretical models such that there are models that incorporate both parallel and serial behavioral processes. Distinctions between parallel and serial processes tend to fall along the divide of more or less cognitively demanding tasks, such that perceptually based, automatic processing is achieved through parallel processes and more complex, cognitively demanding tasks that involve controlled processes are serial (Shiffrin & Schnieder, 1977; Treisman & Gelade, 1980). To illustrate this distinction, feature integration theory of visual search proposes that perceptually based feature search is carried out through parallel processing whereas conjunction search is serial. In studies supporting this position, search reaction time for simple targets such as a green item among red items or an X among Os (feature search) was independent of the size of the set of items searched. It elicits a "pop-out" effect, such that it is easy to detect the target feature. In contrast, search reaction time for conjunctive targets such as a red X

among red Os and green Xs (conjunctive search) increases linearly with the set size. As described in the section above, a linear increase reaction time with increasing set size is considered evidence for serial processing.

Cross References

► Capacity Limitations
► Parallel Processing

References and Readings

Shiffrin, R. M., & Schnieder, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending, and a general theory. *Psychological Review, 84*, 127–190.

Sternberg, S. (1966). High speed scanning in human memory. *Science, 153*, 652–654.

Treisman, A., & Gormican, S. (1980). A feature integration theory of attention. *Cognitive Psychology, 12*, 97–136.

Welford, A. T. (1952). The "psychological refractory period" and the timing of high speed performance: A review and a theory. *British Journal of Psychology, 43*, 2–19.

Serotonergic Syndrome

► Serotonin Syndrome

Serotonin

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Definition

Serotonin is a neurotransmitter substance present in the central and peripheral nervous systems. It is widely distributed along the gastrointestinal tract, the brain, and spinal cord. In the brain, serotonergic cell bodies are located in the raphe nuclei and reticular regions of the brainstem. From these areas, cell bodies send projections to the spinal cord, cerebral cortex, basal ganglia,cerebellum, superior and inferior colliculi, the hypothalamus, and areas of the limbic system, including the hippocampus, amygdala, septum, and olfactory bulbs. Serotonergic neurons also send projections to the substantia nigra, a structure containing dopamine neurons (Feldman, Meyer, & Quenzer, 1997; Iversen, Iversen, Bloom, & Roth, 2009).

Serotonin is synthesized from the amino acid precursor, tryptophan, in a series of chemical reactions involving the catalytic enzymes, tryptophan hydroxylase, and aromatic L-amino acid decarboxylase. Inactivation of serotonin from the synaptic cleft is primarily achieved through a reuptake process via membrane transporter proteins. Degradation of serotonin is achieved through the activity of the enzyme monoamine oxidase A (MAO-A). This enzyme also metabolizes catecholamine neurotransmitters such as dopamine (Feldman et al., 1997). The serotonin metabolite, 5-hydroxyindoleacetic acid (5-HIAA; Feldman et al.) may be measured in the cerebral spinal fluid as an indication of central serotonergic activity.

Serotonin reportedly plays a role in a number of psychological and behavioral processes, including appetite, sleep (Feldman et al., 1997), and psychiatric illnesses such as depression, anxiety disorders, obsessive–compulsive disorder, schizophrenia, and aggression (Iversen et al., 2009). For example, serotonin agonists or enhancers have been developed as appetite suppressants and antidepressants (e.g., selective serotonin-reuptake inhibitors (SSRIs)). Several atypical antipsychotic medications also block a subtype of serotonin receptors (Iversen et al.). Low central serotonergic activity (measured by low 5-HIAA levels) has been associated with violent suicide attempts, and SSRIs have been found to reduce impulsive aggression (Siever, 2008). Hallucinogens such as lysergic acid diethylamide (LSD) and psilocybin also act on serotonin receptors (Iversen et al.).

Current Knowledge

Efficacy of Antidepressants

Recent studies have focused on factors that influence the effectiveness of SSRIs in alleviating symptoms of depression. Approximately 60–70% of patients reportedly show a positive treatment response to antidepressants (*c.f.* 30–50% respond positively to placebos; Iversen et al., 2009). Recent meta-analyses suggest that severity of depression may moderate response to treatment. Compared to placebo controls, antidepressants are effective among persons with more severe symptoms, but are much less

effective among those with mild symptoms (Fournier et al., 2010; Kirsch et al., 2008).

Studies have also examined the role of genetic polymorphisms in relation to efficacy of antidepressants, specifically variations in the serotonin transporter gene (often referred to as 5-HTTLPR). Compared to those with a long (L) allele of this gene, persons with a short allele exhibit poorer response to SSRI treatment. Furthermore, variations in 5-HTTLPR may also influence the experience of side effects (Horstmann & Binder, 2009). These and other studies are likely to help clarify the role of genetics in the effect of serotonin in psychiatric disorders and behavior.

References and Readings

Feldman, R. S., Meyer, J. S., & Quenzer, L. F. (1997). Acetylcholine. In *Principles of neuropsychopharmacology*. Sunderland, MA: Sinauer Associates.

Fournier, J. C., DeRubeis, R. J., Hollon, S. D., Dimidjian, S., Amsterdam, J. D., Shelton, R. C., et al. (2010). Antidepressant drug effects and depression severity: A patient-level meta-analysis. *JAMA, 303*, 47–53.

Horstmann, S., & Binder, E. B. (2009). Pharmacogenomics of antidepressant drugs. *Pharmacology and Therapeutics, 124*, 57–73.

Iversen, L. L., Iversen, S. D., Bloom, F. E., Roth, R. H. (2009). Acetylcholine. In *Introduction to neuropsychopharmacology* (pp. 214–237). New York: Oxford University Press.

Kirsch, I., Deacon, B. J., Huedo-Medina, T. B., Scoboria, A., Moore, T. J., Johnson, B. T. (2008). Initial severity and antidepressant benefits: A meta-analysis of data submitted to the food and drug administration. *PlOS Medicine, 5*, e45.

Serotonin Storm

► Serotonin Syndrome

Serotonin Syndrome

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Synonyms

Hyperserotonemia; Serotonergic syndrome; Serotonin storm; Serotonin toxicity; Serotonin toxidrome

Short Description or Definition

Serotonin Syndrome is a potentially life-threatening adverse drug reaction characterized by muscular overactivity, agitation and hyperthermia. Overstimulation of 5-HT_{1A} and possibly 5-HT_{2A} peripheral serotonin receptors in the central nervous system (CNS), more specifically, the central grey nuclei and medulla, are thought to be responsible for the characteristic symptoms and progression of the syndrome (Boyer & Shannon, 2005).

Interactions between certain serotonergic drugs in overdose or supra-therapeutic doses trigger a spectrum of clinical presentations ranging from minor to fatal. Combinations of serotonergic agents over stimulate postsynaptic serotonin receptors, synergistically increasing synaptic serotonin levels. Overdoses of selective serotonin reuptake inhibitors (SSRIs) alone have not been found to cause serotonin syndrome, but excessive amounts of the single serotonergic agent, irreversible MAOIs as well (Katzung, 2004; Whyte, 2004a, b). This suggests that different mechanisms may be responsible for elevating serotonin to the dangerous levels resulting in the symptoms of serotonin syndrome. No other diseases or conditions have been identified that would account for the symptoms seen in serotonin syndrome. Most cases resolve within a week. Mild to moderate cases usually resolve in 24–72 h. Acute cases require hospitalization and occasionally admission to the ICU with assisted ventilation. Mortality associated with this condition is approximately 11% (Nolan & Scoggin, 2002; Lheureux, Penaloza, DeCottenier, Ullmann, & Gris, 2002).

Categorization

Symptoms of serotonin syndrome can be categorized in a relative manner along a spectrum of intensity or severity of symptoms (Table 1). The spectrum, represented by progressive severity of clinical symptoms, signals increased risk of serotonin toxicity should escalating serotonin levels persist (Gillman, 2004).

Clinical findings across body systems (Table 2) can also be used to categorize symptoms of serotonin syndrome. This method differentiates three areas of overactivation, somatic function changes, excitation of the autonomic nervous system regulatory processes, and cognitive signs of disturbance.

Serotonin Syndrome. Table 1 Spectrum of Symptoms by Severity

Degree of severity	Symptoms
Mild	CNS overstimulation
	Irritability
	Twitching
	Sporadic tremor (myoclonus)
	Overly responsive reflexes (hyperreflexia)
	Shivering
	Tachycardia
	Sweating
	Dilated pupils (mydriasis)
Moderate	Increased myoclonus and hyperreflexia
	Hypertension
	Hyperthermia (up to 104°F)
	Increased bowel sounds
	Mental status changes
	Anxiety
	Agitation
	Hypervigiliance
	Confusion
Severe	Muscular rigidity
	Rhabdomyolysis
	Agitated delirium
	Dangerously elevated hyperthermia (104°–106°F)
	Seizure activity
	Intravascular coagulation
	Renal failure
	Pyramidal and truncal muscle rigidity
	Shock
	Coma
	Fatality

Serotonin Syndrome. Table 2 Spectrum of Symptoms by Body System

Body system	Symptoms
Somatic	Myoclonus
	Tremor
	Hyperreflexia
	Muscle rigidity
	Rhabdomyolysis
Autonomic	Shivering
	Hyperthermia
	Hyperhydrosis
	Hypertension
	Tachycardia
	Sweating
	Dilated pupils (mydriasis)
	Seizure activity
	Gastric dysregulation (increased bowel sounds)
	Intravascular coagulation
	Renal failure
	Impaired respiration
	Shock
	Fatality
Cognitive	Irritability
	Agitation
	Anxiety
	Confusion
	Hallucinations
	Agitated delirium
	Coma

Epidemiology

The neurotransmitter, serotonin (5-HT; 5-hydroxytryptamine), was isolated from intestinal mucosa in the 1800s. Not until the 1940s was serotonin recognized as having a role in the regulation of physiological and psychological states, including sympathetic nervous system outflow, pain perception, migraine, appetite, sleep, anxiety, mood, aggression, motor functions, smooth muscle contraction and blood vessels tone in the intestine (Martin, 1996; Myers, 2006).

Problems associated with excess serotonin were recognized in animals in the early 1950s. In 1955 the first human case was documented (Oates & Sjoerdsma, 1960). Early reports described what is now understood to be the result of 5-HTelevations following a dose-dependent relationship between the serotonin precursor tryptophan, irreversible A and B subtypes of MAOIs, and combined with potent SSRIs (Sternbach, 2003). From the 1960s as new classes of serotonergically active antidepressants and recreational drugs became available, the incidence of serotonin syndrome markedly increased. By

the mid-1980s, significant evidence of a "dose-effect" relationship was apparent.

Natural History, Prognostic Factors, Outcomes

Overstimulation of 5-HT_{1A} receptors appear to account for the majority of symptoms of serotonin syndrome. Medications that increase 5-HT_{1A} availability are more likely to lead to serotonin toxicity in overdose, while preliminary studies of 5-HT_{2A} post-synaptic receptor antagonists suggest reduced chances of toxicity and death. Animal studies have demonstrated decreased death rates from hyperpyrexia, but insufficient evaluation of these agents in humans has been done. At present, the role of the 5-HT_{2A} receptor contribution to the onset or remediation of serotonin syndrome is controversial (Isbister, Bowe, Dawson, & Whyte, 2004; Sun-Edelstein, 2008).

Following overdose of drug combinations that cause excessive serotonin concentrations, within minutes of the second serotonergic drug reaching effective blood levels, clinical signs of serotonin syndrome appear and progress rapidly. Initial symptoms usually involve neuromuscular tremor and excessive reflex responses. As toxic levels increase these neuromuscular manifestations generalize to other parts of the body.

Symptom Progression

Mild symptoms of CNS overstimulation and involuntary somatic activation often include muscle twitches and myoclonus. Hyperreflexia, tachycardia, hyperhydrosis, and mydriasis progress in intensity from the lower extremities upward as the syndrome worsens. More body system involvement and autonomic nervous system overactivation, including sympathetic and parasympathetic excitation suggest progression to moderate symptomology. Hypertension, hyperthermia, nausea, gastric spasticity, increased bowel sounds, diarrhea and respiratory distress exacerbated by decreased thoracic elasticity are exhibited as the syndrome progresses into the moderate to severe range. Changes in mental status escalate including increased anxiety and agitation. Symptoms are considered severe when they include muscular rigidity which can lead to rhabdomyolysis, agitated delirium, hyperthermia in the 104–106°F range, intravascular coagulation, renal failure, further escalation of tachycardia, and hypertension with the potential for shock. At severe levels cognitive changes escalate from headache to confusion. Extreme hyperthermic states can cause hallucinations, seizure, and coma. Pyramidal and truncal muscle rigidity can impair respiration and lead to death.

Risk Factors

Specific risk factors for the development of serotonin syndrome are not yet clear, but research supporting several possible biological vulnerabilities is mounting. In approximately 50% of individuals, excessive stimulation of the post-synaptic 5-hydroxytryptamine receptors, and certain combinations of drugs such as the irreversible MAOIs and SSRIs have posed serious, even life-threatening toxicity risk. Other biological risk factors include individuals who slowly metabolize SSRIs, those with compromised vascular systems or elevated c-reactive protein levels (Nolan and Scoggins, 2008; Gillman, 2005a).

Slow SSRI metabolizers comprise approximately 7% of the population. Risk of serotonin syndrome is increased because the blood levels of serotonin and its active metabolites escalate. While the relationship between the effects of antidepressants, elevated c-reactive protein levels, and cardiovascular or related diseases is unclear, the research suggest prudence because serotonin is a known vasoconstrictor and levels of c-reactive protein are higher in patients with vascular disease and depression. C-reactive protein stimulates platelet activity which induces serotonin release. It also inhibits the production of the enzyme, endothelial nitric oxide synthase which triggers the cessation of platelet-derived serotonin action (Le Doux, Braslow, & Brown, 2004; Saito et al., 2003).

Prognosis

The prognosis of serotonin syndrome is favorable provided implementation of appropriate medical management. Progression from mild to severe symptomology can be rapid, but treatment generally leads to resolution of symptoms within 24 h.

Outcome

Mild to moderate cases usually resolve in 24–72 h. Acute cases require hospitalization with occasional admission to the ICU with assisted ventilation. Mortality associated with this condition is as high as 11% (Nolan & Scoggin, 2002). In severe cases, delirium can persist for several days

(Gillman, 1998). Persistent complaints of symptoms consistent with antidepressant withdrawal or discontinuation syndrome have been reported, as have reports of ongoing muscle pain and weakness. Cases of serotonin syndrome improperly diagnosed or untreated for prolonged periods have resulted in kidney damage due to the breakdown of muscle following uncontrolled muscle spasms (University of Maryland website, 2006).

Neuropsychology and Psychology of Serotonin Syndrome

Serotonin is metabolized relatively rapidly, therefore, it is difficult to increase serotonin to toxic or fatal levels with only one type of serotonergic drug. In healthy adults, the risk of serotonin syndrome causing fatality by an SSRI-only overdose is rare. Levels of toxicity sufficient to cause death are most often the result of combinations of drugs that act on serotonin metabolism in different ways. As the doses of serotonergically active agents increase, a pattern emerges in which multiple body systems become over-stimulated increasing symptom susceptibility and severity. SSRIs combined with MAOIs, and MAOIs in conjunction with tricyclics, have been implicated most frequently in serious and fatal cases. Conversely, the action of irreversible MAOIs, their duration of effect, and the long half-life of SSRIs increase the potential for serotonergic properties to remain active for several weeks following discontinuation. Other dangerous combinations include MAOIs with compounds not typically considered actively serotonergic, such as opiate agonists like meperidine, over-the-counter agents in cold remedies and sleep aids such as dextromethorphan and tryptophan (Gillman, 2006b).

The different mechanisms of action and medication classes thought to trigger the onset of serotonin syndrome when taken in overdose or combined include the following:

1. SSRIs and SRIs (i.e., citalopram, fluoxetine, fluvoxamine, paroxetine, sertraline) that inhibit serotonin uptake.
2. MAOIs (i.e., iproniazid, isocarboxazid, linezolid, methylene blue, moclobemide, phenelzine, procarbazine, selegiline, tranylcypromine) that decrease serotonin metabolism.
3. SNRIs (i.e., venlafaxine, duloxetine and sibutramine) and TCAs (i.e., clomipramine and imipramine) that act as indirect serotonin agonists.
4. $5HT_1$ agonists (i.e., Triptan medication that treat migraine) that stimulate post-synaptic serotonin receptors.
5. CNS stimulants (i.e., phentermine, sibutramine, reserpine, methamphetamine and cocaine), Antihistamines (i.e., chlorpheniramine, brompheniramine), and opioids (i.e., meperidine, methadone, pentazocine, tramadol and fentanyl) that act as indirect agonists, nonspecifically increasing serotonin release.
6. $5HT_3$ antagonists (i.e., the anti-emetic ondansetron) that stimulate serotonin release from digestive tract.
7. The serotonin/dopamine antagonists (i.e., atypical antipsychotics such as olanzapine, risperidone, clozapine) that block $5HT_{2A}$ and $_{2C}$ receptors.

Non-pharmaceutical and herbal supplements (i.e., L-tryptophan, Boswellia, Ginseng, St. John's Wort, Yohimbe) that increase serotonin synthesis and possible serotonin reuptake inhibition (Cassins, Nicol, Quinntel, & Neumann, 2006; Munhoz, 2004; Parrot, 2002; Sun-Edelstein, Tepper, & Shapiro, 2008; Vuori et al., 2003; Whyte et al., 2003).

Evaluation

The spectrum of clinical findings resulting from excessive serotonin activity range from minimally perceptible to fatal. Symptoms are more likely to occur near the start of dosage increases, and when initial doses of the second serotonergic agent are ingested (University of Maryland website, 2006). Symptoms are highly individualized, requiring vigilance, careful review of the drug history, and solid working knowledge of the drugs with potent serotoninergic influence can assist evaluation and treatment of serotonin syndrome (Lheureux et al., 2002).

Differential Diagnosis

All possible causes must be ruled out prior to diagnosing serotonin syndrome, including anxiety or other psychiatric conditions, virus, infections, metabolic dysfunctions, drug overdose, withdrawal, or other neurological disorders. Neuroleptic malignant syndrome (NMS) is the condition most frequently confused with serotonin syndrome. Both share changes in mental status and hyper-arousal of the autonomic nervous system which can complicate diagnostic differentiation (Isbister, Dawson, & Whyte, 2001). Determining what medications were taken at the onset of symptoms, knowledge of medications that treat NMS but exacerbate serotonin syndrome (e.g., ▶ bromocriptine), and understanding subtle but important symptom differences is central to differentiating NMS from serotonin syndrome. Symptoms that differ between

NMS and serotonin syndrome include severe bradykinesia or extrapyramidal, lead-pipe rigidity with NMS versus hyperkinesias, sporadic rigidity and tremor or myoclonus with serotonin syndrome (Martin, 1996). Additionally, serotonin syndrome occurs rapidly following administration of excess serotonergic medications. NMS starts slowly after prolonged exposure to agents that block dopaminertic, but not serotonergic activity such as neuroleptics or withdrawal from dopamine agonists.

Diagnosis

There are no specific tests for serotonin syndrome. A thorough history of the onset and progression symptoms and medication is critical. Knowledge of prescribed, over-the-counter, and non-prescription compounds ingested at the time of and surrounding symptom onset must be gathered. A complete blood chemistry (CBC), thyroid function tests, and electrocardiogram and toxicology screens are tests generally administered to assist in the differential (Olson, 2004).

Several models have been proffered to diagnoses serotonin syndrome, all having in common the ingestion of serotonergic agents, muscular, autonomic, and/or cognitive abnormalities. Presence of a triad of excitatory characteristics including at least three of the following are generally considered sufficient for diagnosis: neuromuscular hyperactivity such as myoclonus, hyperreflexia or ataxia, autonomic hyperactivity such as diaphoresis, diarrhea, fever or shivering, and altered mental status such as anxiety or confusion.

The Hunter Serotonin Toxicity Criteria is another diagnostic system used to diagnose serotonin syndrome. Necessary and sufficient symptoms accordingly include spontaneous, inducible, or ocular clonus with agitation, diaphoresis, hyperreflexia, and hyperthermia with temperature above 100°F (Dunkley, Isbister, Sibbritt, Dawson, & Whyte, 2003).

Treatment

Treatment involves discontinuation of serotonergic medications and supportive care determined by symptoms and severity. Initial supportive measures include measures to treat hypertension, tachycardia, and decreasing hyperthermia. Observation and administration of benzodiazepines are common and generally lead to resolution within 24-h. $GABA_B$ receptor agonists lorazepam and diazepam effectively reduce agitation and myoclonus, but not anxiolytics with other than $GABA_B$ agonism action (Biji, 2004; Isbister & Buckley, 2005).

Treatment for moderate symptoms includes intravenous fluids, measures to reduce elevated temperature, and restoration of cardio-respiratory autonomic stability. Non-specific 5HT serotonin antagonists or partial antagonists such as cyproheptadine and methysergide with antihistaminergic properties, and $5\text{-}HT_{2A}$ antagonists such as propranolol have been used to expedite symptom improvement when vascular compromise has been suspected (Gillman, 1998; US Food and Drug Administration, 2006). Dantrolene, a calcium channel blocker and muscle relaxant used in cases of neuroleptic malignant syndrome to treat hyperpyrexia, has also been effective in reducing severe hyperthermia, spastic and rigid muscles when benzodiazepines were not. Life-threatening cases with neuromuscular paralysis have required intubation, sedation, and medically induced ventilation (Prator, 2006; Nelson et al., 2007).

Cross References

- ▶ Antidepressants
- ▶ Antipsychotics
- ▶ Anxiety
- ▶ Ataxia
- ▶ Delirium
- ▶ Disconnection Syndromes
- ▶ Myoclonus
- ▶ Neuroleptic Malignant Syndrome
- ▶ Neuroleptics
- ▶ Selective Serotonin Reuptake Inhibitors

References and Readings

Biji, D. (2004). The serotonin syndrome. *The Netherlands Journal of Medicine, 62*(9), 309–313.

Boyer, E. W., & Shannon, M. (2005). The serotonin syndrome. *New England Journal of Medicine, 352*(11), 1112–1120.

Brown, T., Skop, B., & Mareth, T. (1996). Pathophysiology and management of the serotonin syndrome. *Annals of Pharmacotherapy, 30*, 527–532.

Cassins, S., Nicol, E. A., Quinntel, & Neumann, P. (2006). The serotonin syndrome: Fatal course of intoxication with citalopram and moclobemide. *Anaesthesist, 55*(11), 1189–1196.

Dunkley, E. J., Isbister, G. K., Sibbritt, D., Dawson, A., & Whyte, I. M. (2003). The Hunter serotonin toxicity criteria: Simple and accurate diagnostic decision rules for serotonin toxicity. *Oxford Journal of Medicine, 96*(9), 635–642.

Gillman, P. K. (1998). Serotonin syndrome: History and risk. *Fundamental and Clinical Pharmacology, 12*(5), 482–491.

Gillman, P. K. (2004). The spectrum concept of serotonin toxicity. *Pain Medicine, 2*, 231–232.

Gillman, P. K. (2005a). Misleading cases. *Journal of the American Medical Directors Association, 6*, 422–423.
Gillman, P. K. (2005b). Monoamine oxidase inhibitors, opioid analgesics and serotonin toxicity. *British Journal of Anaesthesia, 95*, 434–441.
Gillman P. K. (2006a). Serotonin toxicity, serotonin syndrome: 2006 update, overview and analysis. www.psychotropical.com/Serotonin-Toxicity.doc, p. Epub 1–125.
Gillman, P. K. (2006b). A review of serotonin toxicity data: Implications for the mechanisms of antidepressant drug action. *Biological Psychiatry, 59*, 1046–1051.
Gillman, P. K. (2006c). A systematic review of the serotonergic effects of mirtazapine in humans: Implications for its dual action status. *Human Psychopharmacology: Clinical and Experimental, 21*(2), 117–125.
Isbister, G. K., & Buckley, N. A. (2005). The pathophysiology of serotonin toxicity in animals and humans: Implications for diagnosis and treatment. *Clinical Neuropharmacology, 28*(5), 205–214.
Isbister, G. K., Bowe, S., Dawson, A. H., & Whyte, I. M. (2004). Relative toxicity of selective serotonin reuptake inhibitors (SSRIs) in overdose. *Journal of Toxicology: Clinical Toxicology, 42*(3), 277–285.
Isbister, G. K., Dawson, A. H., & Whyte, I. M. (2001). Citalopram overdose, serotonin toxicity, or neuroleptic malignant syndrome? *Canadian Journal of Psychiatry, 46*(7), 657–659.
Katzung, B. G. (2004). Histamine, serotonin, and the ergot alkaloids. In B. Katzung (Ed.), *Basic and clinical pharmacology* (9th ed., pp. 259–280). New York: Lange Medical Books/McGraw-Hill.
Le Doux, M., Braslow, K., & Brown, T. (2004). C-reactive protein and serotonin syndrome. *American Journal of Psychiatry, 161*, 1499.
Lheureux, P., Penaloza, A., DeCottenier, V., Ullmann, U., & Gris, M. (2002). Serotonin syndrome. *Revue Medicale de Bruxelles, 23*(5), 422–428.
Martin, T. G. (1996). Serotonin Syndrome. *Annals of Emergency Medicine, 28*, 520–526.
Munhoz, R. P. (2004). Serotonin syndrome induced by a combination of bupropion and SSRIs. *Clinical Neuropharmacology, 27*(5), 219–222.
Myers, T. (2006). Serotonin (definition). In T. Myers (Ed.), *Mosby's dictionary of medicine, nursing & health professions* (7th ed., pp. 1701). Missouri: Mosby Elsevier.
Nelson, L. S., Erdman, A. R., Booze, L. L., Cobaugh, D. J., Chyka, P. A., Woolf, A. D., et al. (2007). Selective serotonin reuptake inhibitor poisoning: An evidence-based consensus guideline for out-of-hospital management. *Clinical Toxicology, 45*(4), 315–322.
Nolan, S., & Scoggin, J. A. (2008). Serotonin syndrome: Recognition and management. On website http://www.USPharmacist.com *23*, 2.
Nolan, S., & Scoggin, J. S. (2002). Serotonin syndrome: Recognition and management. *U.S. Pharmacist, 23*(2).
Oates, J. A., & Sjoerdsma, A. (1960). Neurologic effects of tryptophan in patients receiving a monoamine oxidase inhibitor. *Neurology, 10*, 1076–1078.
Olson, K. R. (2004). Management of the poisoned patient. In B. Katzung (Ed.), *Basic and clinical pharmacology* (9th ed., pp. 982–994). New York: Lange Medical Books/McGraw-Hill.
Parrot, A. C. (2002). Recreational ecstasy/MDMA, the serotonin syndrome, and serotonergic neurotoxicity. *Pharmacology and the Biochemistry of Behavior, 71*(4), 837–844.
Prator, B. (2006). Serotonin syndrome. *Journal of Neuroscience Nursing, 38*(2), 102–105.
Saito, M., Ishimitsu, T., Minami, J., Ono, H., Ohrui, M., & Matsuoka, H. (2003). Relations of plasma high-sensitivity c-reactive protein to traditional cardiovascular risk factors. *Atherosclerosis, 167*, 73–79.
Sporer, K. A. (1995). The serotonin syndrome: Implicated drugs, pathophysiology and management. *Drug Safety, 13*(2), 94–104.
Sternbach, H. (2003). Serotonin syndrome: How to avoid, identify, and treat dangerous drug interactions. *Current Psychiatry, 2*, 12–24.
Sun-Edelstein, C., Tepper, S. J., & Shapiro, R. E. (2008). Drug-induced serotonin syndrome: A review. *Expert Opinion on Drug Safety, 7*(5), 587–596.
University of Maryland Medical Center website. (2006). http://www.umm.edu/ency/article/007272.htm
US Food and Drug Administration. (2006). *FDA public health advisory: Combined use of 5-hydroxytryptamine receptor agonists (triptans), selective serotonin reuptake inhibitors (SSRIs) or selective serotonin/norepinephrine reuptake inhibitors (SNRIs) may result in life-threatening serotonin syndrome.* Rockville, MD: Center for Drug Evaluation and Research. July 19, 2006.
Vuori, E., Henry, J., Ojanperä, I., Nieminen, R., Savolainen, T., Wahlsten, P., & Jäntti, M. (2003). Death following ingestion of MDMA (ecstasy) and moclobemide. *Addiction, 98*(3), 365–368.
Whyte, I., Dawson, A., & Buckley, N. (2003). Relative toxicity of venlafaxine and selective serotonin reuptake inhibitors in overdose compared to tricyclic antidepressants. *Quarterly Journal of Medicine, 96*(5), 369–374.
Whyte, I. M. (2004a). Neuroleptic malignant syndrome and serotonin toxicity/syndrome. In R. C. Dart (Ed.), *Medical toxicology* (pp. 101–106). Baltimore: Lippincott Williams & Wilkins.
Whyte, I. M. (2004b). Serotonin uptake inhibitors. In R. C. Dart (Ed.), *Medical toxicology* (pp. 843–851). Baltimore: Lippincott Williams & Wilkins.

Serotonin Toxicity

▶ Serotonin Syndrome

Serotonin Toxidrome

▶ Serotonin Syndrome

Sertraline

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Generic Name

Sertraline

Brand Name

Zoloft

Class

Selective serotonin reuptake inhibitor

Proposed Mechanism(s) of Action

Inhibits serotonin reuptake pump. Also known to block dopamine transport/reuptake and sigma. Only SSRI known to protect against hyperprolactinemia.

Indication

Major depressive disorder, panic disorder, social anxiety disorder, obsessive– compulsive disorder, posttraumatic stress disorder, premenstrual dysphoric disorder.

Off Label Use

Generalized anxiety disorder.

Side Effects

Serious

Seizures, mania, suicidal ideation.

Common

Sexual dysfunction, appetite disturbance, dry mouth, diarrhea, constipation, sedation, agitation, tremors, dizziness, sweating, bruising, hyponatremia, hypotension.

References and Readings

Physicians' Desk Reference (62nd ed.). (2007). Montvale, NJ: Thomson PDR.

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

Additional Information

Drug interaction effects: http://www.drugs.com/drug_interactions.html
Drug molecule images: http://www.worldofmolecules.com/drugs/
Free drug online and PDA software: www.epocrates.com
Gene-based estimate of drug interactions: http://mhc.daytondcs.com:8080/cgi bin/ddiD4?ver=4&task=getDrugList
Pill identification: http://www.drugs.com/pill_identification.html

Set Shifting

► Mental Flexibility

Severe Brain Injury

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Synonyms

Severe traumatic brain injury

Definition

A severe brain injury is typically characterized by GCS scores ranging from 3 to 8 (typically in a coma), loss of consciousness or length of post-traumatic amnesia lasting greater than 24 h, and positive neurologic history (e.g., skull fractures or intracranial hemorrhages). Specifically, post-traumatic amnesia lasting 1 to 7 days is classified as being a severe concussion, while post-traumatic amnesia over 7 days is classified as being very severe (Larrabee, 2005). Nearly 5–25% of brain injuries are considered to be severe (Kraus & McArthur, 1996) and of the 50,000 to 75,000 individuals that sustain a severe traumatic brain injury each year, between one-third and one-half die (Whyte, Hart, & Laborde, 1998). Severe brain injuries typically result in a number of cognitive, physical, and emotional/neurobehavioral challenges, many of which may be permanent and can vary over the course of several years. Neuropsychological assessment of a wide range of cognitive functioning can examine depth and breadth of severity and help make predictions related to functioning and return to activities. Typically, severe brain injury is related to poor prognosis with regard to return to work (70% unemployment rates). Primary causes of severe brain injury typically include significant neurotrauma causing internal bleeding or severe diffuse axonal injuries, as well as secondary complications including

DAI, hematomas, intracranial hemorrhages, and hypoxias.

Cross References

- ▶ Coma
- ▶ Epidemiology
- ▶ Head Injury
- ▶ Loss of Consciousness
- ▶ Traumatic Brain Injury

References and Readings

Centers for Disease Control and Prevention. (2007). What is traumatic brain injury? Retrieved December 26, 2007, from http://www.cdc.gov/ncipc/tbi/TBI.htm

Ficker-Terrill, C., Flippo, K., Antoinette, T., & McMorrow, D. B. (2007). Overview of brain injury. In *The essential brain injury guide* (4th ed., pp. 1–24). McLean, VA: Brain Injury Association of America.

Gennarelli, T. A., & Graham, D. I. (2005). Neuropathology. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Textbook of traumatic brain injury* (pp. 27–50). Arlington, VA: American Psychiatric Publishing.

Kraus, J. F., & McArthur, D. L. (1996). Epidemiologic aspects of brain injury. *Neurological Clinics, 14*, 435–448.

Sherer, M., & Madison, C. F. (2005). Moderate and severe traumatic brain injury. In G. J. (Ed.), Larrabee, *Forensic neuropsychology: A scientific approach* (pp. 237–270). New York: Oxford University Press.

Whyte, J., Hart, T., & Laborde, A. (1998). Rehabilitation of the patient with traumatic brain injury. In J. Delisa et al. (Eds.), *Rehabilitation medicine: Principles and practice*. (3rd ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Severe Hypoxia

- ▶ Anoxia

Severe Impairment Battery

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Synonyms

Severe impairment battery-short form; SIB; SIB-S

Description

The severe impairment battery (SIB; Saxton, McGonigle, Swihart, & Boller, 1993) was developed to assess the skills of people with severe dementia (suitable for people aged 51–91). The commercial version consists of 40 simple one-step commands and gestural cues, presented in a relatively naturalistic, conversational style. The SIB requires simple responses from the examinee, with points being awarded for approximations and partially correct responses as well as for fully correct ones, and prompts being included in the event of "don't know" or incorrect responses. The maximum score is 100. An example task is the examiner showing the examinee a picture of a cup and asking "what's this?" then later "show me how you would use it." There are six subscales: attention, orientation, language, memory, visuospatial ability, and construction. Assessment of praxis, social interaction and orientation to name is also included. The battery takes 20–30 min to administer. A shortened version has recently been made available (Saxton, McGonigle, Swihart, & Buller, 2008), which includes 26 of the original SIB items, with a maximum achievable score of 50 points. It takes between 10 and 15 min to administer.

Historical Background

The SIB was developed to assess the cognitive skills of people with severe dementia for whom conventional neuropsychological tests may not be suitable. Experimental versions of the battery have included 50 tasks or more, but the commercial version has 40. The official short form (SIB-S; Saxton et al., 2008) was developed in response to the constraints of testing people with very severe dementia.

Psychometric Data

Saxton, McGonigle-Gibson, Swihart, Miller, & Boller (1990) reported that the original 50-question SIB had very high inter-rater reliability, with the lowest observed correlation of 0.87 for the praxis scale. Correlations for all other scales were between .97 and 1.0. Test-retest reliability was also high (total score $r = .85$), though subscale correlations ranged from .22 (construction) to .86 (praxis). The correlation between SIB and MMSE total scores was .74, an indication of construct validity. Suh & Kang (2005) examined the psychometric properties of their Korean

version of the 40-item SIB, and reported good inter-rater reliability (r = .99), and one-week test-retest reliability (r = 0.97), and found strong correlations with existing measures of cognitive performance (SIB/MMSE=0.87, SIB/cognitive scale of the Alzheimer's Disease Assessment Scale = 0.76), whilst avoiding the floor effects present in these other measures with severely demented patients. Comparable results were found for the French version of the SIB (Panisset, Roudier, Saxton, & Boller, 1994), and Barbarotto, Cerri, Acerbi, Molinari, & Capitani (2000) found evidence of high test-retest reliability for the Italian translation (r = 0.95 at a two-week interval). Wild & Kaye (1998) found the SIB was more useful than the MMSE in tracking the rate of decline of people with Alzheimer's disease over a period of 2 years, the former providing a range of scores whilst many patients were at floor on the MMSE.

Saxton et al. (2005) conducted factor analyses on the standard SIB, in order to identify items appropriate for deletion to form the SIB-S. Separate examinations were undertaken from two groups of dementia patients, one from the USA, and one from France, all with MMSE scores below 10 (and thus categorized as severely impaired). Twenty five items were eventually selected for deletion, largely those identified by the factor analyses, with exceptions where deletion would interfere with test administration. The scores on SIB and SIB-S correlated at r = .99, suggesting that the test's sensitivity was maintained in its shortened form, though the correlation between SIB-S and MMSE scores of 0.68 is somewhat lower than previously reported rates for the long form. There was no evidence of ceiling or floor effects in this patient group (in whom MMSE scores ranged from 0–10).

Clinical Uses

The test can be used for performance-based assessment of low-level skills in patients with advanced dementia and other related populations without resorting to rating-scale measures. Several studies have found that patients scoring in the lowest range of the MMSE (e.g., <6) show a much wider range of scores on the SIB, attesting to its utility in assessing cognitive function and measuring change over time in more impaired patient groups. Saxton et al. (2008) suggest that the SIB-S is more suitable than the SIB for assessing agitated patients or those with MMSE scores below 5 who can find even the standard SIB too taxing. Many clinical trials including patients with more-than-moderate Alzheimer's disease include the SIB as an outcome measure, and German, Italian, Japanese, Korean, Norwegian, and Spanish translations are available. Dalla Barba et al. (2008) have commented that disadvantages of using the SIB include the length of time required for scoring and that patients with milder Alzheimer's disease generally perform at ceiling. Clearly, the latter disadvantage reflects the test's principal aim of assessing people with severe impairments, both in terms of the difficulty level of the items included, and the length of time the test takes to administer.

Cross References

- ▶ Clinical Dementia Rating Scale
- ▶ Mini-Mental State Examination

References and Readings

Barbarotto, R., Cerri, M., Acerbi, C., Molinari, S., & Capitani, E. (2000). Is SIB or BNP better than MMSE in discriminating the cognitive performance of severely impaired elderly patients? *Archives of Clinical Neuropsychology, 15*, 21–29.

Dalla Barba, G., Traykov, L., & Baudic, S. (2008). Neuropsychological examination in dementia. In C. Duyckaerts & I. Litvan (Eds.), *Dementias: Handbook of clinical neurology* (Vol. 89). Amsterdam, The Netherlands: Elsevier.

Panisset, M., Roudier, M., Saxton, J., & Boller, F. (1994). Severe impairment battery: A neuropsychological test for severely demented patients. *Archives of Neurology, 51*, 41–45.

Pelisser, C., Roudier, M., & Boller, F. (2002). Factorial validation of the severe impairment battery for patients with alzheimer's disease: *A pilot study. Dementia and Geriatric Cognitive Disorders, 13*, 95–100.

Saxton, J., Kastango, K. B., Hugonot-Diener, L., Boller, F., Verny, M., Sarles, C. E., Girgis, R. R., Devouche, E., Mecocci, P., Pollock, B. G., & DeKosky, S. T. (2005). Development of a short form of the severe impairment battery. *American Journal of Geriatric Psychiatry, 13*(11), 1–7.

Saxton, J., McGonigle, K. L., Swihart, A. A., & Boller, F. (1993). *The severe impairment battery.* San Antonio, TX: Pearson.

Saxton, J., McGonigle, K. L., Swihart, A. A., & Boller, F. (2008). *The severe impairment battery-short form.* San Antonio, TX: Pearson.

Saxton, J., McGonigle-Gibson, K. L., Swihart, A. A., Miller, V. J., & Boller, F. (1990). Assessment of the severely impaired patient: Description and validation of a new neuropsychological test battery. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 2*, 298–303.

Suh, G. H., & Kang, C. J. (2005). Validation of the severe impairment battery for patients with Alzheimer's disease in Korea. *International Journal of Geriatric Psychiatry, 21*, 626–632.

Wild, K. V., & Kaye, J. A. (1998). The rate of progression of Alzheimer's disease in the later stages: Evidence from the severe impairment battery. *Journal of the International Neuropsychological Society, 4*, 512–516.

Severe Impairment Battery-Short Form

► Severe Impairment Battery

Severe Traumatic Brain Injury

► Severe Brain Injury

Sex Hormone

► Steroids

Sex Therapy

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Definition

Sex therapy is generally seen as a subtype of psychotherapy geared toward improving sexual dysfunction.

Historical Background

Prior to the seminal publication of Masters & Johnson's *Human Sexual Inadequacy* (1970), treating sexual dysfunction often involved long-term psychodynamically oriented therapy.

Rationale or Underlying Theory

Largely as a result of Masters & Johnson's work, sex therapy is predicated on the assumption that sexual dysfunction involves medical, physiological, cognitive, affective, and behavioral factors. Now, treatment often incorporates psychotherapy, medical management, and some form of sexual retraining (see Treatment Procedures for examples).

Goals and Objectives

Sex therapy targets sexual dysfunctions such as premature ejaculation, erectile dysfunction, female sexual arousal disorder, male and female orgasmic disorders, vaginismus and disorders of desire (i.e., hypoactive sexual desire disorder). It is important to note that some patient populations are at greater risk for sexual dysfunction and may thus benefit from being screened for sexual dysfunction and considered for sex therapy. These include those with depression, coronary diseases, renal failure, diabetes, hyperprolactinemia, hypogonadism (in men), bilateral oophorectomy (in women), adrenal disease, neurological and cerebrovascular disease, Parkinson's disease, multiple sclerosis, and head injuries (for a review of sexual dysfunction in neurological patients, see Basson & Schultz, 2007; Rees, Fowler, & Maas, 2007; for information on sexual rehabilitation in the context of disability, see Stiens, Westheimer, & Young, 2002). Medications that commonly result in sexual dysfunction are antipsychotics, antihypertensives, antidepressants, anti-androgens, narcotics, and antiepileptic drugs (Basson & Schultz, 2007).

Treatment Participants

Couples showing the most improvement in sex therapy have good general and sexual relationships and good motivation of the male partner before beginning therapy (Hawton & Catalan, 1986). Failure to complete sex therapy may be associated with lesser education, history of psychiatric treatment in the female partner, poor motivation in the male partner, poor couple communication, and lower levels of self-reported sexual pleasure experience by the female partner (Hawton & Catalan, 1986).

Treatment Procedures

Masters & Johnson (1970) emphasized brief, directive, symptom-oriented therapy involving the couple. Modern sex therapy incorporates several unique components: (1) sexual dysfunctions are shared disorders between couples, (2) psychosexual education is key, (3) identifying and altering negative beliefs about sexuality, (4) eliminating performance anxiety, (5) increasing communication and effectiveness of sexual technique, (6), changing destructive lifestyles and sex roles, and (7) prescribing changes in sexual behavior (LoPiccolo & LoPiccolo, 1978). In regard to the latter point, sex therapy is unique

because of its combination of "systematic structured sexual experiences" with therapy session (LoPiccolo & LoPiccolo, 1978).

A variety of types of sex therapy exist including: (1) structured sexual experiences (e.g., graduated and nongraduated approaches, masturbation therapy), (2) cognitive/attentional techniques (e.g., directing attention to arousal processes rather than anxiety), (3) pause and squeeze techniques (i.e., for premature ejaculation, the inhibition of orgasm at point of maximum stimulation by pausing and/or squeezing the frenulum), (4) group therapy, (5) communication training and marital therapy, (6) psychosexual education and technique training, (7) assertiveness training (i.e., social skills training) and (8) conjoint therapy (i.e., emphasizing the shared aspect of sexual dysfunction; LoPiccolo & LoPiccolo, 1978).

Efficacy Information

There is not a large body of evidence for the effectiveness of sex therapy for treating symptoms of sexual dysfunctions (LoPiccolo & LoPiccolo, 1978). This may be partially due to the variety in types of sex therapy. Also, historically much of the evidence for successful sex therapy is based on nonrandomized groups and case studies that often report therapeutic benefit without symptom reduction. For example, systematic desensitization is a technique that appears to reduce sex-related anxiety, but not the symptoms of sexual dysfunction in women (LoPiccolo & LoPiccolo, 1978). Recent reviews of sexual disorders such as vaginismus indicate a lack of well-controlled studies and little therapeutic benefit associated with treatments such as systematic desensitization (McGuire & Hawton, 2001). Another systematic review of psychosocial interventions for erectile dysfunction concluded that sex-group therapy improved erectile function (Melnik, Soares, & Nasello, 2007). Although there does not appear to be a strong body of evidence suggesting the efficacy of sex therapy overall, this may reflect methodological limitations in extant sex therapy research, given the plethora of successful noncontrolled group and case studies (see Masters & Johnson, 1970).

Outcome Measurement

Although there are objective measures of arousal (vaginal plethysmograph for women, Barlow gauge for men), outcomes in sex therapy are predominantly self-reported. For example, self-reported specific symptom reduction of the patient would be a treatment outcome in individual therapy. Examples might be the maintenance of an erection satisfactory to both partners, ability to experience orgasm or lengthening the intra-ejaculatory latency period (a measure of ejaculation time for premature ejaculation). Given that a diagnosis of sexual disorder by the DSM-IV requires impairment and/or distress, self-report measures are likely appropriate therapeutic outcomes. In addition to symptom reduction, any sex-related anxiety may be another target of individual treatment.

Qualifications of Treatment Providers

Sex therapy is an unregulated practice and does not require licensure to practice. However, groups such as the American Association of Sexuality Educators, Counselors and Therapists (AASECT) provide guidelines and certification for sex therapists. To be certified by the AASECT as a sex therapist, one must be a licensed mental health professional able to provide psychotherapy with specialization in patients with sexual dysfunction.

Cross References

- ▶ Hypersexuality/Hyposexuality
- ▶ Sexual Disinhibition
- ▶ Sexual Surrogate

References and Readings

Basson, R., & Weijmar Schultz, W. (2007). Sexual sequelae of general medical disorders. *Lancet, 369*, 409–424.

Basson, R., Rees, P., Wang, R., Montejo, A., & Incrocci, L. (2010). Sexual function in chronic illness. *Journal of Sexual Medicine, 7*, 374–388.

Hawton, K., & Catalan, J. (1986). Prognostic factors in sex therapy. *Behaviour Research and Therapy, 24*, 377–385.

LoPiccolo, J., & LoPiccolo, L. (Eds.). (1978). *Handbook of sex therapy.* New York: Plenum.

Masters, W. H., & Johnson, V. E. (1970). *Human Sexual Inadequacy.* Toronto, NY: Bantam Books. ISBN 0-553-20699-0

McGuire, H., & Hawton, K. K. E. (2001). Interventions for vaginismus. *Cochrane Database of Systematic Reviews*, Issue 2, Art. No.: CD001760, doi: 10.1002/14651858.CD001760.

Melnik, T., Soares, B., & Nasello, A. G. (2007). Psychosocial interventions for erectile dysfunction. *Cochrane Database of Systematic Reviews*, Issue 3, Art. No.: CD004825, doi: 10.1002/14651858.CD004825.pub2.

Rees, P. M., Fowler, C. J., & Maas, C. P. (2007). Sexual function in men and women with neurological disorders. *Lancet, 369*, 512–525.

Stiens, S. A., Westheimer, R. K., & Young, M. A. (2002). Sexual satisfaction despite disability. In *Physical medicine & rehabilitation secrets*, (2nd ed.). Philadelphia, PA: Hanley & Belfus.

Sexual Disinhibition

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Definition

Sexual disinhibition refers to socially or contextually inappropriate sexual behavior and is usually associated with frontal and temporal lobe pathology. There is some evidence that sexual disinhibition occurs due to loss of cerebro-cortical inhibitor mechanisms, which results in the following behaviors: increased sexual interest, inappropriate cuddling, touching of the genitals, sexual propositions, grabbing and groping, use of obscene language, and masturbating in inappropriate settings without shame. Similar symptoms are noted in fronto-temporal dementia, focal fronto-temporal lesions, mania, and following a seizure or treatment of Parkinson's disease. Several medications, including antidepressants, anxiolytics, hormones, and lithium, have been used to treat sexual disinhibition.

Cross References

- ► Kluver-Bucy Syndrome
- ► Parkinson's Disease
- ► Pick's Disease

References and Readings

Hashmi, F. H., Krady, A. I., Qayum, F., & Grossberg, G. T. (2000). Sexually disinhibited behaviour in the cognitively impaired elderly. *Clinical Geriatrics, 8*(11), 61–68.

Nagaratnam, N., & Gayagay, G. (2002). Hypersexuality in nursing care facilities: A descriptive study. *Archives of Gerontology and Geriatrics, 35*, 195–203.

Pilleri, G. (1966). The Kluver-Bucy syndrome in man: A clinicoanatomical contribution to the function of the medial temporal lobe structures. *Psychiatria et Neurologia, 152*, 65–103.

Sexual Surrogate Therapy

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Synonyms

Surrogate partner therapy

Definition

A method or treatment, seldom employed in the United States, which is sometimes recommended for individuals with traumatic brain injury and other neurologic disorders that significantly alter or compromise sexual functioning. Advocates view the use of surrogate therapy as an important aspect of a holistic approach to rehabilitation aimed at improving quality of life and fulfilling basic human intimacy needs. When this therapy is practiced in a professional manner a client, a trained sexual therapist and a trained surrogate partner will form a three-person therapeutic team with the individual undergoing therapy. The surrogate participates with the client in structured and unstructured experiences that are designed to build client self-awareness and skills in the areas of physical and emotional intimacy. These therapeutic experiences include relaxation techniques in intimate situations, effective communication, sensual and sexual touching, and social skills training. The therapist is crucial to the process in terms of helping the client learn from the experiences and generally supervising the process. When the client is higher functioning, the goal is to transition a mutually initiated, non-surrogate relationship. While there are several case reports in the scientific literature, there are no treatment studies addressing the efficacy of surrogacy treatment to date.

References and Readings

Aloni, R., & Katz, S. (2003). *Sexual difficulties after traumatic brain injury and ways to deal with it*. Springfield, IL: Charles C. Thomas.

SF-36/SF-12

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Synonyms

Medical Outcomes Study Short-Form Health Survey 12; Medical Outcomes Study Short-Form Health Survey 36; Short-Form 12; Short-Form 36

Description

One of the most frequently used health-related quality of life measures, the SF-36, contains 35 questions, which load onto 8 scales: physical function (10 items), role limitations due to physical health problems (4 items), body pain (2 items), general health (5 items), vitality (4 items), social functioning (2 items), role limitations due to emotional problems (3 items), and emotional well-being (5 items). Some items use a 3-point rating scale, while others use a 5-point scale; some items call for frequency ratings, while others require judgments of quality of performance. The scales can be combined into two summary measures: physical component summary (PCS) and mental component summary (MCS) scores. The 36th question asks about health change and does not contribute to the scales or summary measures. The SF-12 contains 12 questions from the SF-36, which can also yield PCS and MCS scores.

The SF-36/SF-12 was designed for use in individuals of 14 years or older. It can be self-administered or given by a trained interviewer in person or by telephone. For each scale and the PCS and MCS, responses are added and the total is converted to a scale ranging from 0 to 100 with higher scores indicating better functioning.

The SF-36/SF-12 has been revised; currently version 2 is available (www.sf-36.org). In addition, an eight-item version is available (SF-8).

Historical Background

The SF-36 was developed as a part of the Medical Outcomes Study at the New England Medical Center in Boston, Massachusetts and the Rand Corporation in Santa Monica, California by John Ware and his colleagues (Ware, 1993).

Psychometric Data

Reliability: The reliability of the eight scales and two summary measures has been estimated using both internal consistency and test–retest methods. With rare exceptions, published reliability statistics have exceeded the minimum standard of 0.70 recommended for measures used in group comparisons in more than 25 studies (Tsai, Bayliss, & Ware, 1997); most have exceeded 0.80 (McHorney, Ware, Lu, & Sherbourne, 1994; Ware, 1993).

The trends in reliability coefficients for the SF-36 scales and summary measures have also been replicated across 24 patient groups differing in socio-demographic characteristics and diagnoses (McHorney et al., 1994; Ware, 1993; Ware, Gandek, & the IQOLA Project Group, 1994). Reliability estimates consistent with these trends have been published in more than 200 studies; results from more than 30 test–retest studies have also been summarized (Turner-Bowker, Bartley, & Ware, 2002).

Validity: Studies of validity generally support the intended meaning of high and low SF-36 scores as documented in the original user's manuals (Ware, 1993, 1994). Because of the widespread use of the SF-36 across a variety of applications, evidence from many types of validity research is relevant to these interpretations. Studies to date have yielded content, concurrent, criterion, construct, and predictive evidence of validity. The content validity of the SF-36 has been compared to that of other widely used generic health surveys and suggests that the SF-36 contains eight frequently measured health concepts (Ware, 1995; Ware, 1993). Predictive studies of validity have linked the SF-36 scales and summary measures to utilization of health care services (Ware et al., 1994), the clinical course of depression (Beusterien, Steinwald, & Ware, 1996; Wells, Burnam, Rogers, Hays, & Camp, 1992), loss of job within 1 year (Ware et al., 1994), 180-day survival (Rumsfield et al., 1999), and 5-year survival (Ware et al., 1994).

Clinical Uses

Using a cutoff score of 42, the MCS had a sensitivity of 74% and a specificity of 81% in detecting patients diagnosed with the depressive disorder (Ware et al., 1994). The three scales with the most mental factor content (MH, RE, and SF) in factor-analytic studies have been shown to be the most responsive in comparisons of patients before and after recovery from depression (Ware, 1995), change in the severity of depression (Beusterien et al., 1996), as well as drug treatment and

interpersonal therapy for depression (Coulehan, Schulberg, Block, Madonia, & Rodrigues, 1997).

Clinical studies have shown that three of the scales (PF, RP, and BP) with the greatest physical factor content tend to be the most responsive to the benefits of knee replacement (Kantz, Harris, Levitsky, Ware, & Davies, 1992), hip replacement (Kantz et al., 1992; Lansky, Butler, & Waller, 1992), and heart valve surgery (Phillips & Lanky, 1992).

References and Readings

Beusterien, K. M., Steinwald, B., & Ware, J. E. (1996). Usefulness of the SF-36® Health Survey in measuring health outcomes in the depressed elderly. *Journal of Geriatric Psychiatry and Neurology, 9*, 1–9.

Coulehan, J. L., Schulberg, H. C., Block, M. R., Madonia, M. J., & Rodrigues, E. (1997). Treating depressed primary care patients improves their physical, mental, and social functioning. *Archives of Internal Medicine, 157*, 1113–1120.

McHorney, C. A., Ware, J. E., Lu, J. F. R., & Sherbourne, C. D. (1994). The MOS 36-Item Short-Form Health Survey (SF-36®): III. Tests of data quality, scaling assumptions and reliability across diverse patient groups. *Medical Care, 32*(4), 40–66.

Kantz, M. E., Harris, W. J., Levitsky, K., Ware, J. E., & Davies, A. R. (1992). Methods for assessing condition-specific and generic functional status outcomes after total knee replacement. *Medical Care, 30* (Suppl. 5), MS240–MS252.

Lansky, D., Butler, J. B. V., & Waller, F. T. (1992). Using health status measures in the hospital setting: From acute care to 'outcomes management'. *Medical Care, 30*(Suppl. 5), MS57–MS73.

Phillips, R. C., & Lansky, D. J. (1992). Outcomes management in heart valve replacement surgery: Early experience. *Journal of Heart Valve Disease, 1*(1), 42–50.

Rumsfield, J. S., MaWhinney, S., McCarthy, M., Shroyer, A. L., Villa-Nueva, C. B., O'Brien, M., et al. (1999). Health-related quality of life as a predictor of mortality following coronary artery bypass graft surgery. *The Journal of the American Medical Association, 218*(14), 1298–1303.

Tsai, C., Bayliss, M. S., & Ware, J. E. (1997). *SF-36® Health Survey annotated bibliography: Second edition (1988–1996)*. Boston: Health Assessment Lab, New England Medical Center.

Turner-Bowker, D. M., Bartley, P. J., & Ware, J. E., Jr. (2002). *SF-36® Health Survey & "SF" bibliography: Third edition (1988–2000)*. Lincoln: QualityMetric Incorporated.

Ware, J. E. (1993). *SF-36 Health Survey: Manual and interpretation guide*. Boston: Nimrod Press.

Ware, J. E. (1995). The status of health assessment 1994. *Annual Review of Public Health, 16*, 327–354.

Ware, J. E., Gandek, B., & the IQOLA Project Group (1994). The SF-36® Health Survey: Development and use in mental health research and the IQOLA Project. *International Journal of Mental Health, 23*(2), 49–73.

Wells, K. B., Burnam, M. A., Rogers, W., Hays, R., & Camp, P. (1992). The course of depression in adult outpatients: Results from the Medical Outcomes Study. *Archives of General Psychiatry, 49*, 788–794.

SF-MPQ

► McGill Pain Questionnaire

SF-MPQ-2

► McGill Pain Questionnaire

Shaken Baby Syndrome (SBS)

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Synonyms

Abusive head trauma; Inflicted childhood neurotrauma; Nonaccidental or inflicted traumatic brain injury

Short Description or Definition

Shaken baby syndrome (SBS) is a form of inflicted traumatic brain injury in an infant or small child who is violently shaken (or thrown). Rapid rotation and movement of the brain within the cranial vault of a small child result in significant neural trauma.

Categorization

A form of child abuse, SBS occurs as a result of a baby's weak neck muscles in relation to its proportionally large head being unable to compensate for rapid shaking, accelerating movements, or impact to the head. The American Academy of Pediatrics states that the forces involved in accidental falls or play are not adequate for this type of injury, and rather the shaking or impact involved in SBS would be easily recognized by the observers as dangerous and life threatening for the child (AAP, 2001).

Epidemiology

Determination of frequency, morbidity, and mortality is difficult because oftentimes no clear, visible injury occurs.

In addition, given the abusive nature of the injury, significant motives to avoid detection often make investigating possible cases of SBS difficult. SBS likely occurs most often in children under 2 years of age, but can occur through 5 years of age. SBS can occur in the context of additional abusive injuries, either current or historical. A review of research indicates that up to 30–40% of children who present for medical care for SBS have evidence of prior injuries (www.dontshake.org), and children with prior additional abusive injuries often had nonspecific signs in the past of head trauma such that those nonfatal, non-life-threatening incidences were not detected. Overall mortality is estimated to range up to almost 40%, with upward of 60% of infants who have been shaken dying or experiencing profound long-term medical and cognitive consequences.

Natural History, Prognostic Factors, and Outcomes

In the early 1970s, the term "whiplash shaken baby syndrome" was used to describe symptoms consistent with SBS, which had been identified over 2 decades prior (Caffey, 1974). Over the last 4 decades, debate has centered around the methods for standardizing the diagnosis of SBS, as well as the implications of various nomenclature to describe the trauma. Most significantly, educational campaigns have gained momentum to help new parents expect intense crying, to recognize frustration that is theorized to lead to abusive shaking, and to leave the baby in a safe place and walk away. Unlike other forms of abuse, risk for SBS cuts across all social classes, races, and gender. Likely risk for perpetration includes nonrelated males, fathers, female caregivers, and mothers. More subtle signs of neurotrauma can be mistaken for flu-like symptoms or other common presentations of childhood (i.e., colic), and in some cases when a child is shaken into a comatose state, the caretaker may believe that the child has gone to sleep or finally stopped crying. Such complicating factors may result in significant delays before a child is brought for medical attention and lead to significant mortality rates. Children who survive SBS often experience blindness if they have had retinal hemorrhaging, as well as significant long-term neurological conditions such as mental retardation, seizure disorders, muscular spasticity or cerebral palsy, and structural abnormalities such as microcephaly, hydrocephaly, cerebral atrophy, or encephalomalacia.

Neuropsychology and Psychology (or Neurobiological Symptoms) of SBS

Symptoms of SBS vary with the severity of the inflicted neural trauma. The neural injury typically includes acceleration injury during shaking and deceleration when the shaking is stopped or the child is slammed or thrown into an object. The brain rotates around on the center of gravity, resulting in tearing of blood vessels on the surface of the brain and possibly also axonal shearing within the brain. As a result, the most common neurobiological effects of SBS include initial subdural hemorrhage, subarachnoid hemorrhage, or both. Because shaking is often in a forward to backward motion, the most common location for hemorrhage is retinal (believed to occur in the vast majority of cases), frontal, and occipital. Further, injury occurs with resultant cerebral edema. Additional pathology often includes spine and neck injuries, rib fractures, and fractures to the long bones of the infant (particularly if they have been held by an appendage while shaken). As a result of neurological pathology, immediate observable symptoms can include lethargy, irritability, a high-pitched cry, poor sucking and feeding, problems breathing, and a blue or pale pallor, vomiting, seizures, and even coma (often interpreted as sleeping initially). Long-term outcomes of these injuries are difficult to study and quantify in a rigorous manner. Therefore, most descriptions of SBS are based on the case studies, indicating that children who survive are more severely impaired as they experience more severe injuries, including lacerations of brain tissue, excessively elevated intracranial pressure, infarcts, and hemorrhage.

Evaluation

Diagnosis of SBS requires extensive medical evaluations including radiology studies as well as sociological evaluations of the caregivers. Evidence of additional physical injuries indicative of abuse on the child should be documented. The presence or absence of retinal hemorrhages should be evaluated by a pediatric ophthalmologist or neurologist. Laboratory values can indicate the presence of blood in the cerebral spinal fluid, coagulation changes following cerebral injury, and possible pancreas and liver damage. X-ray can reveal rib injuries. Most significantly, CT scan is the primary method of evaluation for brain injury. Consecutive CT scans can reveal ongoing changes in a recently injured child as well as the evidence of older brain injuries. Utilizing MRI as an adjunct in longer-term diagnosis can reveal more subtle white matter changes related to injury.

Treatment

Treatment of SBS most often involves life-saving measures initially, including neurosurgery to address active bleeding and excessive intracranial pressure. Medical life support may be required for some time while swelling subsides. Long-term treatment of SBS involves addressing the most salient of the resulting neurobiological symptoms. Children with more significant injuries often begin rehabilitation therapies immediately, much like other children who experience accidental brain injuries. Neuropsychological evaluations across development are often useful to detect specific cognitive, functional, or educational impairments, with the most common believed to be attention, executive, and visual perceptual deficits.

Cross References

- ► Cerebral Edema
- ► Subarachnoid Hemorrhage
- ► Traumatic Brain Injury

References and Readings

American Academy of Pediatrics Committee on Child Abuse and Neglect. (2001). Shaken Baby Syndrome: Rotational cranial injuries – technical report. *Pediatrics, 108*(1), 206–210.

Barlow, K. M., Thomson, E., Johnson, D., & Minns, R. A. (2005). Late neurologic and cognitive sequelae of inflicted traumatic brain injury in infancy. *Pediatrics, 116*, 174–185.

Caffey, J. (1974). The whiplash-shaken infant syndrome: Manual shaking by the extremities with whiplash-induced intra cranial and intra ocular bleeding, linked with residual permanent damage and mental retardation. *Pediatrics, 54*(4), 396–403.

National Institutes of Health, National Institute of Neurological Disorder & Stroke NINDS Shaken Baby Syndrome Information page: www.ninds.nih.gov/disorders/shakenbaby

Shaken Baby Association, Inc: www.shakenbaby.net/main.html

The National Center on SBS: www.dontshake.org

Shaking

- ► Essential Tremor
- ► Physiologic Tremor
- ► Postural Tremor
- ► Resting Tremor
- ► Tremor

Shaking Palsy

- ► Paralysis Agitans

Shearing Injury, Shear Strain

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Synonyms

Diffuse axonal injury

Definition

Shear injury is a traumatic brain injury that occurs as white matter and white matter connections are disrupted from acceleration–deceleration, or rotational acceleration mechanisms of force. The axons of neurons are disturbed from a biomechanical, and often also, a biochemical standpoint. These disconnections of white matter can result in axonal, and ultimately cell, death. Functional consequences of these injuries may be slowed cognitive processing speed, decreased motor coordination, disturbance in language function, and disturbance in higher-level executive functions.

Current Knowledge

Research is currently focused on defining clinical signs or hallmarks of shear strain injury. Although controversial, dilated Virchow–Robin spaces have been suggested to be a radiologic marker of shear strain injury following trauma (Inglese M et al., 2006). Further study is needed to determine the extent to which shear strain injuries are reversible with rehabilitation therapies and pharmacologic interventions in the acute and post-acute recovery stages.

Cross References

- ► Acceleration Injury
- ► Biomechanics of Injury
- ► Rotational Acceleration

References and Readings

Inglese, M., Grossman, R. I., Diller, L., Babb, J. S., Gonen, N., Silver, J. M., & Rusinek, H. (2006). Clinical significance of dilated Virchow-Robin spaces in mild traumatic brain injury. *Brain Injury, 20*, 15–21.

Scheid, R., Walther, K., Guthke, T., Preul, C., & Von Cramon, D. Y. (2006). Cognitive sequelae of diffuse axonal injury. *Archives of Neurology, 63*, 418–424.

Graham, D. I., Saatman, K. E., Marklund, N., Conte, V., Morales, D., Royo, N., & McIntosh, T. K. (2006). The neuropathology of trauma. In R. W. Evans (Ed.), *Neurology and Trauma* (2nd ed., pp. 45–94). New York: Oxford University Press.

Shell Shock

► Posttraumatic Stress Disorder

Sheltered Employment

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Synonyms

Sheltered workshop

Definition

Sheltered employment is a setting in which people with disabilities receive services and training to develop work-related skills and behaviors. "Sheltered," as a concept, originally implied an environment, which was safe and protected for individuals with disabilities. The first sheltered employment (workshop) was the Perkins Institute for the Blind established in 1840. Within the workshop setting, individuals are to be prepared for competitive employment. Workshops are segregated in nature.

In the field of rehabilitation, workshops have been criticized as programs in which people with significant disabilities were referred and remained for years, exploited, and/or did not see competitive employment outcomes in their community. Another criticism is that individuals with significant disabilities often have difficulty generalizing skills and knowledge from one environment to another. Thus, job readiness programs are not effective with this population. The focus of training and learning should occur in the actual environment in which the activity or task will be performed.

Typically, the individual working in a sheltered setting is paid a piece rate wage versus a competitive wage. Workshops solicit contracts with industries that are involved in mass production. As these industries decline, there is a concern that workshops cannot provide steady work for people with disabilities. In 2001, the Rehabilitation Services Administration of the US Department of Education amended the regulations governing the State Vocational Rehabilitation Program. This amendment redefines the term employment outcome defining it as an individual with a disability working in an integrated setting. Sheltered employment is no longer considered a successful employment outcome. However, in many communities, sheltered employment is still considered a placement option within a continuum of services provided to individuals with significant disabilities.

An alternative to sheltered employment is supported employment. This model focuses upon training individuals within the context of community-based employment settings where they receive competitive wages.

Cross References

► Customized Employment
► Supported Employment

References and Readings

Department of Education. (Monday, January 22, 2001). Part XVI 34 CFR Part 361 State Vocational Rehabilitation Services Program; Final Rule VerDate 11<MAY> 2000 19: 17 Jan 20, 2001 . . . http://www.ed.gov/legislation/FedRegister/finrule/2001-1/012201a.html

Kregel, J., & Dean, D. (2001). *Sheltered vs supported employment: A direct comparison of long-term earnings outcomes for individuals with cognitive disabilities.* Retrieved December 18, 2009, from http://www.worksupport.com/main/downloads/dean/shelteredchap3.pdf

Sheltered Workshop

► Sheltered Employment

Shenjing Shuairuo

► Neurasthenia

Shipley Institute of Living Scale

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Synonyms

Shipley-Hartford; SILS

Description

The Shipley Institute of Living Scale (SILS; Shipley & Burlingame, 1941) is a measure of intellectual deficit and provides a screening measure for intelligence. The SILS contains two sections: a multiple choice vocabulary section and a verbal reasoning section. The vocabulary section consists of 40 multiple-choice items requiring participants to select the one word out of four choices that is closest in meaning to a target word. The abstract reasoning section consists of 20 items requiring participants to determine solutions for abstract verbal and arithmetic problems. Total test administration time is approximately 25 min and requires a sixth grade reading level (Bradford, 1960).

Raw scores are derived based on the number of items correctly answered for each section of the test: Vocabulary, Abstraction and Total Score. Standardized scores, or the Conceptual Quotient (CQ) is a ratio of the abstraction score to the vocabulary score, and a Discrepancy Score is computed as vocabulary minus the abstraction score. Further, the manual allows for age-corrected *t*-score conversions of all three raw scores, as well as predicted Wechsler Adult Intelligence Scale (WAIS) and Wechsler Adult Intelligence Scale-Revised (WAIS-R) IQ scores from Total Scores. CQ scores can also be correlated to the likelihood of organic impairment using the manual. Age-corrected WAIS IQ estimates were added by Paulson and Lin (1970).

In addition to the Paulson and Lin (1970) age-corrected IQ estimates, which are generally considered to be superior to most other estimation approaches (Dennis, 1973; Jacobsen & Tamkin, 1988) for converting SILS scores to Wechsler Adult Intelligence Scale (WAIS) or Wechsler Adult Intelligence Scale-Revised (WAIS-R) IQ scores, there are a number of other published tables for these conversions including Bartz and Loy (1970), Grayson (1951), Tamkin & Jacobsen (1987), Sines & Simmons (1959), and Zachary (1986). Further, there are two published equations for converting SILS scores to WAIS-R IQ scores:

1. Zachary, Crumpton, and Spiegel (1985)

$$\text{WAIS-R IQ} = (((27.650 + (1.320 \times \text{SILS Vocabulary})) - (85.859 + (1.196 \times \text{Age}) - (0.017 \times \text{age}^2))/(12.481 = (0.581 \times \text{age}) - (0.006 \times \text{age}^2)) \times 15) + 100)$$

2. Watson et al. (1992)

$$\text{WAIS-R FSIQ} = 0.63 (\text{SILS IQ score derived using Zachary, 1986 table}) + 36.2$$

Historical Background

The SILS was developed as a measure of intellectual impairment in 1940 based on a normative sample of 1,046 individuals (Shipley, 1940).

Psychometric Data

The evidence for the reliability of the SILS is somewhat mixed. Although some studies report satisfactory test–retest reliabilities for the SILS (Nixon, Parsons, Schaeffer, & Hale, 1995), overall the SILS appears to have low test–retest reliabilities, especially on the abstraction scale (e.g., Goodman, Streiner, & Woodward, 1974). There is also mixed evidence regarding the relationship of SILS performance to age. More specifically, some studies suggest that SILS Vocabulary scores appear to increase with age (e.g., Harnish, Beatty, Nixon, & Parsons, 1994), while others report that Vocabulary scores do not change with age (e.g., Tamkin & Jacobsen, 1987). Similarly, there are mixed findings in regard to the relationship between age and Abstraction scores; while the majority of studies find that Abstraction scores decrease with age (e.g., Tamkin & Jacobsen, 1987), one study reported that it increases with age (Corotto, 1966), while others have been unable to find a significant relationship between the two (e.g., Harnish et al., 1994). There is more consistent evidence that the SILS CQ decreases with age (e.g., Harnish et al., 1994),

providing a rationale for why this score has fallen out of favor for use in clinical determinations. Further, almost all studies have been unable to find a relationship between SILS performance and gender (for review see Phay, 1990). Additionally, performance on the SILS appears to vary by race, with most studies reporting that African Americans perform poorer than Caucasians (e.g., Dalton & Dubnicki, 1981). Similarly, both higher SES status (Eisenthal & Harford, 1971) and more education appears to be positively related to test performance (e.g., Harnish et al., 1994).

Validity studies on the SILS have been promising. Concurrent validity, is adequate with significant relationships between the SILS and the Raven's Progressive Matrices Test (Eisenthal & Hartford, 1971), WAIS Full Scale IQ (FSIQ; 0.65–0.90; e.g., Sines & Simmons, 1959; Watson et al., 1992), WAIS-R FSIQ (0.65–0.79; for review see Phay, 1990), Army Alpha (Mathae, 1968), Wide Range Achievement Test (WRAT; Martin, Blair, & Vickers, 1979), and Wonderlic Personnel Test (Frisch & Jessop, 1989). The SILS also has acceptable predictive validity particularly with regard to WAIS and WAIS-R FSIQ scores (e.g., Frisch & Jessop, 1989).

Clinical Uses

Neurochemical data has confirmed that the left temporal and parietal lobes of the brain are involved in Abstraction performance (Parks, Barker, Dodrill, & Duara, 1985) and that the left frontal lobe is involved in both Vocabulary and Abstraction performance in patients with Alzheimer's disease (Parks et al., 1990). Further, there is evidence that the SILS differentiates individuals with and without brain injuries (e.g., Shipley & Burlingame, 1941), with and without cognitive problems due to alcohol dependence (e.g., Nixon et al., 1995), and with and without schizophrenia (Lewinsohn, 1963). Finally, the SILS may not be appropriate for use with individuals with subaverage intelligence (e.g., Watson, Klett, Kucala, Nixon, Schaefer, & Gasser, 1981). Therefore, the SILS is most often used as a brief screening instrument for intellectual impairment, as well as an indicator of premorbid ability (Vocabulary score) and appears to be an adequate measure of IQ testing when individual testing is not feasible (Dalton, Pederson, & McEntyre, 1987).

Cross References

- ▶ Abstract Reasoning
- ▶ Intelligence
- ▶ Kaufman Brief Intelligence Test
- ▶ Performance IQ
- ▶ Premorbid Estimate
- ▶ Reasoning
- ▶ Verbal IQ
- ▶ Wechsler Abbreviated Scale of Intelligence

References and Readings

Jacobsen, R. H., & Tamkin, A. S. (1988). Converting Shipley Institute of Living Scale Scores to IQ: A comparison of methods. *Journal of Clinical Psychology, 44*(1), 72–75.

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

Phay, A. J. (1990). Shipley Institute of Living Scale: Part 1—Moderator variables. *Medical Psychotherapy, 3*, 1–15.

References

Bartz, W., & Loy, D. (1970). The Shipley-Hartford as a brief IQ screening device. *Journal of Clinical Psychology, 26*, 74–75.

Bradford, E. J. (1960). Shipley-Institute of Living Scale for measuring intellectual impairment. In O. K. Buros (Ed.), *The third mental measurements yearbook,* (pp. 194–195). Highland Park, NJ: Gryphon Press,

Corotto, L. V. (1966). Effects of age and sex on the Shipley-Institute of Living Scale. *Journal of Consulting Psychology, 30*(2), 179.

Dalton, J. E., & Dubnicki, C. (1981). Sex, race, age, and education variables in Shipley-Hartford scores of alcoholic inpatients. *Journal of Clinical Psychology, 37*(4), 885–888.

Dalton, J. E., Pederson, S. L., & McEntyre, W. L. (1987). A comparison of the Shipley vs. WAIS-R subtests in predicting WAIS-R Full Scale IQ. *Journal of Clinical Psychology, 43*(2), 278–280.

Dennis, D. (1973). Predicting Full Scale WAIS IQs with the Shipley-Hartford. *Journal of Clinical Psychology, 29*, 366–368.

Eisenthal, S., & Hartford, T. (1971). Correlation between the raven progressive matrices scale and the Shipley Institute of Living Scale. *Psychological Reports, 24*, 213–215.

Frisch, M. B., & Jessop, N. S. (1989). Improving WAIS-R estimates with the Shipley-Hartford and Wonderlic personnel tests: Need to control for reading ability. *Psychological Reports, 65*, 923–928.

Goodman, J. T., Streiner, D. L., & Woodward, C. A. (1974). Test-retest reliability of the Shipley Institute of Living Scale practice effects or random variation. *Psychological Reports, 35*, 351–354.

Grayson, H. (1951). *Psychological admissions testing program and manual.* Los Angeles: Veterans Administration Center.

Harnish, M. J., Beatty, W. W., Nixon, S. J., & Parsons, O. A. (1994). Performance by normal subjects of the Shipley Institute of Living Scale. *Journal of Clinical Psychology, 50*(6), 881–883.

Jacobsen, R. H., & Tamkin, A. S. (1988). Converting Shipley Institute of Living Scale Scores to IQ: A comparison of methods. *Journal of Clinical Psychology, 44*(1), 72–75.

Lewinsohn, A. J. (1963). Use of the Shipley-Hartford conceptual quotient as a measure of intellectual impairment. *Journal of Consulting Psychology, 27*, 444–447.

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
Martin, J. D., Blair, G. E., & Vickers, D. M. (1979). Correlation of the quick word test and the wide range vocabulary test with the Shipley-Hartford Institute of Living Scale. *Educational and Psychological Measurement, 39*, 935–937.
Mathae, D. E. (1968). Norms and correlations of scores from the modified alpha examination form 9 and the Shipley Institute of Living Scale for hospitalized female neuropsychiatric inpatients. *Newsletter of Research in Psychology, 10*, 22–26.
Nixon, S. J., Parsons, O. A., Schaeffer, K. W., & Hale, R. L. (1995). A methodological study of the Shipley Institute of Living Scale in alcoholics and non-alcoholics: Reliability, discriminating items, and alternate forms. *Applied Neuropsychology, 2*, 155–160.
Parks, R. W., Barker, W. W., Dodrill, K., & Duara, R. (1985). Shipley Institute of Living Scale (SILS) and positron emission tomography in dementia. *Journal of Clinical and Experimental Neuropsychology, 7*, 617.
Parks, R. W., Cassens, G., Crockett, D. J., Herrera, J. A., Latterner, R., Lorenzo, G., et al. (1990). Correlation of the Shipley Institute of living Scale with regional cerebral glucose metabolism as measured by positron emission tomography in dementia. *International Journal of Clinical Psychology, XII*, 14–19.
Paulson, M., & Lin, T. (1970). Predicting WAIS IQ from Shipley-Hartford scores. *Journal of Clinical Psychology, 26*, 453–461.
Phay, A. J. (1990). Shipley Institute of Living Scale: Part 1—Moderator variables. *Medical Psychotherapy, 3*, 1–15.
Shipley, W. C. (1940). A self-administered scale for measuring intellectual impairment and deterioration. *Journal of Psychology, 9*, 371–377.
Shipley, W. C., & Burlingame, C. C. (1941). A convenient self-administered scale for measuring intellectual impairment in psychotics. *American Journal of Psychiatry, 97*, 1313–1325.
Sines, L. K., & Simmons, H. (1959). The Shipley-Hartford Scale and the Doppelt Short Form as estimators of WAIS IQ in a state hospital population. *Journal of Clinical Psychology, 15*, 452–453.
Tamkin, A. S., & Jacobsen, R. H. (1987). Age-corrected norms for Shipley Institute of Living Scale Scores derived from psychiatric inpatients. *Journal of Clinical Psychology, 43*(1), 138–142.
Watson, C. G., Klett, W. G., Kucala, T., Nixon, C., Schaefer, A., & Gasser, B. (1981). Prediction of WAIS scores from the 1973 Henmon-Nelson Revision. *Journal of Clinical Psychology, 37*, 840–842.
Watson, C. G., Plemel, D., Schaefer, A., Raden, M., Alfano, A. M., Anderson, P. E. D., et al. (1992). The comparative concurrent validities of the Shipley Institute of Living Scale and the Henmon-Nelson tests of mental ability. *Journal of Clinical Psychology, 48*(2), 233–239.
Zachary, R. A. (1986). *Shipley Institute of Living Scale: Revised manual.* Los Angeles, CA: Western Psychological Services.
Zachary, R. A., Crumpton, E., & Spiegel, D. E. (1985). Estimating WAIS-R IQ from the Shipley Institute of Living Scale. *Journal of Clinical Psychology, 41*(4), 532–540.

Shipley-Hartford

► Shipley Institute of Living Scale

Short IQCODE

► Informant Questionnaire on Cognitive Decline in the Elderly

Short Physical Performance Battery

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Synonyms

SPPB

Description

The Short Physical Performance Battery (SPPB) is a performance-based test of lower extremity function designed for elderly participants. It consists of three parts: the Balance Test, the Gait Speed Test, and the Chair Stand Test. In the Balance Test, the participant holds his/her balance for 10 s in three standing positions with eyes open: feet side by side, feet in semi-tandem stance (big toe of one foot touching heel of other foot), and feet in tandem stance (heel to toe). Each stance is demonstrated by the tester before the participant attempts it; clear instructions are given regarding what the participant can and cannot do to maintain his/her balance (i.e., can bend knees or put arms out, but should not move his/her feet or use an other aid), and the examiner remains close to the participant in order to assist in the event he/she cannot maintain his/her balance. If the participant fails one aspect of the test, subsequent aspects are not to be attempted. Only one attempt is permitted for each stance. In the Gait Speed Test, participants walk a 4-m marked course at their usual walking pace, with the examiner timing their walk with a stopwatch. A 3-m course may be substituted if a 4-m space is not available. Two attempts are allowed on this test, with only the fastest recorded time being used for the overall score. In this portion of the test, assistive devices may be used, but if the participant feels safe to walk the course without them, then this is preferred. The Chair Stand Test examines a

person's ability to rise from a sitting to a standing position from an armless chair. After examiner demonstration, the participant is asked to repeat one chair stand with his/her arms folded across his/her chest. If they are unable to rise, they are allowed to use their arms to assist them, but the test is then discontinued. If successful on the first stand, the examiner demonstrates the final part of the SPPB, a series of five consecutive chair stands, which should be performed as quickly as possible. The examiner times the participant's performance with a stopwatch, counting aloud the number of stands completed, but providing no other encouragement. Video-based training for administering the SPPB is available (resources and instructional material can be downloaded from http://www.grc.nia.nih.gov/branches/ledb/sppb/index.htm), and must be completed prior to administering the battery for research or clinical use. The SPPB can be administered in approximately 10 min. No aspect of the test should be attempted unless both the examiner and participant feel it is safe to do so. If a participant fails any aspect of the test, the type of failure must be recorded on the score sheet (e.g., participant felt it was unsafe to attempt, maintained balance for 4 s only, etc.).

Historical Background

The SPPB was developed by Jack Guralnik and colleagues as part of a National Institute on Aging project, the Established Populations for Epidemiologic Studies of the Elderly (EPESE). It has been used in large-scale epidemiological studies, and as such has an excellent normative basis.

Psychometric Data

Guralnik et al. (1994) provide gender- and age-stratified norms for the SPPB subtests (where age is stratified into bands of 71–79 and 80+ years). They also reported that there was increased risk of admission to a nursing home, and of death, with decreasing scores on the SPPB, showing its predictive validity. Guralnik, Ferrucci, Simonsick, Salive, and Wallace (1995) found that those with low SPPB scores, despite having no objective disability, were more likely than people with high SPPB scores at baseline to have a disability involving mobility and/or Activities of Daily Livings (ADLs) 4 years later. Specifically, those with the lowest SPPB scores were four to five times more likely than those with the highest SPPB score to have a disability 4 years later, with intermediate scores showing a linear reduction in risk. Subsequent analyses (Guralnik et al., 2000) have found that the score from the Gait Speed Test alone is almost as good a predictor of subsequent disability as the SPPB total score, and equations are provided for estimating the probability of future disability in either mobility or ADLs both 1 and 4 years from testing. Ostir, Volpato, Fried, Chaves, and Guralnik (2002) have investigated the reliability of the SPPB, along with its responsiveness to change. They found that test–retest reliability at a 1-week interval was excellent (Intra-Class Coefficients (ICCs) from 0.88 to 0.92 for SPPB scores from three pairs of weeks from a dataset comprising weekly testing on for a 6-month period). The longer-term reliability was lower but still acceptable, with average ICCs falling from 0.77 at 6 months to 0.51 at 36 months. The sensitivity to change was examined by comparing scores of people who experienced medical events (myocardial infarction, congestive heart failure, stroke, or hip fracture) during the course of the study, prior to and following the event, against those who did not experience such an event. Change scores were significantly greater for people who experienced one of the listed medical events than for those who did not, and scores for those who experienced a medical event were also found to rise upon later testing, suggesting the measure is responsive to change.

Clinical Uses

The SPPB is a good means of evaluating lower extremity function's impact upon activities of daily living. It can be useful in identifying ADLs that the participant may have problems with, and consequently the degree of support that is needed. It can also be used for identifying people at risk of disability, and hence those who may benefit from intervention strategies or programs.

Cross References

► Six Minute Walk Test

References and Readings

Guralnik, J. M., Ferrucci, L., Pieper, C. F., Leveille, S. G., Markides, K. S., Ostir, G. V., et al. (2000). Lower extremity function and subsequent disability: Consistency across studies, predictive models, and value of gait speed alone compared with the short physical performance battery. *Journal of Gerontology: Medical Sciences, 55A*, M221–M231.

Guralnik, J. M., Ferrucci, L., Simonsick, E. M., Salive, M. E., & Wallace, R. B. (1995). Lower-extremity function in persons over the age of 70 years as a predictor of subsequent disability. *The New England Journal of Medicine, 332*, 556–561.

Guralnik, J. M., Simonsick, E. M., Ferrucci, L., Glynn, R. J., Berkman, L. F., Blazer, D. G., et al. (1994). A short physical performance battery assessing lower extremity function: Association with self-reported disability and prediction of mortality and nursing home admission. *Journal of Gerontology: Medical Sciences, 49*, M85–M94.

Ostir, G. V., Volpato, S., Fried, L. P., Chaves, P., & Guralnik, J. M. (2002). Reliability and sensitivity to change assessed for a summary measure of lower body function. Results from the Women's Health and Aging Study. *Journal of Clinical Epidemiology, 55*, 916–921.

Short-Form 12

► SF-36/SF-12

Short-Form 36

► SF-36/SF-12

Short-Fuse Syndrome

► Frustration Tolerance

Short-Term Memory

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Synonyms

Active memory; Primary memory

Definition

Short-term memory holds a limited amount of information to be used for a short period of time. When rehearsal is prevented and thus the short-term memories are not consolidated into long-term memories, the period of time that short-term memory is held is for a few seconds. The magic number, coined by George Miller, for short-term memory is seven items, plus or minus two items.

Current Knowledge

Difference Between Short-Term Memory and Working Memory

Short-term memory is different from working memory, as working memory is more involved in temporarily storing and manipulating information; short-term memory, on the other hand, can be consolidated to long-term memories. Unlike working memory, decay appears to be the most common cause for the loss of short-term memory. According to Baddley's 1986 model for working memory, there are two types of short-term memory storages: the phonological loop, which deals with auditory input, and the visuospatial sketchpad, which is concerned with visual memory. Memories can be retained for a longer period through rehearsal, which can also aid in the process of consolidating short-term memories into long-term memories.

Memory Span

While the average span of short-term memory is seven items, plus or minus two, it is possible to increase one's memory span. Chunking involved grouping information together with a meaningful connection, and thus expanding an individual's ability to remember items in the short term. One documented case had an All-American cross-country runner's memory span at 79 digits, as he grouped the list of numbers read to him as race times. However, he was unable to perform as well when the items were not numbers, as they no longer had the same meaningful significance. The method behind chunking can also work for overall memory: by linking either a visual image or a significance to a piece of information, it can increase the likelihood of retention.

Amnesia

In anterograde amnesia, short-term memory remains unaffected; rather, damage to the hippocampus prevents the short-term memories from being converted into

long-term memories. In the case of H.M., he could retain his short-term memory until he was distracted, then it would be lost. Instead, he relied upon long-term memories created before his brain damage as reference. Otherwise, his short-term memory remained undisrupted and had the same span as that of a normal adult. Through studies done on H.M, the duration of short-term memories have been shown to not last much longer than a few seconds. Without consolidating into long-term memories, the information will not be retained.

Selective Attention

In addition, short-term memories are incredibly tied in with selective attention. According to Broadbent's theory, human cognition filters out multiple messages to focus on a single message. Broadbent's theory, also known as the "single channel hypothesis," can be illustrated by the cocktail party problem proposed by Colin Cherry – while surrounded by multiple conversations at a cocktail party, people are able to filter through them and pay attention to the conversation that interests them the most, and thus retain that pertinent information. Just as selective attention is needed to filter information, a diversion in selective attention can result in the loss of information retained only through short-term memory.

Future Directions

Functional magnetic resonance imaging (fMRI) is being used to see how the brain retains information. Research by the University of Oregon and University of California San Diego used fMRIs to record brain activity in the visual cortex 10 s after exposure to an object, when the brain was involved with memory and storage processing.

Cross References

- ▶ Memory
- ▶ Recent Memory
- ▶ Working Memory

References and Readings

Baddeley, A. D., Thomson, N., & Buchanan, M. (1975). Word length and the structure of short term memory. *Journal of Verbal Learning and Verbal Behavior, 14*, 575–589.

Broadbent, D. (1958). *Perception and communication*. London: Pergamon Press.

Cherry, E. C. (1953). Some experiments on the recognition of speech with one and two ears. *Journal of the Acoustical Society of America, 25*, 975–979.

Ericsson, K. A., Chase, W. G., & Faloon, S. (1980). Acquisition of a memory skill. *Science, 208*, 1181–1182.

Miller, G. (1956). The magical number seven, plus or minus two: Some limits on our capacity for processing information. *Psychological Review, 63*, 81–97.

Serences, J. T., Ester, E. F., Vogel, E. K., & Awh, E. (2009). Stimulus-specific delay activity in human primary visual cortex. *Psychological Science, 20*, 207–214.

Shuffling

▶ Festination

Shunts

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Synonyms

Lumboperitoneal shunt (LP shunt); Ventriculoatrial shunt (VA shunt); Ventriculoperitoneal shunt (VP shunt); Ventriculopleural shunt

Definition

Shunts are used for treatment of Hydrocephalus, a disorder where the fluid within and around the brain and spinal cord is either over-produced, under-absorbed, or its normal circulation impaired. Approximately 150 ml is produced per day and, in normal persons, it remains under regulated pressure.

Cerebral shunt systems are surgically placed to drain excess cerebrospinal fluid (CSF) from the ventricles or other parts of the central nervous system. Ventricular shunting remains the most common treatment for hydrocephalus. The most common shunt used is the ventriculoperitoneal (VP) shunt. A catheter is placed in the lateral ventricle of the nondominant hemisphere. It is attached,

subcutaneously, through a one-way, pressure-regulated valve to a catheter entering the peritoneal cavity. The excess CSF is drained into the peritoneal cavity. There are different shunt-valve types available to meet patient needs. Programmable, variable pressure valves are now available, which allows a controlled titration of pressures to the patient's symptoms.

Other types of shunts are ventriculoatrial, ventriculopleural, and lumboperitoneal.

Cross References

► Hydrocephalus
► Ventricles

References and Readings

Gildenberg, P., & Tasker, R. (1998). *Textbook of stereotactic and functional neurosurgery*. New York: McGraw-Hill Professional.
Greenberg, M. S. (2006). *Handbook of neurosurgery* (6th ed., p. 366). New York: Thieme Medical Publishers.
http://adam.about.com/encyclopedia/Ventriculoperitoneal-shunt.htm
http://www.hydroassoc.org/newsletter/shunt.html
Kaye, A. (2005). *Essential neurosurgery* (3rd ed.). Malden, MA: Blackwell Publishing.
Schmidek, H. & Roberts, D. W. (2000). *Operative neurosurgical techniques: Indications, methods, and results* (5th ed.). Philadelphia: W.B. Saunders Company.

Shy–Drager Syndrome

► Multisystem Atrophy (MSA)

SI

► Suicidal Ideation

SIB

► Severe Impairment Battery

SIB-S

► Severe Impairment Battery

Sick Building Syndrome

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Synonyms

BRI; Building-related illness; Medically unexplained symptoms (MUS); SBS; Tight building syndrome

Definition

Sick building syndrome (SBS) is an illness associated with a constellation of vague symptoms that occur as the result of an individual's occupation of a specific building. A building is diagnosed as "sick" based on the density of complaints made by building occupants (Godish, 1995; Murphy, 2006).

Categorization

A list of typical symptoms associated with SBS is shown in Table 1. These symptoms appear with prolonged exposure to a particular environment, often dissipating within an hour after leaving that space, such as when going home from work for the day, during weekends, or going on vacation.

Epidemiology

The WHO estimated that as many as 30% of newly constructed buildings would be classified as "sick buildings." There are no widely accepted standards regarding when it is appropriate to designate a building as "sick," but in practice, having at least 20% of the occupants suffering from frequent symptoms of the types described below seems to be the norm (Godish, 1995). Below this level, a building is considered "healthy."

Sick Building Syndrome. Table 1 Symptoms associated with Sick Building Syndrome

	Symptoms associated with Sick Building Syndrome
Physiological	• Mucus membrane irritation: Eye, nose, throat
	• Upper respiratory: Dry cough, high frequency of airway infections
	• Hoarseness
	• Wheezing
	• Skin: Scaly, itchy
	• Asthma-like symptoms
	• Vomiting
	• Diarrhea
	• Unspecific hypersensitivity: Unpleasant odor and taste perceptions
	• Erythema
Psychological	• Headache
	• Nausea
	• Dizziness
	• Confusion
	• Difficulty in concentrating
	• Fatigue
	• Mental fatigue

Natural History, Prognostic Factors, Outcomes

Sick building syndrome was first researched by the World Health Organization in the early 1980s. It was during this time that energy efficiency was the priority in building construction, making buildings "tighter" with better insulation, reduced ventilation, and reduced heating and cooling costs. High rise buildings were being constructed more frequently with windows that cannot be opened. The buildings were to be more energy efficient, using recirculated air. This recirculated air led to an accumulation of chemical compounds within the buildings and also created conditions well suited for the growth of an assortment of molds and mildews (Godish, 1995).

The illness was initially seen as psychological only, manifesting in those individuals who did not want to be at work or who had psychological dysfunction. Over time, studies have revealed that there are indoor air quality factors that seem to play a significant role giving this illness more medical credibility (Turiel, 1985). The costs of the revealed association of these illnesses to indoor air quality have resulted in some insurance companies denying or limiting mold and/or water damage coverage.

Psychology of SBS

There were some scientists and physicians who initially considered sick building syndrome as a form of mass psychogenic illness or mass hysteria. This diagnosis was often issued to women and illness episodes among women. Mass psychogenic illness is a psychosomatic disorder, the shared collective symptoms and beliefs found among two or more individuals with no identifiable causal agent (Murphy, 2006). Sick building syndrome was considered as a mass psychogenic illness through an explanation suggesting that it was a misattribution of symptoms to a physical external cause rather than its true psychological origin (Murphy, 2006; Turiel, 1985). With time and improved technologies for detection and measurement, causal attribution for sick building syndrome shifted from psychological causes to environmental causes – specific physical environmental causes within buildings could be better identified.

Evaluation

It is difficult to identify the causal agents for those afflicted with symptoms attributed to SBS, but factors to consider generally come from three levels: (1) workplace and organizational, (2) building, and (3) individual. The workplace/organization involves factors related to the job, such as equipment used, number of people assigned per office, job characteristics, and stress. Building factors include a wide range of elements including air-conditioning, adequacy of ventilation, air temperature, availability of natural light, condition of building systems, recent renovations, and air quality (Leslie and Lunau, 1992). Personal, or individual level, factors include locus of control, acute illness, smoking behavior, job satisfaction, gender, and age.

Treatment

Treatment for SBS occurs at the building level, once potential causal agents are identified. Building investigations

are typically only conducted when occupant complaints become prolific enough to overcome the skepticism of building management. Diagnosis is usually conducted by industrial hygiene or specialized indoor air quality consulting firms.

Cross References

► Multiple Chemical Sensitivity

References and Readings

Godish, T. (1995). *Sick buildings: Definition, diagnosis and mitigation.* Boca Raton, FL: CRC Press.

Leslie, G. B., & Lunau, F. W. (1992). *Indoor air pollution: Problems and priorities.* Cambridge, UK: Cambridge University Press.

Murphy, M. (2006). *Sick building syndrome and the problem of uncertainty.* Durham, NC: Duke University Press.

Turiel, I. (1985). *Indoor air quality and human health.* Stanford, CA: Stanford University Press.

Sickle-Cell Anemia

► Sickle-Cell Disease

Sickle-Cell Disease

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Synonyms

Hemoglobin SS disease; Sickle-cell anemia

Definition

Sickle-cell disease is a hereditary condition in which red blood cells form abnormal crescent shapes, instead of their usual disk shapes. These abnormal cells tend to clump together more frequently than do normal red blood cells, and because of this clumping, they occlude blood vessels more readily than do normal cells.

Current Knowledge

The blood vessel blockage in sickle-cell disease causes a variety of conditions. Pain episodes ("pain crises") occur in almost all patients, affecting any or all of the bones in the body, and lasting several hours to several days, at times requiring hospitalization. Ischemia from the lack of blood supply can result in strokes, skin ulcers, and internal organ damage. Other associated problems include abdominal pain, breathlessness, fatigue, fever, pallor, excessive thirst, rapid heart rate, reduced vision, and delayed growth. Causes of death include organ failure and infection. Some people with the disease experience minor, brief, and infrequent episodes. Others experience severe, long-term, frequent episodes with many complications.

Monitoring and treatment of patients with sickle-cell disease should be ongoing, even when there is no crisis. It is directed toward reducing the frequency, duration, and severity of the crises and complications. Folic acid supplements to produce red blood cells, maintaining fluid intake to reduce the cell aggregation, and pain medications to reduce the pain are usual methods of management. Occasionally, certain medications like hydroxyurea are used to reduce the number of pain episodes, and blood transfusions may be administered to treat a sickle-cell crisis. Bone marrow transplant is showing promise as treatment for the disease, but this procedure is associated with many complications.

Sickle-cell anemia is caused by an abnormal type of hemoglobin called hemoglobin S, which distorts the shape of red blood cells, especially when there are low levels of oxygen. These distorted red blood cells are shaped like crescents or sickles, and deliver less oxygen to the body's tissues. They also can clog more easily in small blood vessels, and break into pieces that disrupt blood flow, causing ischemia. Sickle-cell anemia is inherited from both parents. Sickle-cell disease is much more common in people of African and Mediterranean descent than in Caucasians. It is also seen in people from South and Central America, the Caribbean, and the Middle East.

Cross References

► Ischemia

References and Readings

Ballas, S. K., Lieff, S., Benjamin, L. J., Dampier, C. D., Heeney, M. M., Hoppe, C., et al. (2010). Investigators, comprehensive sickle cell

centers: definitions of the phenotypic manifestations of sickle cell disease. *American Journal of Hematology, 85*, 6–13.
Roseff, S. D. (2009). Sickle cell disease: a review. *Immunohematology, 25*, 67–74.

Sickness Impact Profile

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Synonyms

SIP; SIP-68

Definition

The sickness impact profile (SIP) was designed to measure perceived health status and to be used as an outcome measure for evaluation, program planning, and policy formation related to health care (Bergner, Bobbitt, Carter, & Gilson, 1981). It was intended to be sensitive to changes and/or differences in health status and to be applicable across a broad range of types and severities of illness, as well as different demographic and cultural groups. The final form of the original SIP, published in 1981, contains 136 items concerning 12 areas of activity: sleep and rest; eating; work; home management; recreation and pastimes; ambulation; mobility; body care and movement; social interaction; alertness behavior; emotional behavior; and communication. In 1994, a shorter version of the SIP, the SIP-68, was created and evaluated; this version contained 68 items in six areas of activity: somatic autonomy; mobility control; psychological autonomy and communication; social behavior; emotional stability; and mobility range (deBruin, Buys, deWitte, & Diederiks, 1994).

For both the SIP and SIP-68, individuals endorse items that are related to their health status and that apply to their situation on the day that they fill out the test. Scores are computed by adding a weight to each statement and are expressed as a percentage of the total dysfunction possible; higher scores denote more disability or lower health status. For the SIP-68, a total score is obtained by summing the number of endorsed items, with a 0 corresponding to "best health" and a 68 representing "worst health."

Current Knowledge

The test-retest reliability for the full SIP was shown to be high ($r = 0.92$) in a random sample of 53 participants with a 23 h test-retest interval (Bergner et al., 1981). Internal consistency was also high (Cronbach's alpha = 0.94). Correlations among SIP scores and self-assessment of sickness and dysfunction, clinician assessment of sickness and dysfunction, and an index of disability derived from the National Health Interview Survey ranged between 0.40 and 0.69, with the highest correlation reported between SIP scores and self-assessment of dysfunction. The SIP has shown good utility in a wide range of conditions and impairments; however, it does demonstrate severe ceiling effects. For a more complete review, please see Andresen & Meyers, 2000.

For the SIP-68, retest intraclass correlations were above 0.75 for all scales and dimensions except physical ($r = 0.61$). Proxy reliability is reported to range between 0.26 for the psychological autonomy and communication scale to 0.85 for somatic autonomy. Correlations between the SIP-68 and SIP were 0.94 (Nanda, McLendon, Andresen, & Armbrecht, 2003). Comparisons with the appropriate SF-36 scales showed moderate correlations, with the highest for the physical health scales.

Cross References

► SF-36/SF-12

References and Readings

Andresen, E. M., & Meyers, A. R. (2000). Health-related quality of life outcomes measures. *Archives of Physical Medicine & Rehabilitation, 81*(Suppl. 2), S30–S45.
Bergner, M., Bobbitt, R. A., Pollard, W. E., Martin, D. P., & Gilson, B. S. (1976). The sickness impact profile: Validation of a health status measure. *Medical Care, 14*, 57–67.
Bergner, M., Bobbitt, R. A., Carter, W. B., & Gilson, B. S. (1981). The sickness impact profile: Development and final revision of a health status measure. *Medical Care, 19*, 787–805.
deBruin, A. F., Buys, M., deWitte, L. P., & Diederiks, J. P. M. (1994). The sickness impact profile: SIP68, a short generic version. First evaluation of the reliability and reproducibility. *Journal of Clinical Epidemiology, 47*, 863–871.
Nanda, U., McLendon, P. M., Andresen, E. M., & Armbrecht, E. (2003). The SIP68: An abbreviated sickness impact profile for disability outcomes research. *Quality of Life Research, 12*, 583–595.

Signal Detection Theory

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Definition

Decision making is often associated with uncertainty. Signal detection theory (SDT) provides a framework for systematically characterizing decision making in the presence of uncertainty, taking into account that the outcome of a decision making process is attributable to both objective information and subjective bias of the observer. SDT has a wide range of applications, including in psychophysical experiments, examination of cognitive processes such as attention and memory, and medical and psychological diagnosis.

Historical Background

SDT is considered an extension of the psychophysical theory of Gustav Theodor Fechner (1801–1887), and later of Louis Leon Thurstone (1887–1955). Chapter 1 of Swets (1996) provides a comprehensive overview of the development of SDT.

Current Knowledge

A simple yes/no detection task will be used here to illustrate the basic concepts of SDT. Discussion of more advanced applications can be found in the references below. Consider a hypothetical example involving the use of a newly developed measure for diagnosing a disorder. The measure was administered to a pool of individuals, 100 of which were healthy, and 100 were previously identified with the disorder. In this context, the disorder represents the *signal* to be detected, and healthy individuals without the disorder represent the *noise* in the detection. A criterion value was chosen so that individuals who scored more than 1 standard deviation (SD) above the normative sample mean were classified as meeting the diagnosis. We denote this criterion value in units of SD by λ. Using $\lambda = 1$, it was found that 80 patients with the disorder, and 84 healthy individuals were correctly classified. Accordingly, 20 patients were misclassified as healthy, and 16 healthy individuals were misclassified as patients. The proportion of correctly diagnosed patients is referred to as the *hit rate* (h = 80%). The proportion of healthy individuals misclassified as patients is termed the *false alarm rate* (f = 16%). The corresponding rates of *misses* (m) and *correct rejections* (cr) are 20% (= 1 – h) and 84% (= 1 – f), respectively.

Consider the scenario where a decreased false alarm rate is desired, so that fewer healthy individuals are misclassified as patients. A more conservative λ of 1.4 is selected, with the following outcome.

As can be seen, the decrease in false alarm rate was accompanied by a corresponding decrease in hit rate. In other words, there is a trade-off between the ability to correctly detect the presence of a signal and the ability to correctly reject a lack of signal (also see entries on ▶ Sensitivity and Specificity). This example illustrates the utility of examining the hit and false alarm rates together for the characterization of a detection task as the criterion level changes.

In the SDT framework, test scores for patient and healthy individuals are considered random variables, denoted here by X_s (for signal) and X_n (for noise). Assuming that X_s and X_n follow normal distributions with equal variance, the following figures illustrate the distributions of test scores in the two groups, along with the hit and false alarm rates at two different criterion levels.

Assigning the mean of the noise distribution $(\mu_n) = 0$, and the mean of the signal distribution $(\mu_s) = d'$, with equal variance between the distributions, $\sigma^2 = 1$, we have

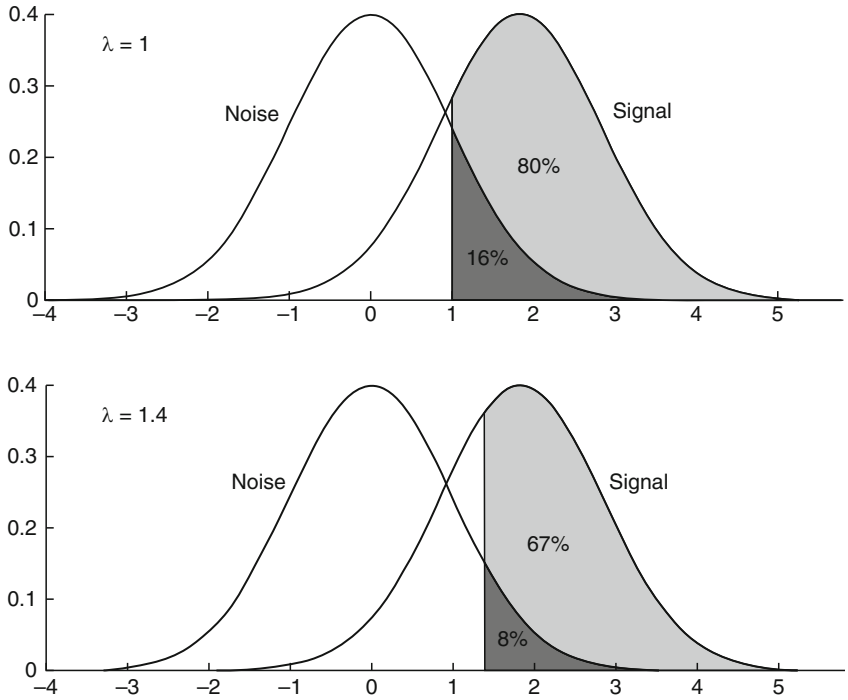
$$X_n \sim N(0,1) \text{ and } X_s \sim N(d',1)$$

Signal Detection Theory. Table 1

$\lambda = 1$	Response	
	No	Yes
Noise	84% (*cr*)	16% (*f*)
Signal	20% (*m*)	80% (*h*)

Signal Detection Theory. Table 2

$\lambda = 1.4$	Response	
	No	Yes
Noise	92% (*cr*)	8% (*f*)
Signal	33% (*m*)	67% (*h*)



Signal Detection Theory. Figure 1 Distributions of test scores in patients (X_s: signal) and healthy individuals (X_n: noise). Setting the criterion level (λ) higher results in a decreased false alarm rate. However, this also results in a correspondingly decreased hit rate. (See Tables 1 and 2 above)

Thus, the hit and false alarm rates can be expressed in terms of cumulative standard normal distribution function (Φ).

$$f = 1 - \Phi(\lambda) \text{ and } h = 1 - \Phi(\lambda - d')$$

The parameter d' describes the distance between the noise and signal distributions ($d' = \mu_s - \mu_n$), and therefore provides a measure of signal strength. Given these relationships, the parameters λ and d' can be estimated from observed hit and false alarm rates as

$$\lambda = -\Phi^{-1}(f) \text{ and } d' = \Phi^{-1}(h) - \Phi^{-1}(f)$$

where Φ^{-1} denotes the inverse cumulative standard normal distribution function.

To further characterize a signal detection task, it is often helpful to obtain a measure of response bias, that is, a preference to respond either *yes* or *no* to a detection task. Two common measures of response bias are presented here. λ_{center} (sometimes denoted c in the literature) expresses the position of the criterion value relative to a point halfway between the signal and the noise distributions. $\lambda_{\text{center}} = 0$ therefore indicates a lack of *yes* or *no* response bias, and $\lambda_{\text{center}} < 0$ indicates a *yes* bias and vice versa.

Signal Detection Theory. Table 3

λ	h	f	d'	λ_{center}	log β
1	80%	16%	1.84	0.08	0.14
1.4	67%	8%	1.85	0.48	0.89

$$\lambda_{\text{center}} = \lambda - \frac{d'}{2}$$

The ratio β reflects the relative heights of the signal and noise probability density functions at the criterion value. β has a value of 1 when there is no response bias. A drawback of β is the fact that its values are asymmetrical on either side of 1 such that $\min(\beta) = 0$ and $\max(\beta) = \infty$. β is therefore commonly expressed in its natural logarithm to eliminate the asymmetry. Thus, $\log \beta = 0$ indicates a lack of response bias, and $\log \beta < 0$ indicates a *yes* bias and vice versa.

$$\log\beta = d'\left(\lambda - \frac{d'}{2}\right) = \frac{\left[\Phi^{-1}(f)\right]^2 - \left[\Phi^{-1}(h)\right]^2}{2}$$

The following table shows the hit and false alarm rates, signal strength, and response bias measures for the hypothetical detection task above at two criterion levels of $\lambda = 1$ and 1.4.

Cross References

- ▶ Receiver Operating Characteristics Curve (ROC)
- ▶ Sensitivity
- ▶ Specificity

References and Readings

Green, D. M., & Swets, J. A. (1966). *Signal detection theory and psychophysics.* New York: Wiley.
Swets, J. A. (1996). *Signal detection theory and ROC analysis in psychology and diagnostics: Collected papers.* New Jersey: Lawrence Erlbaum.
Wickens, T. D. (2002). *Elementary signal detection theory.* New York: Oxford University Press.

Signs

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Definition

Signs are objective physical manifestations of an injury or disease perceptible to the examiner, as opposed to symptoms which are the subjective evidence of disease experienced by the patient. Signs can be felt, heard, seen, or measured by the diagnostician.

Current Knowledge

Recognizing signs of disease is an important part of diagnosing disease. Signs are any abnormality indicative of a disease discoverable on an examination of the patient. They include pulse, respiration, blood pressure, and physical evidence such as bleeding, broken skin, bruising, etc. Along with symptoms, signs are diagnostic "tools" which help the assessor determine the condition of the patient. There are a number of signs that have been discovered throughout the fields of medicine that are described as being very specific for various illnesses. Examples of these include Kernig's and Brudzinski's signs in meningitis, Chvostek's sign in facial irritability and tetany, Tinel's sign in nerve injury and regeneration, Romberg's sign in assessing proprioceptive control, and Homan's sign in venous thrombosis of the leg.

References and Readings

Stedman, T. L. (1995). *Stedman's medical dictionary* (26th ed.). Baltimore: Williams & Wilkins.

SILS

- ▶ Shipley Institute of Living Scale

Simultanagnosia

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Short Description

Persons with simultanagnosia are unable to perceive multiple elements of an object or scene simultaneously. Simultagnosia typically occurs in the absence of visual field deficits. Although the ability to perceive and name individual objects regardless of their location within the visual field remains intact, patients with simultagnosia exhibit an inability to perceive and interpret the overall gestalt of the scene.

Simultagnosia usually results from bilateral lesions to the parietal-occipital regions, though some cases have been reported following damage to the superior occipital

or inferior parietal lobes. Individuals with neurodegenerative diseases may also present with simultagnosia. Neuroimaging studies have associated simultagnosia with dorsal occipital lobe lesions in Brodmann's areas 18 and 19. Simultagnosia may occur in isolation, or it may occur as part of Balint's syndrome – a more complex disorder that is characterized by a triad of visual-spatial dysfunction: simultagnosia, optic ataxia, and ocular-motor apraxia.

Categorization

It has been proposed that there may be two different types of simultagnosia: dorsal simultagnosia and ventral simultagnosia. Dorsal simultagnosia, as suggested by Farah (1990), arises from bilateral lesions of the occipital and/or parietal lobes, whereas ventral simultagnosia arises from lesions of the left temporal-occipital region. Both types of simultanagnosic patients are unable to combine elements of a scene into a whole, despite being able to recognize single objects within the scene. The two types of simultagnosia differ in that, for patients with dorsal simultagnosia unattended objects are not perceived at all, resulting in significant navigation difficulties (e.g., bumping into objects), whereas patients with ventral simultagnosia demonstrate less difficulty when walking in unfamiliar spaces. This suggests that patients with ventral simultagnosia may process some visual information without being able to register this information at a more conscious level.

Some argue that it is premature to classify different varieties of simultagnosia due to the lack of reported cases and systematic assessment of patients. It is possible that what is currently characterized as ventral simultagnosia may represent a less severe form of dorsal simultagnosia. Although the anatomic pathology of the two types of simultagnosia appears distinct and it remains plausible that dorsal and ventral simultagnosia may represent two separate syndromes, more research is needed to support this differentiation.

Natural History, Prognostic Factors, Outcomes

The prognosis for patients with simultagnosia varies depending on the etiology. Patients with degenerative diseases usually experience a declining course, while some patients with acute infarction may demonstrate improved functioning over time.

Neuropsychology and Psychology of Simultagnosia

Patients with simultagnosia commonly ignore or neglect all other objects once one object in the visual field has been fixated upon. In a classic example, Hécaen and Ajuriaguerra (1954) described a patient with simultagnosia who, when focusing on the tip of his cigarette, was unable to perceive the match flame being offered to him which was held only a few inches away from his cigarette. Simultanagnosic patients demonstrate difficulties with counting and when required to identify or select items within an array. When presented with a letter identification task, patients are often able to identify single letters, but they may demonstrate difficulty identifying multiple letters in a random string. Reading can also be impaired; however, patients may be able to read some words (which may suggest that the letters have been grouped into a single linguistic element). Patients with simultagnosia experience difficulty with visual search and cannot conduct visual surveys across large areas of space. Interpreting complex scenes is also impaired.

In general, the literature suggests that simultagnosia does not result from visual field deficits or ocular-motor impairments, as these abnormalities do not account for the deficits displayed in all cases of simultagnosia. Alternatively, it has been suggested that other visual impairments displayed by patients, including impairments in speed of information processing and basic visual processes (e.g., Humphreys & Price, 1994) as well as impairments in attention (e.g., Humphreys & Riddoch, 1992), may be the underlying causes of the disorder. As with many disorders that occur infrequently, more research is needed to further characterize the fundamental deficits that result in simultagnosia.

Evaluation

In assessing patients presenting with symptoms of simultagnosia, it is important that their visual fields be examined fully as some types of visual field abnormalities (e.g., extensive peripheral scotomata) can mimic simultagnosia.

During a neuropsychological assessment, one may ask 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). 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, which will aid

in distinguishing symptoms more commonly associated with hemineglect from symptoms of simultagnosia. Individuals with hemineglect may describe items on one side of the picture only. In contrast, patients with simultagnosia are often able to identify discrete items in the picture across all quadrants, yet they are frequently unable to integrate the various elements of the picture into a coherent story. Patients with simultagnosia will demonstrate significant impairments on counting and visual search tasks. Letter identification and reading abilities may also be assessed for functional purposes. Ishihara color plates may also be administered, as it has been reported that some patients fail to integrate the multiple dots into an image (i.e., a number), despite being able to perceive the individual dots and their colors (Brazis, Graff-Radford, Newman, et al., 1998). When testing materials are limited, such as during a bedside examination, patients may be asked to count and/or pick up a number of items (e.g., coins, pens, or other small objects) placed randomly on a table.

Cross References

- ▶ Balint's Syndrome
- ▶ Neglect
- ▶ Visual Field Deficit

References and Readings

Brazis, P. W., Graff-Radford, N. R., Newman, N. J., & Lee, A. G. (1998). Ishihara color plates as a test for simultanagnosia. *American Journal of Ophthalmology, 126*, 850–851.

Farah, M. J. (1990). *Visual agnosia.* Cambridge, England: Cambridge University Press.

Hécaen, H., & Ajuriaguerra, J. (1954). Balint's syndrome (psychic paralysis of visual fixation) and its minor forms. *Brain, 77*, 373–400.

Humphreys, G. W., & Price, C. J. (1994). Visual feature discrimination in simultanagnosia: A study of two cases. *Cognitive Neuropsychology, 11*, 393–434.

Humphreys, G. W., & Riddoch, M. J. (1992). Interactions between object and pace systems revealed through neuropsychology. In D. E. Meyer & S. Kornblum (Eds.), *Attention and performance XIV: Synergies in experimental psychology, artificial intelligence and cognitive science* (pp. 143–162). Cambridge, MA: MIT Press.

Simultanapraxia

- ▶ Motor Impersistence

Simultaneous Processing

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Synonyms

Holistic; Gestalt

Definition

Simultaneous processing is the process of combining discrete and unconnected stimuli into a single group or whole to assist in comprehension and interpretation. It involves the comprehension of the relationships of and between separate entities and its relation or position to the whole. The integration of distinct yet interrelated stimuli can also facilitate the ability to uncover underlying patterns in verbal and nonverbal information.

Simultaneous processing can be helpful to accomplish tasks where the focus is on solving problems where the objective of the task demands conceptualization of parts into a cohesive whole. Spatial characteristics are often associated with simultaneous processing for this reason. Additionally, simultaneous processing has been applied to utilization and comprehension of logical and grammatical statements. For example, in order for an individual to fully understand meaning of an idea presented, he or she needs to demonstrate knowledge of word relationships, pragmatics, and tone.

Simultaneous processing is often measured by completing patterns or organization of information in order to produce a correct answer. Tasks evaluating simultaneous processing skills may incorporate visual or auditory stimuli or both. Examples include subtests that require pattern completion (Matrix Reasoning from the Wechsler Intelligence Scale for Children, Fourth Edition), understanding logical and grammatical descriptions (Verbal-Spatial Relations from the Cognitive Assessment System), understanding parts within a whole (Triangles from the Kaufman Assessment Battery for Children, Second Edition). It is also important to note that the vast majority of tools used to measure simultaneous processing also incorporate a strong visual-spatial component.

In Luria's theory, simultaneous processing is one of two complementary information processing strategies comprising the second functional unit (occipital, parietal,

and temporal lobes of the brain; Das, Naglieri, & Kirby, 1994). Simultaneous processing is predominantly accommodated by the right hemisphere and specifically, in the occipital, parietal, and temporal regions (Reynolds & French, 2005).

Cross References

- ▶ Cognitive Assessment System
- ▶ Sequential Processing
- ▶ Successive Processing

References and Readings

Horton, A. M., Jr., & Wedding, D. (Eds.). (2006). *The neuropsychology handbook*. New York: Springer Publishing Company.

Reynolds, C. R., & French, C. L. (2005). The brain as a dynamic organ of information processing and learning. In R. C. D'Amato, E. Fletcher-Janzen, & C. R. Reynolds (Eds.), *Handbook of school neuropsychology* (pp. 86–119). Hoboken, NJ: Wiley.

Single Photon Emission Computed Tomography

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Definition

Tomography is the use of X-ray to "slice" an organ or a mass. The invention consists of reciprocal linear or curved motion of the X-ray tube and film cassette. The target level is enhanced and the other levels blurred. CT technology employs a tightly collimated 1–10-mm beam to circle the patient. Single Photon Emission Computed Tomography (SPECT) adds to this technology by applying an externally derived radioisotope. Consequently, SPECT is used to evaluate blood flow. For example, regional, three-phase SPECT bone scan can be directed at upper limbs in order to document and confirm the presence of reflex sympathetic dystrophy (RSD). In fact it adds an objective test to an otherwise subjective phenomenon. During seizure activity, it will delineate ictal foci. A special type of SPECT modification, to examine a dopamine transporter has been used to evaluate patients who were thought to have early parkinsonism.

Current Knowledge

An objective method applied to the documentation of RSD (also complex regional pain disorder or CRAPS), ictal foci during seizure disorders, parkinsonism.

Cross References

- ▶ Head Injury
- ▶ Parkinsonian

References and Readings

American Academy of Neurology. (1991). SPECT and neurosonology qualifications approved. *Neurology, 41*, 13A.

Kaufmann, H., & Schatz, I. J. (2004). Pure autonomic failure. In D. Roberston (Ed.), *Primer on the autonomic nervous system*, (2nd ed., pp. 309–311). Amsterdam: Elsevier.

Sinus Thrombosis

- ▶ Central Venous Thrombosis

SIP

- ▶ Sickness Impact Profile

SIP-68

- ▶ Sickness Impact Profile

SIS

- ▶ Stroke Impact Scale

SIS 2.0

► Stroke Impact Scale

SIS 3.0

► Stroke Impact Scale

SIS-16

► Stroke Impact Scale

Six-Minute Walk Test

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Synonyms

6MWD; 6MWT

Description

The six-minute walk test (6MWT) measures the distance (6MWD) that a person can quickly walk on a flat, hard surface in 6 min. The test is submaximal and self-paced, with rest breaks allowed as needed.

Historical Background

Historically, aerobic functional capacity has been evaluated by asking a person "how many flights of stairs can you climb?" or "how many blocks can you walk?" Due to poor reliability and validity of such questioning, more objective methods have been established. In 1963, Balke created a simple test of the distance a person could walk in a specified amount of time. In 1968, Cooper described a 12 min overground walking test for healthy individuals, which was adapted for patients with chronic bronchitis in 1976. The 6MWT was created to accommodate persons with respiratory disease for whom a 12 min span was excessive. The test has been adopted for use for persons with a wide range of different cardiopulmonary diseases as well as orthopedic and neuromuscular diagnostic groups.

Psychometric Data

Concurrent validity of the 6MWT has been established by significant correlations with peak oxygen uptake ($r = 0.56$ to $r = 0.88$) and by better correlation with quality of life measures than peak oxygen uptake. Furthermore, intervention-related changes in 6MWD correlate with subjective improvement in dyspnea.

The 6MWT also has good test–retest reliability, with an ICC of 0.75–0.97 and a coefficient of variation of approximately 8%, which is better than that of functional status questionnaires (22–33%).

Clinical Uses

The 6MWT evaluates global aerobic exercise capacity, which is dependent on the responses of the cardiovascular and pulmonary systems, blood, neuromuscular units, and muscular metabolism. Unlike maximal cardiopulmonary exercise testing, the 6MWT cannot determine peak oxygen uptake, diagnose the cause of exertional dyspnea, or determine the mechanism of exercise limitation. It is instead designed to evaluate aerobic functional capacity for activities of daily living, which are performed at submaximal levels of exertion. The 6MWT has been used to assess baseline functional limitations, disease progression, and as an outcome measurement for a wide range of cardiopulmonary diseases. It has also been used for persons with various orthopedic and neuromuscular diagnoses, including hip fracture, stroke, and spinal cord injury.

Absolute contraindications for the test include recent unstable angina or myocardial infarction (during the previous month). Relative contraindications include resting heart rate >120 bpm, systolic blood pressure >180 mmHg, diastolic blood pressure >100 mmHg or stable exertional angina (patients should perform the test after

using their antiangina medication, and rescue nitrate medication should be readily available).

Enright and Sherill (1998) collected normative data (cross reference) for the 6MWT on healthy adults aged 40–80 years and found a mean 6MWD of 576 m for men and 494 m for women. The following normative equations were also derived:

$$\begin{aligned}\text{6MWT distance} &= (7.57 \times \text{height cm}) - (5.02 \times \text{age}) \\ &\quad - (1.76 \times \text{weight kg}) \\ &\quad - 309 \text{ m for men} \\ \text{6MWT distance} &= (2.11 \times \text{height cm}) \\ &\quad - (2.29 \times \text{weight kg}) - (5.78 \times \text{age}) \\ &\quad + 667 \text{ m for women}\end{aligned}$$

For an individual patient with COPD, a 6MWD improvement of 71–86 m can be interpreted as meaningful change with 95% confidence.

Cross References

- ▶ Contraindication
- ▶ Functional Capacity Evaluation
- ▶ Myocardial Infarction
- ▶ Spinal Cord Injury
- ▶ Stroke

References and Readings

ATS Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories (2002). ATS statement: Guidelines for the six-minute walk test. *American Journal of Respiratory and Critical Care Medicine, 166*(1), 111–117.

Balke, B. (1963). A simple field test for the assessment of physical fitness. rep 63–6. *[Report]. Civil Aeromedical Research Institute (U.S.)*, 1–8.

Butland, R. J., Pang, J., Gross, E. R., Woodcock, A. A., & Geddes, D. M. (1982). Two-, six-, and 12-minute walking tests in respiratory disease. *British Medical Journal (Clinical Research Ed.), 284*(6329), 1607–1608.

Cooper, K. H. (1968). A means of assessing maximal oxygen intake. Correlation between field and treadmill testing. *JAMA: The Journal of the American Medical Association, 203*(3), 201–204.

Du, H., Newton, P. J., Salamonson, Y., Carrieri-Kohlman, V. L., & Davidson, P. M. (2009). A review of the six-minute walk test: Its implication as a self-administered assessment tool. *European Journal of Cardiovascular Nursing: Journal of the Working Group on Cardiovascular Nursing of the European Society of Cardiology, 8*(1), 2–8.

Enright, P. L., & Sherrill, D. L. (1998). Reference equations for the six-minute walk in healthy adults. *American Journal of Respiratory and Critical Care Medicine, 158*(5 Pt 1), 1384–1387.

Kim, C. M., Eng, J. J., & Whittaker, M. W. (2004). Effects of a simple functional electric system and/or a hinged ankle-foot orthosis on walking in persons with incomplete spinal cord injury. *Archives of Physical Medicine and Rehabilitation, 85*(10), 1718–1723.

Latham, N. K., Mehta, V., Nguyen, A. M., Jette, A. M., Olarsch, S., Papanicolaou, D., et al. (2008). Performance-based or self-report measures of physical function: Which should be used in clinical trials of hip fracture patients? *Archives of Physical Medicine and Rehabilitation, 89*(11), 2146–2155.

McGavin, C. R., Gupta, S. P., & McHardy, G. J. (1976). Twelve-minute walking test for assessing disability in chronic bronchitis. *British Medical Journal, 1*(6013), 822–823.

Savci, S., Inal-Ince, D., Arikan, H., Guclu-Gunduz, A., Cetisli-Korkmaz, N., Armutlu, K., et al. (2005). Six-minute walk distance as a measure of functional exercise capacity in multiple sclerosis. *Disability and Rehabilitation, 27*(22), 1365–1371.

States, R. A., Pappas, E., & Salem, Y. (2009). Overground physical therapy gait training for chronic stroke patients with mobility deficits. *Cochrane Database of Systematic Reviews (Online), 3*(3), CD006075.

Wise, R. A., & Brown, C. D. (2005). Minimal clinically important differences in the six-minute walk test and the incremental shuttle walking test. *COPD, 2*(1), 125–129.

Sjogren's Syndrome

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Short Description

Sjogren's Syndrome (SS) is a chronic lymphoproliferative, autoimmune disease that has been found primarily among post-menopausal women. SS inflames tear ducts and salivary glands (Meijer, Pijpe, Bootsma, Vissink, & Kallenberg, 2007), and causes disturbances of Y lymphocytes, B lymphocytes, and exocrine glands, essentially causing one's white blood cells to "attack" the individual's body (Hansen, Lipsky, & Dorner, 2007).

Categorization

There are two categories of SS. The primary type is a chronic autoimmune disorder marked with dryness of the mouth, eyes, and other mucous membranes (Goransson et al., 2006). Secondary SS occurs in individuals diagnosed with other connective tissue disorders such as rheumatoid arthritis, progressive systemic sclerosis, and systemic lupus erythematosus.

Epidemiology

SS affects all populations worldwide. The disease is most prevalent in people in their forties and fifties, and has a 9:1 female to male ratio (Meijer et al., 2007). The prevalence of the disease is reported to be 0.2% of the general population, however, diagnosis is complex. Lack of consistency in classification has meant that SS has been falsely diagnosed as other connective tissue disorders and vice versa.

Natural History, Prognostic Factors, Outcomes

SS was named after the Swedish ophthalmologist Henrik Sjogren, who first described the clinical and pathological findings of xerostomia (dry mouth) and keratoconjunctivitis sicca (dry eye) (Morgen, McFarland, & Pillemer, 2004). Although, Sjogren's syndrome can rarely shorten a person's life, dryness in the eyes generally leads to keratoconjunctivitis and corneal ulcers (Goransson et al., 2006). Additionally, oral dryness can lead to carries, fissures, and difficulty in swallowing, which reduce the quality of life. Musculoskeletal fatigue, dry skin, and vaginal dryness has also been reported in SS patient population. Due to infiltration of white blood cells, greater risk of lymphoma has also been reported to occur in the SS patient population.

Neuropsychology of Sjogren's Syndrome

SS causes disturbances of T lymphocytes, B lymphocytes, and exocrine glandular cells (Hansen et al., 2007), leading white blood cells to infiltrate moisture producing glands, which leaves exocrine glands dry. Researchers have suggested that SS is a disorder that displays a strong interaction of neural and endocrine systems, as the tearing reflex involves a neural loop where afferent nerve signals from the eyes are relayed to the medulla, and then back to the ocular surface through efferent nerves which then stimulate the secretion glands, which produce water for tears (Alexander, 1993). SS is associated with the release of white blood cells on this tearing mechanism, which release antibodies that cause damage to exocrine glandular cells, which, in turn overexcite muscarine MR 3 receptors that leave the tear and saliva producing glands deficient. In turn, mononuclear lymphoid infiltration replaces the glandular epithelium causing dryness of the exocrine glands. Recent research has suggested that that SS is also associated with increased frequency of central nervous system white matter lesions, but more neuroimaging research is needed (Morgen et al., 2004). Researchers have demonstrated evidence that SS patients exhibit central and peripheral sensorimotor neuropathy, affecting the sensory neurons in the basal ganglia (e.g., associated with movement coordination), and often leading to ganglionitis (e.g., inflammation of ganglion neurons) (Goransson et al., 2006). In addition, deficits such as hemisensory deficits, hemiparesis, seizure disorders, cognitive dysfunction, and psychiatric abnormalities have been found in SS patients, which may be associated with decreased cortical thickness and basal ganglia dysfunction (Alexander).

Evaluation

There are no laboratory assessments specific to SS; however, a number of diagnostic tests can be used to properly diagnose the disease. Salivary biopsies are most common diagnostic techniques, as those who exhibit lymphocyte infiltration in their saliva are more than likely diagnosed with SS (Morgen et al., 2004). People experiencing SS should have hemoglobin measured; as low hemoglobin count is a sign of SS. If rheumatoid factor is positive, then there is a higher likelihood that the person may have SS, as 80–90% of Sjogren's patients exhibit a positive hemoglobin factor (Alexander, 1993). In addition, those with hypothyroidism antibodies may also have SS.

Treatment

The cause of SS is unknown, although growing evidence suggests that there is a genetic link to the disease (Hansen et al., 2007). Although there is no cure for SS, symptom treatment is available, wherein SS patients are prescribed eye drops to treat dryness, as well as immune suppressants to decrease the aggressive nature of cytokine infiltration of the exocrine glands.

Cross References

▶ Auto-Immune Disorders

References and Readings

Alexander, E. (1993). Sjogren's syndrome and MS. *Neurology, 43*(2), 455–456.

Goransson, L. G., Herigstad, A., Tjensvoll, A. B., Harboe, E., Mellgren, S. I., & Omdal, R. (2006). Peripheral neuropathy in primary Sjogren syndrome: a population-based study. *Archives of Neurology, 63*(11), 1612–1615.

Hansen, A., Lipsky, P. E., & Dorner, T. (2007). B cells in lymphoproliferation in chronic inflammatory rheumatic disorders. *Nature Clinical Practice. Rheumatology, 3*(10), 561–569.

Meijer, J. M., Pijpe, J., Bootsma, H., Vissinki, A., & Kallenberg, C. G. (2007). The future of biologic agents in the treatment of Sjogren's syndrome. *Clinical Reviews in Allergy and Immunology, 32*(3), 292–297.

Morgen, K., McFarland, H. F., & Pillemer, S. R. (2004). Central nervous system disease in primary Sjogrens syndrome: the role of magnetic resonance imaging. *Seminars in Arthritis and Rheumatis, 34*(3), 623–630.

Cross References

► Depressed Skull Fracture

References and Readings

Graham, D. I., Saatman, K. E., Marklund, N., Conte, V., Morales, D., Royo, N., & McIntosh, T. K. (2006). The neuropathology of trauma. In R. W. Evans (Ed.), *Neurology and Trauma* (2nd ed., pp. 45–94). New York: Oxford University Press.

Victor, M., & Ropper, A. H. (2001). *Principles of neurology* (7th ed., pp. 925–53). New York: McGraw-Hill.

Skull Fracture

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Definition

Skull fracture is the fracture of the bone surrounding the brain. Skull fractures occur most commonly in motor vehicle accidents, in physical assault, following falls from extreme heights, and less commonly, in severe sports-related injuries. The presence of skull fracture denotes a more severe degree of head injury. Meninges and blood vessels surrounding the brain can be damaged leading to a collection of blood in the sinuses or on the brain surface. This may lead to initial brain edema (swelling). Cerebrospinal fluid, which flows over the surface of the brain, may also be displaced and leak out through the nose or ear. Types of skull fractures include: simple, linear, compound, and depressed fractures. Simple skull fracture is characterized by a break in the skull bone without displacement of the fractured fragment. Linear skull fracture is a simple break in the skull bone that follows a straight line; the fracture causes the bone surrounding the area of impact to bend outward, whereas the fractured bone segment bends inward. A compound skull fracture, also known as open skull fracture, involves fracture of the bone that may also lacerate the scalp and the underlying meninges of the brain and/or mucous membranes of the face. Depressed skull fracture describes the situation in which bone fragments impact brain tissue directly, causing further damage and susceptibility to bacterial infection.

Sleep Apnea Syndrome

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Definition

Central Sleep Apnea Syndrome is characterized by a cessation or decrease of ventilatory effort during sleep usually associated with oxygen desaturation. Diagnostic criteria by the ICSD-R include in part:

1. Frequent episodes of shallow or absent breathing during sleep
2. Associated features include at least one of the following: gasps, grunts, or choking during sleep; frequent body movements; cyanosis during sleep
3. Polysomnographic monitoring demonstrates:
   (a) Apneic pauses during sleep greater than 10 seconds (20 s in infancy).
   (b) The apnea is caused by central nervous system malfunction rather than obstruction.
   (c) One or more of the following: frequent arousals from sleep associated with the apneas, bradytachycardia, and oxygen desaturation in association with the apneic episodes.

Central sleep apnea is rare; therefore, the neuropsychological effects of central sleep apnea alone (without coexisting obstructive sleep apnea) have not been studied in detail. In obstructive sleep apnea, which also results in oxygen desaturation and disrupted sleep, the largest

neuropsychological effects appear to be in attention and vigilance.

Cross References

► Obstructive Sleep Apnea Syndrome

References and Readings

American Academy of Sleep Medicine. (2001). *International classification of sleep disorders, revised: Diagnostic and coding manual.* Chicago: American Academy of Sleep Medicine.

Sleep Disorder

► Sleep Disturbance

Sleep Disturbance

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Synonyms

Dyssomnias; Parasomnias; Sleep disorder

Definition

Sleep disturbance is a general symptom of which there are many types and many causes.

Current Knowledge

The American Academy of Sleep Medicine in association with the European Sleep Research Society, the Japanese Society of Sleep Research, and the Latin American Sleep Society have published the International Classification of Sleep Disorders, Revised (ICSD-R). Sleep disorders are also described in the North American version of the World Health Organization's International Classifications of Diseases-9 (ICD-9) as well as the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV). The ICSD-R provides perhaps the most extensive listing.

The causes of sleep disturbance are quite varied depending on the disorder. Sleep disturbance can be a brief event triggered by environmental stimuli such as noise or light. Sleep disturbance can be related to physical factors such as diet or exercise. Sleep can be disturbed by psychological factors such as depression or stress. Sleep can also be disturbed by neurological or other medical disorders, and sleep can be affected by medical trauma such as traumatic brain injury.

Broadly, the organization of sleep disturbance can be divided in three major types: dyssomnias, parasomnias, and sleep disorders associated with mental, neurologic, or other medical disorders. Dyssomnias are disorders that produce either difficulty initiating sleep, difficulty maintaining sleep, or excessive sleepiness. They are broadly characterized by disturbances in the amount, quantity, or time of sleep. Parasomnias are disorders that intrude into the sleep process and are not primarily disorders of the sleep and wake states per se. They are characterized by abnormal behavioral or physiological events occurring in association with sleep, specific sleep stages, or sleep-wake transitions. They represent the activation of the autonomic nervous system, motor system, or cognitive processes during sleep.

According to the ICSD-R, the dyssomnias can be further subdivided into intrinsic sleep disorders (originate within the body or arise from causes within the body), extrinsic sleep disorders (external factors are integral in these disorders), and circadian rhythm sleep disorders (related to timing of sleep within the 24 h day). It is acknowledged that some intrinsic disorders may have some external factors that precipitate or exacerbate the disorder. Also, some extrinsic disorders may depend on factors within the body for expression of the disorder. However, the ICSD-R asserts that in intrinsic disorders, the primary cause of the disorder is an abnormality in the physiology or pathology within the body and though removal of external factors may improve the disorder, the disorder will continue to occur due to the intrinsic cause. Similarly, the ICSD-R asserts that in extrinsic disorders, the external factors are essential for the disorder to occur and continue. Table 1 lists the specific disorders listed under each subtype of dyssomnia.

According to the ICSD-R, the parasomnias can be further subdivided into arousal disorders (manifestations

of partial arousal that occur during sleep), sleep-wake transition disorders (occur mainly during the transition from wakefulness to sleep or from one sleep stage to another), parasomnias usually associated with REM sleep, and other parasomnias. Table 2 lists the specific disorders listed under each subtype of parasomnia.

Sleep Disturbance. Table 1 Dyssomnias

Intrinsic sleep disorders
1. Psychophysiological insomnia
2. Sleep state misperception
3. Idiopathic insomnia
4. Narcolepsy
5. Recurrent hypersomnia
6. Idiopathic hypersomnia
7. Posttraumatic hypersomnia
8. Obstructive sleep apnea syndrome
9. Central sleep apnea syndrome
10. Central alveolar hypoventilation syndrome
11. Periodic limb movement disorder
12. Restless legs syndrome
13. Intrinsic sleep disorder NOS
Extrinsic sleep disorders
1. Inadequate sleep hygiene
2. Environmental sleep disorder
3. Altitude insomnia
4. Adjustment sleep disorder
5. Insufficient sleep syndrome
6. Limit-setting sleep disorder
7. Sleep-onset association disorder
8. Food allergy insomnia
9. Nocturnal eating (drinking) syndrome
10. Hypnotic-dependent sleep disorder
11. Stimulant-dependent sleep disorder
12. Alcohol-dependent sleep disorder
13. Toxin-induced sleep disorder
14. Extrinsic sleep disorder NOS
Circadian rhythm disorders
1. Time zone change (jet lag) syndrome
2. Shift work sleep disorder
3. Irregular sleep-wake pattern
4. Delayed sleep phase syndrome
5. Advanced sleep phase syndrome
6. Non-24-h sleep-wake disorder
7. Circadian rhythm sleep disorder NOS

The final broad classification in the ICSD-R is sleep disorders associated with mental, neurologic, or other medical disorders. This section lists those disorders that are not primarily sleep disorders but are mental, neurologic or other medical disorders that have a sleep disturbance or excessive sleepiness as a major feature. This section of the ICSD-R is not meant to be exhaustive, but rather a listing of the most common disorders associated with sleep symptoms. Table 3 lists those disorders specifically described in the ICSD-R.

There are many disorders in Tables 1–3 that are familiar to neuropsychologists. For example, neuropsychologists working frequently with traumatic brain injury may also be familiar with the intrinsic dyssomnia of posttraumatic hypersomnia. It is classified as intrinsic despite the fact that it stems from an external event (brain injury)

Sleep Disturbance. Table 2 Parasomnias

Arousal disorders
1. Confusional arousals
2. Sleepwalking
3. Sleep terrors
Sleep-wake transition disorders
1. Rhythmic movement disorder
2. Sleep starts
3. Sleep talking
4. Nocturnal leg cramps
Parasomnias usually associated with REM sleep
1. Nightmares
2. Sleep paralysis
3. Impaired sleep-related penile erections
4. Sleep-related painful erections
5. REM sleep-related sinus arrest
6. REM sleep behavior disorder
Other parasomnias
1. Sleep bruxism
2. Sleep enuresis
3. Sleep-related abnormal swallowing syndrome
4. Nocturnal paroxysmal dystonia
5. Sudden unexplained nocturnal death syndrome
6. Primary snoring
7. Infant sleep apnea
8. Congenital central hypoventilation syndrome
9. Sudden infant death syndrome
10. Benign neonatal sleep myoclonus
11. Other parasomnia NOS

Sleep Disturbance. Table 3 Sleep disorders associated with mental, neurological or other medical disorders

Mental disorders
1. Psychoses
2. Mood disorders
3. Anxiety disorders
4. Panic disorders
5. Alcoholism
Neurologic disorders
1. Cerebral degenerative disorders
2. Dementia
3. Parkinsonism
4. Fatal familial insomnia
5. Sleep-related epilepsy
6. Electrical status epilepticus of sleep
7. Sleep-related headaches
Other medical disorders
1. Sleeping sickness
2. Nocturnal cardiac ischemia
3. Chronic obstructive pulmonary disease
4. Sleep-related asthma
5. Sleep-related gastroesophageal reflux
6. Peptic ulcer disease
7. Fibromyalgia

because the primary cause is thought to be of central nervous system origin and the disorder persists after the traumatic event has ended. According to the ICSD-R, posttraumatic hypersomnia is a disorder of excessive sleepiness that occurs as a result of a traumatic event involving the central nervous system. Table 4 for specific diagnostic criteria. The sleepiness is an alteration from the patient's pretrauma sleep patterns. The disorder is characterized by frequent daytime sleepiness, which may or may not be able to be resisted, with resulting sleep episodes. This sleepiness is typically seen in the context of other posttraumatic encephalopathic features, such as headaches, fatigue, difficulty sleeping, and memory impairment. The course of the disorder is such that the sleepiness is typically most evident in the immediate posttraumatic period and resolves over weeks to months. However, some residual sleepiness or sleep complaint may persist or very worsen. The likelihood of disabling sleepiness increases as the severity of initial head trauma increases.

Pediatric specialists may be more familiar with those sleep disorders that are more common in childhood or adolescence, such as limit-setting sleep disorder, sleep-onset association disorder, delayed sleep phase disorder, sleep walking, sleep terrors, rhythmic movement disorder, nightmares, or sleep enuresis. Geriatric specialists may be more familiar with those sleep disorders that are more common in the elderly or disorders with associations to neurodegenerative illnesses such as REM sleep behavior disorder, advanced sleep phase disorder, or sleep disruption associated with dementia.

Sleep Disturbance. Table 4 Diagnostic criteria for posttraumatic hypersomnia

A. The patient has a complaint of excessive sleepiness
B. Frequent daily sleep episodes occur
C. The onset of the sleepiness is temporally associated with head trauma
D. Polysomnography demonstrates all of the following:
1. Normal timing, quality, and duration of sleep
2. A mean sleep latency of less than 10 min on Mean Sleep Latency Test (MSLT)
3. Fewer than two sleep-onset REM periods on MSLT
E. No medical disorder is present that could account for the symptom
F. The symptoms do not meet the criteria of other sleep disorders that produce sleepiness (e.g., narcolepsy)
Minimal criteria: A plus B plus C

Treatment

Given the wide variety of sleep disturbances and the causes of sleep disturbances, it is not surprising that the treatments vary depending on the disorder. Treatments can involve behavioral changes (e.g., sleep hygiene, cognitive behavior therapy), mechanical devices (CPAP, bedwetting alarm), or pharmacological treatment (hypnotics, benzodiazepines), or some combination of the above.

For example, treatments for primary insomnia or psychophysiologic insomnia include nonpharmacologic therapies such as sleep hygiene recommendations, relaxation therapy, biofeedback training, time in bed restriction and stimulus control, and cognitive behavioral therapy. Pharmacological management options for insomnia are quite diverse ranging from benzodiazepines [e.g., Restoril (temazepam), Xanax (alprazolam), Avitan (lorazepam), Valium (diazepam), Klonopin (clonazepam)], nonbenzodiazepine hypnotics [e.g., Ambien (zolpidem), Sonata (zaleplon), Estorra (eszopiclone), Lunesta (eszopiclone)], and antidepressants [e.g., Elavil (amitriptlyline),

trazodone], and over the counter medications containing antihistamine (e.g., Benadryl).

In the context of neuropsychological evaluation with adults, most sleep disturbances cause excessive daytime sleepiness. Generally, sleep disturbances result in decreased alertness and neuropsychological deficits in attention, concentration, working memory, and executive functions. In children, sleep disturbances also cause daytime sleepiness, but they may also display some agitation/hyperactivity or irritability as well.

Cross References

- ▶ Central Sleep Apnea
- ▶ Hypersomnia
- ▶ Insomnia
- ▶ Narcolepsy
- ▶ Obstructive Sleep Apnea

References and Readings

American Academy of Sleep Medicine. (2001). *International classification of sleep disorders, revised diagnostic and coding manual (ICSD-R).* Retrieved April 4, 2008, from http://www.absm.org/PDF/ICSD.pdf

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

Owens, J., Spirito, A., McGuinn, M., & Nobile, C. (2000). Sleep habits and sleep disturbance in elementary school-aged children. *Journal of Developmental & Behavioral Pediatrics, 21*, 27–36.

Perlis, M. L., Jungquist, C., Smith, M. T., & Posner, D. (2005). *Cognitive behavioral treatment of insomnia.* New York: Springer.

SLE

▶ Systemic Lupus Eythematosus

Slow Virus Infection

▶ Kuru

Slowly Progressive Aphasia

▶ Primary Progressive Aphasia

Slowness of Movements

▶ Parkinsonism

SMAF (from the Original Title "Système De Mesure De L'Autonomie Fonctionnelle")

▶ Functional Autonomy Measurement System

Small Vessel Ischemic Disease

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Synonyms

Ischemic leukoaraiosis; Lacunar infarction; Periventricular white matter change; Selective incomplete white matter infarction; Subcortical vascular leukoencephalopathy; White matter disease.

Definition

Cerebral small vessel ischemic disease (SVID) results from compromised functioning of the small penetrating cerebral arteries, causing reduced blood flow to deep white matter regions of the brain, leading to focal lacunar infarcts, diffuse ischemia (leukoaraiosis), vascular cognitive impairment, and dementia.

Historical Background

Because the fatality of SVID is so low and postmortem evidence of SVID is rarely acute, our current body of knowledge and understanding of SVID is still in its infancy. Although descriptions of SVID can be found in the literature as far back as the late nineteenth century, it was not until the late twentieth century that we began to examine the pathophysiology and clinical consequences of cerebral SVID. This interest in SVID coincided with the

recognition of the distinction between arteriosclerotic dementia (i.e., dementia resulting from hardening of the arteries) and degenerative dementia due to Alzheimer's disease or other "nonvascular" causes. C. Miller Fisher, an influential figure in the study of SVID, provided some of the first descriptions of the clinical implications of SVID in a series of papers spanning three decades of research from the 1960s through the 1990s. When Hachiniski, Lassen, & Marshall (1974) clinically defined "multi-infarct dementia" as a subtype of dementia resulting from multiple small vessel infarcts, the clinical significance of SVID was further reinforced. SVID is now recognized as a serious consequence of cerebrovascular disease, a significant contributor to cognitive impairment, and an important risk factor for stroke.

Current Knowledge

SVID has become increasingly recognized as a significant contributor to age-related decline in functioning and an underlying cause of vascular cognitive impairment and dementia. Although SVID is revealed postmortem among two-thirds of elderly individuals without vascular dementia, the presence of SVID is believed to be indicative of pathology rather than the normal, healthy aging process.

The precise cause of SVID is unknown. However, there is a genetic form of SVID, known as cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), which is caused by a mutation in the *Notch3* gene on chromosome 19. CADASIL typically has an earlier onset, between 30 and 50 years of age, and is characterized by migraines, recurrent transient ischemic attacks, and strokes, leading to cognitive decline and dementia.

SVID pathology is distinct from that of Alzheimer's disease, although the two are now known to frequently coexist. It is suspected that a complex interaction occurs when both are present, causing them to act synergistically. The presence of both SVID and Alzheimer's disease appears to be related to higher rates of dementia.

Clinical presentation: The clinical manifestation of SVID typically involves a slow, progressive decline in cognition, with most prominent deficits in the areas of processing speed and executive functions, although as the disease progresses other areas of cognition also begin to decline. Motor impairments such as balance difficulties, gait abnormalities, and lower-limb weaknesses are also seen. Many individuals also present with dysarthria, dysphagia, and urinary continence. Depression and generalized apathy are commonly reported in those with SVID. The amount and severity of white matter lesions positively correlate with the progressive decline in cognitive and physical functioning. SVID has been reported to be the most common cause of vascular dementia.

Imaging: Although both CT and MRI can be used to diagnose SVID, MRI has been shown to be most sensitive at detecting the associated pathology. MRI findings typically reveal patchy T2 hyperintensities in the periventricular white matter, as well as subcortical lacunar infarcts, most commonly in the basal ganglia, thalamus, pons, internal capsule, brainstem, and cerebral white matter.

Pathology: Two primary underlying pathophysiological processes have been identified. One involves lipohyalinosis, a pathological change in the arteries that is associated with hypertension and results in a loss of vessel elasticity, thickening of vessel walls, and narrowing of penetrating arterioles that supply oxygenated blood to the deep white matter. These changes cause chronic decreases in oxygen diffusion capacity and in the perfusion of the periventricular white matter, leading to ischemic demyelination of deep white matter, low-grade tissue infarction, dilatation of perivascular spaces, reactive gliosis, and arteriolosclerosis (McManus & Stott, 2005). Additionally, there is increasing evidence that leukoaraiosis (patchy hyperintensities in the periventricular white matter) may also involve endothelial dysfunction that causes not only cerebral hypoperfusion, but also impaired autoregulation and/or increased blood–brain barrier permeability (Markus, 2007). The second mechanism involves microatheromata, that is, micro-occlusions in the orifices of the penetrating arteries.

Subtypes: Consistent with two primary pathophysiological processes, the current conceptualization of SVID involves two subtypes. The first subtype, thought to be associated primarily with lipohyalinosis, is characterized by multiple small lacunar infarcts with leukoaraiosis. The second, thought to be associated primarily with microatheromata, is characterized by a single, or a small number of, larger lacunar infarcts, in the absence of leukoaraiosis. Additionally, amyloid angiopathy is another variant of this form of SVID, and is characterized by the presence of amyloid deposits in cerebral blood vessels, which increase the risk of hemorrhagic stroke and ischemic infarcts.

Risk factors: Risk factors include hypertension, high cholesterol, diabetes, metabolic syndrome, obesity, smoking, systemic inflammation, and hypotension. Additionally, there is evidence, although not conclusive, that genetic factors play an important role in the development of SVID, and there are a number of genes involved in endothelial regulation currently under investigation.

Prognosis: There is currently no cure for SVID. Active management of vascular risk factors, such as the use of medications to treat hypertension and hypotension, may reduce or slow the progression of SVID, although in most cases the onset of vascular cognitive impairment is inevitable. Nevertheless, treatment of symptoms may improve overall functioning of the patient. For example, physiotherapy may help to improve gait and balance impairments, and various medications have demonstrated modest efficacy in improving cognitive functioning.

Future Directions

Because SVID is considered a leading cause of vascular dementia, better understanding of the cause of SVID and its underlying pathophysiology is greatly needed. SVID research has been hampered by the low mortality rate of SVID relative to other types of strokes, and thus current knowledge is based on studies using small sample sizes. Additionally, definitions and criteria for positive imaging findings and diagnosis of dementia have been inconsistent across studies. The correlation between SVID and dementia is certainly less than perfect, and better understanding of why certain cases of SVID appear to be silent is needed. Larger sample sizes and replication of findings will be important for further advancement. Large-scale collaborative studies, such as the UK Stroke Genetics Group's Yound Lacunar Stroke Resource (www.strokegenetics.co.uk), hope to further elucidate the genetic components of SVID.

Cross References

► Binswanger's Disease
► CADASIL
► Lacunar Infarcts
► Leukoaraiosis
► Vascular Cognitive Impairment

References and Readings

Fisher, C. M. (1991). Lacunar infarcts: A review. *Cerebrovascular Diseases, 1*(6), 311–320.

Hachinski, V. C., Lassen, N. A., Marshall, J. (1974). Multiinfarct dementia: A cause of mental deterioration in the elderly. *Lancet, 2*, 207–210.

Hachinski, V. C., 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*, 2220–2241.

Markus, H. S. (2007). Genes, endothelial function and cerebral small vessel disease in man. *Experimental Physiology, 93*, 121–127.

McManus, J., & Stott, D. J. (2005). Small-vessel cerebrovascular disease in older people. *Reviews in Clinical Gerontology, 15*, 207–217.

Prins, N. D., van Dijk, E. J., den Heijer, T., Vermeer, S. E., Jolles, J., Koudstaal, P. J., et al. (2005). Cerebral small-vessel disease and decline in information processing speed, executive function and memory. *Brain, 128*, 2034–2041.

Small Writing

► Micrographia

Smell

► Olfaction

Snout Reflex

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Definition

The snout reflex is elicited by lightly tapping the lateral side of the closed lips, causing the mouth to purse, resembling a snout. Each side should be tested and reported separately as the snout reflex may be present unilaterally or bilaterally. It is one of the *frontal release signs*, primitive reflexes that are normal in infants, disappear with brain maturation allowing inhibition, and reappear (are "released") in disorders that affect the frontal lobes. Like most primitive reflexes, the snout reflex probably has evolutionary/adaptive advantage in infant apes, assisting them in suckling.

References and Readings

Goetz, C. G. (2007). *Textbook of clinical neurology* (3rd ed.). Philadelphia: Saunders.

SNST

► Stroop Neuropsychological Screening Test (adult)

Social and Occupational Functioning Assessment Scale (SOFAS)

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Definition

The Social and Occupational Functioning Assessment Scale (SOFAS) was derived as a rating scale for Axis V, the clinician's judgment of overall level of functioning, in the *Diagnostic and Statistical Manual for Mental Disorders* 4th Edition (American Psychiatric Association, 2000). The SOFAS is a global rating of current functioning ranging from 0 to 100, with lower scores representing lower functioning. The SOFAS differs from the similar Global Assessment of Functioning (GAF) scale by focusing on social and occupational functioning independent of the overall severity of the individual's psychological symptoms. It also differs from the GAF by including impairments that are caused by both physical and mental disorders, thereby making it a useful assessment tool for traumatic brain injury and other neurological disorders. To be scored, impairments need to be direct effects of mental and physical health problems rather than a consequence of lack of opportunity or environmental limitations.

References and Readings

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

Social Cognition

► Awareness, Social Awareness

Social Competence

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Definition

Social competence can be defined as a repertoire of skills necessary to facilitate reciprocal interpersonal interactions in social environments. The term not only describes the ability to form relationships but to resolve conflict, thus requiring a basic set of social, emotional, cognitive, and communication skills (Larson, Whitton, Hauser, & Allen, 2007; McCabe & Meller, 2004).

Current Knowledge

According to Cavell (1990), the tri-component model views social competence as a multi-construct that comprises social adjustment, performance, and skill. Understanding these components is thought by Cavell to help understand problems in social relating, including those of typically developing individuals as well as individuals with developmental and acquired disabilities (e.g., ► autism, ► nonverbal learning disabilities, ► traumatic brain injuries) (e.g., Friedman et al., 2003; Janusz, Kirkwood, Yeates, & Taylor, 2002).

Although social competence is often judged according to societal values, and to a large extent peer acceptance, there are requisite skills that many agree on. Individuals who fail to develop this competence are not only at risk for social problems but a number of psychiatric, behavioral, and academic problems, in particular, school drop-out (see Cavell, 1990, for review).

References and Readings

Cavell, T. A. (1990). Social adjustment, social performance, and social skills: A tri-component model of social competence. *Journal of Clinical Child Psychology, 19*(2), 111–122.

Friedman, S. R., Rapport, L. J., Lumley, M., Tzelepsis, A., Vanvoorhis, A., Stettner, L., et al. (2003). Aspects of social and emotional competence in adult attention-deficit/hyperactivity disorder. *Neuropsychology, 17*(1), 50–58.

Janusz, J. A., Kirkwood, M. W., Yeates, K. O., & Taylor, H. G. (2002). Social problem-solving skills in children with traumatic brain injury: Long-term outcomes and prediction of social competence. *Child Neuropsychology, 8*(3), 179–194.

Larson, J. J., Whitton, S. W., Hauser, S. T., & Allen, J. P. (2007). Being close and being social: Peer ratings of distinct aspects of young adult social competence. *Journal of Personality Assessment, 89*(2), 136–148.

McCabe, P. C., & Meller, P. J. (2004). The relationship between language and social competence: How language impairment affects social growth. *Psychology in the Schools, 41*(3), 313–321.

Rose-Krasnor, L. (1997). The nature of social competence: A theoretical review. *Social Development, 6*(1), 111–135.

Social Language

► Pragmatic Communication

Social Problem-Solving

► Problem-Solving Training

Social Relations Intervention

► Social Skills Training

Social Skills Training

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Synonyms

Social relations intervention

Definition

Social skills training is a therapeutic approach used to improve interpersonal relations. The therapy focuses on verbal and nonverbal behaviors common in social relationships. For example, participants may be encouraged to use eye contact when speaking with other people or maintain a certain amount of personal space with the person they are speaking with. Subtleties such as inflection and tone and their impact on conversational style may be of focus during training. People often participate in social skills training because they have never learned such skills (e.g., children entering school and children with developmental disabilities); need to improve upon previously learned skills (e.g., people experiencing shyness, couples in marriage counseling, or executives taking leadership training); or need to relearn behaviors lost secondary to a mental illness or acquired brain injury (e.g., ► schizophrenia, ► ADHD, and ► traumatic brain injury). Social skills training can be conducted through individual sessions or group therapy. Therapy often incorporates role-playing to enhance generalization of skills to real-life situations.

Cross References

► Behavior Modification
► Behavioral Therapy
► Executive Functioning
► Group Therapy

References and Readings

Cook, C., Gresham, F., Kern, L., Barreras, R., Thornton, S., & Crews, S. (2008). Social skills training for secondary students with emotional and/or behavioral disorders: A review and analysis of the meta-analytic literature. *Journal of Emotional & Behavioral Disorders, 16*(3), 131–144.

Gresham, F., Mai Bao, V., & Cook, C. (2006). Social skills training for teaching replacement behaviors: Remediating acquisition deficits in at-risk students. *Behavioral Disorders, 31*(4), 363–377.

King, G., Specht, J., Schultz, I., Warr-Leeper, G., Redekop, W., & Risebrough, N. (1997). Social skills training for withdrawn unpopular children with physical disabilities: A preliminary evaluation. *Rehabilitation Psychology, 42*(1), 47–60.

Kurtz, M., & Mueser, K. (2008, November). Social skills training is effective for improving functioning among schizophrenics. *Clinician's Research Digest,* 6.

Tse, J., Strulovitch, J., Tagalakis, V., Linyan, M., & Fombonne, E. (2007). Social skills training for adolescents with Asperger syndrome and high-functioning autism. *Journal of Autism & Developmental Disorders, 37*(10), 1960–1968.

Wallace, C. (1998). Social skills training in psychiatric rehabilitation: Recent findings. *International Review of Psychiatry, 10*(1), 9–19.

Soft Signs

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Synonyms

Developmental coordination disorder; Minimal brain dysfunction; Neurological soft signs

Definition

Soft signs refer to subtle abnormalities in sensory-perceptual, motor, or other central nervous system functions. They are findings that are pathological at any age, but more subtle manifestations of hard signs or behaviors that are abnormal because they persist beyond a normal age. In contrast to hard neurological signs, they are nonspecific indicators of impairment, and are not associated with focal brain dysfunction or any specific disease process. They may not cause functional impairment. Examples of neurological soft signs (NSS) include clumsiness, motor incoordination, motor overflow, difficulty with motor sequencing or rapid successive movements, stereognosis or graphesthesia, right-left confusion, and extinction in response to double simultaneous stimulation.

Historical Background

The concept of soft signs originated in the early twentieth century. Samuel Orten was the first to suggest that children with dyslexia may also exhibit abnormalities on neurological exam such as mild motor incoordination. Bender was the first to use the term "soft neurological signs" in her study of children with schizophrenia (Spreen, Risser, & Edgell, 1995). NSS were also a component of "minimal brain injury" (MBI). The concept of MBI was developed from the work of Strauss and colleagues. They observed that some children without mental retardation or known neurological involvement exhibited behaviors and learning problems characteristic of brain injury. They attributed these behaviors to a syndrome termed MBI, and concluded that MBI could be inferred solely on the basis of behavioral signs.

Eventually, the inference of brain injury from behavioral signs was questioned, and the term MBI was replaced with "minimal brain dysfunction" (MBD). MBD referred to children with near average, average, or above average intelligence with learning or behavioral disabilities, which were presumed to be associated with deviant central nervous system function. The concept of MBD was not without critics (Rutter, 1982). Etiology in most cases was unclear or described as "congenital," covering a wide range of pre-, peri-, and postnatal events (Spreen, Risser, & Edgell, 1995). The idea that MBD resulted in a homogenous syndrome was also questioned. Characteristics of MBD included increased activity level or hyperkinesis, attention problems, and reduced impulse control, as well as perceptual-cognitive and learning difficulties. NSS were said to be present in "perhaps" one half of children with MBD. Over time, many children with no indication of brain damage were recognized to display behaviors thought to constitute MBD. The American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders-third edition (DSM-III) abandoned the concept of MBD, which was replaced with "specific developmental disorders" and "attention deficit disorder."

Current Knowledge

NSS have been studied in a wide range of child and adult populations. Higher rates of NSS are found in children with learning and reading difficulties, behavioral problems, hyperactivity, and low birth weight. NSS are associated with lower intelligence and have been found to predict later withdrawal, anxiety, and emotional disorders in children. In adults, an increased incidence of NSS has been noted in patients with obsessive–compulsive disorder, social phobia, and post-traumatic stress disorder. NSS also vary developmentally and by gender, becoming less common with age and in girls before boys (Martins et al., 2008).

NSS have been studied extensively in schizophrenia (Chan & Gottesman, 2008). They are more prevalent in patients with schizophrenia compared to individuals with other psychiatric disorders. In patients with schizophrenia, they correlate with lower intelligence, neurocognitive impairment, and poor premorbid adjustment, and have been shown to be an early precursor in children who later develop schizophrenia. Higher rates of NSS have been found in patients with first-episode psychosis, suggesting

that neurological dysfunction is not the result of disease progression or neuroleptic use, but may represent a biological marker for disordered neurodevelopment in schizophrenia.

Several studies have examined the neuroanatomical correlates of NSS (Dazzan et al., 2006). NSS in patients with psychosis have been associated with reduced volumes in the basal ganglia and heteromodal cortex (areas of the prefrontal, superior temporal, and inferior parietal cortices). In healthy adults, higher rates of NSS have been associated with reduced volumes in several brain regions. Findings suggest that certain cortical areas may represent a neuroanatomical substrate for NSS in both healthy adults and patients with psychosis.

Finally, the concept of developmental coordination disorder (DCD) is related to NSS. DCD refers to children with normal intelligence who have poor motor coordination without clear evidence of a neurological pathology such as cerebral palsy. Children with DCD are at higher risk for learning and behavioral difficulties than children without DCD. Children with both DCD and attention problems have been described as showing deficits in attention, motor control, and perception (DAMP, Lendgren et al., 1996).

Future Directions

Further insight could be gained through research examining neuroanatomical and neurocognitive correlates of DCD and NSS.

Cross References

- ▶ Babinksi Reflex
- ▶ Double Simultaneous Stimulation
- ▶ Finger Agnosia
- ▶ Graphesthesia
- ▶ Stereognosis

References and Readings

Chan, R. C. K., & Gottesman, I. I. (2008). Neurological soft signs as candidate endophenotypes for schizophrenia: A shooting star or a Northern star? *Neuroscience and Biobehavioral Reviews, 32*, 957–971.

Dazzan, P., Morgan, K. D., Chitnis, X., Suckling, J., Morgan, C., Fearon, P., et al. (2006). The structural brain correlates of neurological soft signs in healthy individuals. *Cerebral Cortex, 8*, 1225–1231.

Deuel, R. K. (2002). Motor soft signs and development. In S. J. Segalowitz, & I. Rapin (Eds.), *Handbook of neuropsychology* (2nd ed., Vol. 8, Part I, pp. 367–383).

Landgren, M., Petterson, F., Kjellman, B., & Gillberg, C. (1996). ADHD, DAMP, and other neurodevelopmental/psychiatric disorders in 6-year-old children: epidemiology and comorbidity. *Developmental Medicine & Child Neurology, 38*, 891–906.

Martins, I., Lauterbach, M., Slade, P., Luis, H., DeRouen, T., Martin, M., et al. (2008). A longitudinal study of neurological soft signs from late childhood into early adulthood. *Developmental Medicine and Child Neurology, 50*, 602–607.

Rutter, M. (1982). Syndromes attributed to "Minimal Brain Dysfunction" in childhood. *American Journal of Psychiatry, 139*, 21–33.

Spreen, O., Risser, A. H., & Edgell, D. (1995). Soft neurological signs and their significance. In *Developmental neuropsychology* (pp. 347–362). New York: Oxford University Press.

Somatization

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Synonyms

Conversion disorder; Hypochondriasis; Hysteria; Psychosomatic; Somatization disorder

Definition

Presenting with physical symptoms (i.e., somatic) that are not fully explained by medical conditions. As a psychological process, somatization is the experience and presentation of somatic symptoms rather than psychological distress, even when psychological explanations would better account for the origin of the symptoms. As such, somatization is considered to be an unconscious process or at least distinct from malingering or the intentional feigning of symptoms. It is often associated with the avoidance of psychological experiences and explanations for the distress. Thus, a person with depression may experience depression in symptoms such as fatigue, aches, and pains instead of as affective and cognitive symptoms such as feelings of sadness and guilt.

When severe enough, somatization can be diagnosed as somatoform disorders such as conversion disorder and somatization disorder. The latter is given to patients who consistently exhibit a number of physical symptoms that do not have an identifiable medical cause and for which patients seek medical treatments. Patients with somatization disorder often describe their symptoms in a vivid and

detailed manner, which can seem exaggerated or which can make the patient appear preoccupied with bodily functions and complaints. Alternately, some presentations are more stoic, as if a patient is alexithymic and can only report physical manifestations of psychological distress. However, even after medical evaluation, no explanation can be found for their symptoms or the severity of the symptoms.

Historical Background

Somatization in its various forms has been recognized for centuries, and, for example, the term *hysteria* (lit. "wandering uterus") originated with Hippocrates. In the nineteenth century, French psychiatrist Briquet carefully described hundreds of cases of hysteria, and thus somatization disorders are sometimes referred to as Briquet's disorder in dated sources. Pierre Janet and Sigmund Freud provided genuinely psychological theories of somatization, and their influence continues to this day.

Current Knowledge

Early theories of somatization were psychodynamic, and one etiological view is that the patients' psychological conflicts are unconsciously experienced and exhibited as physical symptoms. Thus, internal conflicts are literally "converted" into somatic complaints. Furthermore, the types of distress thought most likely to result in somatization are traumatic stressors that the patient may have difficulty acknowledging consciously. Psychodynamic theories of somatization could be criticized for the same weaknesses cited against all psychodynamic theories (e.g., non-falsifiable, paucity of empirical support), but there are now alternatives such as behavioral and attachment-based theories of somatization. Cultural factors often shape the precise presentation of symptoms, and the co-occurrence of somatiform disorders within families suggests that both genetic and environmental factors (e.g., learning, cultural norms) contribute to somatization.

Somatization (including somatization disorder) motivates patients to "doctor shop" in search of treatment that reduces distress. Therefore, the prevalence of somatization in medical settings, and in neurology and neuropsychology in particular, is much higher than the base rate in the general population. While somatization may seem to be only a "nuisance variable" in neuropsychological practice (i.e., largely irrelevant to brain function), it is nonetheless important to consider its impact on test performance and on patient self-report of symptoms (Lamberty, 2008). Furthermore, the presence of somatization does not rule out the possibility of coexisting neurologic disease.

Assessment of somatization often involves the use of the second edition of the Minnesota Multiphasic Personality Inventory (MMPI-2), which includes scales related to somatization. The first scale was originally termed the "Hypochondriasis" scale, and scale 3 the "Hysteria" scale. When these scales are elevated but the depression scale (scale 2) is much lower, this is considered a classic profile indicative of a high degree of somatization. In assessment contexts, it is important to differentiate somatization, which is supposed to be an unconscious process indicative of distress, from malingering, which is a more intentional exaggeration of feigning of symptoms toward the end of obtaining the patients' goals (e.g., financial compensation). Contemporary "symptom validity" measures can be essential tools in this regard.

Future Directions

Assessment and treatment of somatization would be aided by additional theoretical work, improved assessment protocols, and better treatment strategies. Cognitive theories of somatization that take into account cultural factors and local (i.e., family) influences on patients' interpretation of physical symptoms may help to explain the mechanisms responsible for the phenomenology of somatization that has been widely observed for centuries and described in great detail by early theorists such as Freud, Janet, and Breuer. Progress is being made in the assessment of somatoform disorders, which ideally will result in moving beyond mere symptom validity and malingering measures to assessments that capture the essence of somatization. Treatment approaches to somatization need perhaps the most work, as empirically validated approaches to some types of somatization (i.e., management of chronic pain) are more advanced than others (e.g., somatization disorder).

Cross References

- ▶ Malingering
- ▶ MMPI
- ▶ Pain (Neurogenic, Psychogenic)
- ▶ Symptom Validity Assessment

References and Readings

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

Lamberty, G. (2008). *Understanding somatization in the practice of clinical neuropsychology.* New York: Oxford University Press.

Lipowski, Z. J. (1988). Somatization: The concept and its clinical application. *American Journal of Psychiatry, 145*(11), 1358–1368.

Mayou, R., Bass, C., & Sharpe, M. (1995). *Treatment of functional somatic symptoms.* London: Oxford University Press.

Somatization Disorder

► Somatization

Somatoform Disorders

► Cogniform Disorder

Somatognosis

► Asomatognosia
► Body Schema

Somatosensory Cortex

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Definition

Neocortical areas involved in the processing of sensory information received from the muscles, joints, and surface of the skin.

Current Knowledge

The somatosensory cortex is thought to consist of both primary (**SI**) and secondary (**SII**), as well as unimodal association areas. SI is located along the postcentral gyrus, represented by Brodmann's areas 3a, 3b, 1, and 2. Each of these areas reflect unique, well-defined somatotopic organization with the face area being the most ventral (closest to the lateral sulcus), followed dorsally by the hand, arm, trunk, and upper leg. The lower leg and foot are represented along the medial surface of the hemispheres. The major input to this region is from the ventral posterior lateral (**VPL**) and ventral posterior medial (**VPM**) nuclei of the thalamus. In turn, the VPL nucleus has received input from the medial lemniscus and the spinothalamic tracts carrying sensations of pain, touch, temperature, stereognosis, and position sense from the trunk and extremities, while the VPM nucleus receives comparable information from the face, ears, tongue, and throat via the trigeminothalamic tracts.

A second somatosensory area (**SII**), located ventrally in the parietal operculum, is also topographically organized, but, unlike SI, which appears to primarily receive contralateral somatosensory input, SII has a substantial amount of ipsilateral, as well as contralateral, innervation, both from SI and the thalamus. Most of the input to SII, especially to its more caudal regions, seems to represent spinothalamic fibers mediating pain and temperature fibers via the posterior thalamic nuclei.

Brodmann's areas 5 and 7 in the superior parietal lobule represent the main portion of the unimodal somatosensory association cortices, although the exact role (s) of these areas remain unclear. It is generally accepted, however, that the area(s) closest to the central sulcus (areas 3, 1, and 2) likely represent more elementary somatosensory functions, while the more posterior regions, which include areas 5 and 7, represent higher levels of somatosensory integration. In many respects, the somatosensory system appears more complex and less well understood than the auditory and visual systems. This should not be surprising, given the wide range of stimuli and types of receptors from which somatosensory information is derived.

Cross References

► Somatosensory System

References and Readings

Mendoza, J. E., & Foundas, A. L. (2008). The cerebral cortex – Part III: theories of the functional organization of the brain. In J. E. Mendoza & A. L. Foundas (Eds.), *Clinical neuroanatomy – a neurobehavioral approach* (pp. 373–499). New York: Springer.

Mesulam, M.-M. (2000). Behavioral neuroanatomy: large-scale networks, association cortex, frontal syndromes, the limbic system, and hemispheric specialization. In M. Mesulam (Ed.), *Principles of behavioral and cognitive neurology* (Chapter 1, pp. 1–120). New York: Oxford University Press.

Somatosensory Evoked Potentials

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Definition

Somatosensory evoked potentials (SEPs) are electrophysiological responses detected from the brain or spinal cord in the central nervous system following repeated sensory stimulation of a peripheral nerve.

Current Knowledge

Advances in computer technology have facilitated the acquisition of electrophysiological responses such as SEPs from cortical and other central nervous system structures. Various stimuli such as tactile, vibrational, electrical or painful may be used to evoke responses from peripheral nerves. In SEPs, an electrical stimulus is most commonly used since it is easy to administer and reliable. Repeated electrical stimulus of a peripheral nerve results in multiple sensory evoked potentials and the resultant electrophysiological response is detected and averaged from the spinal cord or cortical structures of the brain. The electrophysiological responses elicited can be then assessed based on normal standard characteristics of the potential elicited and its temporal relationship to the site of stimulus.

Clinical Application of SEPs and Brain Injury

SEPs have been tested and proved useful in the clinical setting in predicting outcome in comatose patients with severe traumatic brain injury (Lew, Lee, Pan, & Chiang, 2007). Short-latency SEP (SSEP) is a part of the SEP occurring within 50 s of stimulation of the peripheral nerve and is relatively independent of level of consciousness. In early evaluation of traumatic coma patients SEPs have proved most reliable and effective in predicting poor as opposed to good outcome (Lew et al., 2003). For example, in traumatic coma bilaterally absent cortical responses predicted a 95% probability of non-awakening from coma (Robinson, 2004; http://www.neuraxis.net/services.htm). However, caution must be used in the interpretation and analysis of SEPs in the clinical setting, for example an increased level of sedation may reduce the amplitude of cortical SEP and other central nervous system insults such as spinal injuries can also compromise results. In the clinical setting SEPs are always used and interpreted in conjunction with repeated clinical evaluations and other tools of assessment such as laboratory and imaging studies (Fig. 1).

Somatosensory Evoked Potentials. Figure 1 Neuraxis use in somatosensory evoked potential (Reproduced with permission from 2005 Neuroaxis monitoring group. http://www.neuraxis.net/services.htm)

Cross References

- ► Brainstem Auditory Evoked Potentials
- ► Visual Evoked Potentials

References and Readings

Lew, H. L., Dikman, S., Slimp, J., Temkin, N., Lee, E. H., Newell, D. et al. (2003). Use of somatosensory evoked potentials and cognitive event related potentials in predicting outcome in patients with severe traumatic brain injury. *American Journal of Physical Medicine & Rehabilitation, 82*, 53–61.

Lew, H. L., Lee, E. H., Pan, S. S. L., & Chiang, J. Y. P. (2007). Electrophysiological assessment techniques: Evoked potentials and electroencephalography. In N. D. Zasler, D. L. Katz, & R. D. Zafonte (Eds.), *Brain Injury Medicine. Principles and Practice*. Demos.

Picture 2005 Neuroaxis monitoring group. http://www.neuraxis.net/services.htm

Robinson, L. R. (2004). Somatosensory evoked potentials in coma prognosis. In G. L. Kraft & H. L. Lew (Eds.), *PM&R clinics of North America*. Philadelphia: WB Saunders.

Robinson, L. R., Mickelsen, P. J., Tirschwell, D. L., & Lew, H. L. (2003). Predictive value of somatosensory evoked potentials for awakening from coma. *Critical Care Medicine, 31*, 960–967.

Somatosensory Strip

► Postcentral Gyrus

Somatosensory System

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Definition

The somatosensory system is a component of the nervous system that detects and allows for perception of pain, temperature, head and body position and movement, and touch.

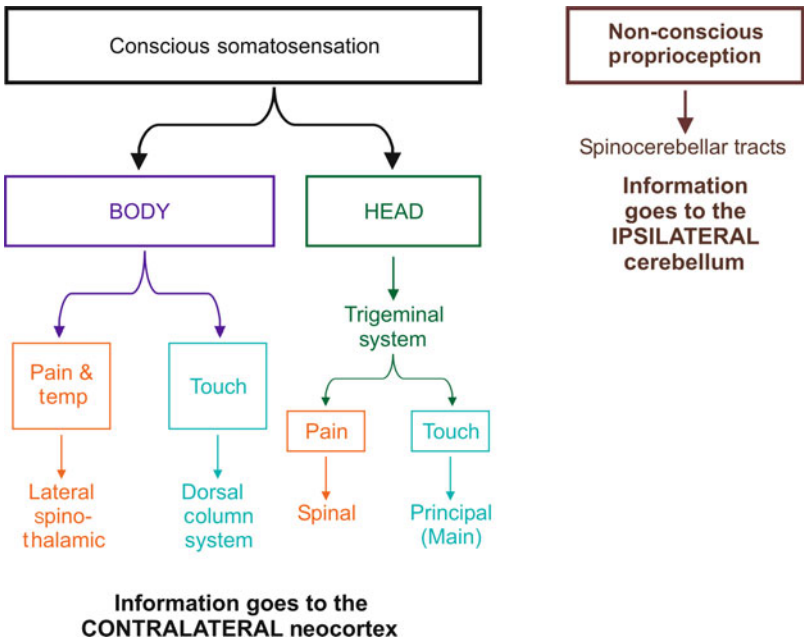
Current Knowledge

The somatosensory system is a component of the nervous system that detects and allows for perception of the modalities (sense) of pain, temperature, head and body position (called proprioception), head and body movement (called kinesthesia), and touch. The modality of touch includes the senses of pressure, vibration, and the higher-order sense of stereognosis, which is the ability to recognize and identify objects based on touch alone (without vision, audition, olfaction, and gustation). The modality of pain includes the sense of itch and tickle. The *primary* receptor surface for the somatosensory system is located in the skin; however, there are also peripheral components in muscles, tendons, and internal organs. The somatosensory system consists of a number of neural pathways that carry these senses from the starting point in skin, muscles, tendons, and internal organs to the central nervous system and ultimately to consciousness, which occurs in the neocortex. Somatosensation begins with receptors or end organs with various morphologies specialized to detect particular somatosensory modalities.

In order to further understand the somatosensory system, it is helpful to divide the neural pathways into groups based on modality and termination location. The pathways can be first divided into the group that conveys nonconscious proprioception and the group that conveys conscious somatosensation (Fig. 1). For the pathways that convey nonconscious proprioception, information about limb and body location and movement begins with receptors in muscles and tendons and ends in the ipsilateral (same side) cerebellum. Pathways that convey conscious somatosensation begin in the skin as well as in muscles and tendons and typically end in the contralateral (opposite side) neocortical hemisphere. Thus, your left cortical hemisphere processes information from the right side of the body, and right hemisphere from left side of the body.

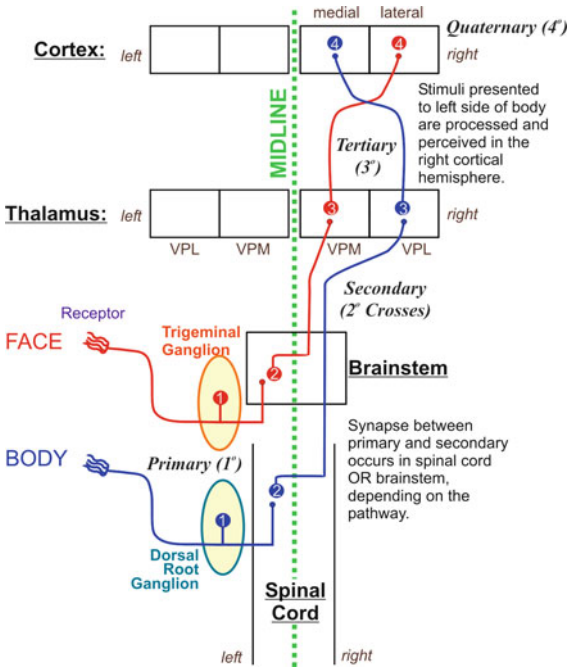
For conscious somatosensation, it is helpful to divide the pathways into those that carry information from the head and those that carry somatosensation from the body. The trigeminal system conveys information from the head. This system has a spinal component that conveys pain and temperature information, and a principal or main component that conveys fine discrimination touch. Fine discrimination touch is characterized by the ability to recognize stimuli with a high degree of spatial resolution. Pain and temperature information from the body is carried by the anterolateral system, which includes the lateral spinothalamic tract. The dorsal column system, including the medial lemniscus tract carries fine discrimination touch from the body.

The anterolateral system, located in the anterolateral portion of the spinal cord is the more primitive, slower, more crudely coding system that conveys pain and temperature. The dorsal lateral system located in the dorsal lateral spinal cord is the more recently evolved, faster, higher resolution system that conveys all aspects of touch and proprioception. The difference in speed of the pathways is due to the diameter of the primary afferent axons as well as to the amount of myelination of these axons. Myelination is a fatty substance that insulates segments of neuronal axons, allowing the action potential to "jump" those segments, and thereby speeding conduction. The primary afferents for the dorsal column system are large diameter (12–22 μm), heavily myelinated axons, allowing them to be fast conducting (80–120 m/s). In contrast, pain and temperature primary afferents of the anterolateral system are smaller diameter (0.1–1 μm), thinly or unmyelinated axons.



Somatosensory System. Figure 1 Divisions of the somatosensory system. The first division separates information going to consciousness (neocortex) from that traveling to the cerebellum for coordination of movement. Information going to the cerebellum ends up primarily ipsilaterally, while that traveling to neocortex will be located in the hemisphere contralateral to where the information was initiated. There are separate neural pathways for information from the body versus information from the head. The pathways can be further divided based on modality (sense). Pain and temperature information travels in spinal components, while fine discrimination touch information travels in the dorsal column system for pathways from the body and in the principal component of the trigeminal system for information from the head

Although there are unique pathways for pain versus touch, all of the pathways for conscious somatosensation follow a basic plan (Fig. 2). The first step is that a stimulus evokes a generator potential (depolarization graded relative to the intensity and form of the stimulus) in the specialized end organ. The end organ is associated with the first-order neuron that has the morphology of a pseudo-unipolar neuron. This morphology means that if the generator potential reaches threshold, then an action potential is generated in the axon at the junction with the end organ that then travels past the cell body and into the central nervous system. The cell body of the first-order neuron is located outside the central nervous system, in the dorsal root ganglion for information from the body, and in the trigeminal ganglion for information from the head. These ganglia are collections of neuronal cell bodies. The central portion of the first-order neuron's axon makes a synaptic connection with the second-order neuron in the pathway at the level of either the spinal cord or the brain stem. For pain and temperature information from the body, this synapse occurs in the spinal cord. For all other pathways, it occurs in the brain stem. The axon of the second-order neuron crosses the midline at the level of the cell body. So for pain and temperature information from the body, this crossing occurs in the spinal cord. For all other pathways, the crossing occurs in the brain stem. In order to associate symptoms with the site of damage or lesions, it is essential to know where the pathway crosses the midline. Lesions that occur in the pathway peripheral to the crossing will create an ipsilateral loss of sensation, while those that occur central to the crossing will create a contralateral loss of sensation. After the axon of the second-order neuron crosses the midline, it ascends to the level of the thalamus, where it synapses with the third-order neuron. Information from the body synapses within the ventral posterior lateral nucleus of the thalamus, while that from the head synapses in the ventral posterior medial nucleus of the thalamus. The thalamic neurons send their axons to the ipsilateral primary somatosensory cortex. The primary somatosensory cortex, also known as Brodmann's areas 3, 1, and 2 is the neocortex located on the postcentral gyrus.



Somatosensory System. Figure 2 Basic plan for somatosensory information traveling to consciousness. The pathway begins with a receptor or end organ, typically in the skin. The receptor is associated with a primary afferent axon that is part of the first order neuron. The first order cell body is located outside the central nervous system in the dorsal root ganglion for information from the body, and the trigeminal ganglion for information from the head. The first order neuron makes a synaptic connection with the second order neuron at the level of either the spinal cord or brainstem. The axon of the second order neuron crosses the midline at the level of the second order cell body. After crossing, the axon ascends to the thalamus to synapse with the third order neuron. The third order neuron is located in the ventral posterior lateral (VPL) nucleus of the thalamus for information from the body and the ventral posterior medial (VPM) nucleus for information from the head. The third order neuron sends its axon to somatosensory cortex where it synapses with the fourth order neuron. At each level of the pathway, there is a map of the body. The map is oriented with the head medial in thalamus and lateral in the cortex

End Organ Function

Somatosensation begins with receptors or end organs with various morphologies specialized to detect particular somatosensory modalities. The function of these end organs is to transduce mechanical energy (i.e., pressure) into electrical energy (a change in electrical potential). The end organs code the type of stimulation (e.g., pressure versus vibration), as well as the timing, intensity, and location of the stimulus.

Stimulus Type Coding

The morphology of the receptor is predictive for the type of stimulation that best activates it. Proprioceptors code position and movement of the body and head. Muscle spindle proprioceptors lie parallel to the muscle fibers, so that when the muscle fibers are stretched, the spindle is also stretched, deforming it and producing the graded potential. Golgi tendon organ proprioceptors lie in series with the muscle fibers, so that contraction of the muscle stretches and deforms the golgi tendon organ, causing the graded depolarization. Free nerve endings are activated by pain and temperature stimuli. Mechanoreceptors, including Pacinian corpuscles, Meissner's corpuscles, Merkel's disks, and Ruffini endings are best activated by various types of touch. Pacinian corpuscles are best activated by deep vibration stimuli, while Merkel's disks are well activated by light touch and texture.

Stimulus Timing Coding

The primary afferent axon associated with some end organs is activated only at the initial presentation of the stimulus and again when the stimulus is removed. This type of response is called rapidly adapting or phasic. Other primary afferent axons are activated at a high rate initially, with subsequent decrease in action potential firing rate, but are still active during the entire presentation of the stimulus. This type of response is called slowly adapting or tonic. Pacinian corpuscles produce phasic responses due to their morphology. The fibrous capsule allows the afferent axon in the center of the receptor to retake its normal shape while the stimulus is still present. The axon will only fire when the center of the receptor is deformed. When the stimulus is removed, the outer layers of capsules retake their normal shape, causing a temporary deformation of the inner layers and once again causing the axon to fire. This behavior, along with their location deep within the skin, and their large receptive fields make the Pacinian corpuscle best at sensing deep vibration. The receptive field is the area of a sensory surface that when stimulated activates a sensory neuron. In contrast, Merkel's disks fire tonically during the stimulus presentation. This behavior combined with their location in the superficial regions of the skin and their small

receptive fields make the Merkel's disks best at fine discrimination touch, particularly in the fingertips.

Stimulus Intensity Coding

Stimulus intensity is coded in two ways. First, increasingly intense stimuli will cause larger graded depolarizations in the end organ and a greater number of action potentials in the primary afferent axon associated with the end organ. This is called frequency modulation. The second method of intensity coding is through recruitment of additional axonal fibers. Increasingly intense stimuli will activate an increasing number of afferent axons.

Stimulus Location Coding

At each level of the somatosensory pathway, the relative location of the information is maintained such that a somatotopic or body-ordered map is created. For example, the neurons that will respond to touch or pain of the hand are adjacent to the neurons that will respond to touch or pain of the forearm. The resolution of spatial coding varies across the body. Resolution can be measured using a two-point discrimination test, where the distance between two stimuli required in order for perception of two separate stimuli is determined. Resolution of spatial coding is much higher on the fingertips than on the trunk. This means that the distance between two points distinguished as separate stimuli can be much smaller on the fingertips than on the trunk. This higher resolution on the fingertips is due in part to smaller receptive fields. Individual sensory neurons will respond to stimulation of a large area of the trunk (therefore a large receptive field). In addition to small receptive fields, the fingertips also have a higher density of receptors. Another contributor to higher spatial resolution is that more neocortex is devoted to processing information from the fingertips relative to the trunk. Humans explore their world primarily with fingers, which may explain why high resolution is necessary on the fingertips. In contrast, rats explore their environment primarily with their whiskers. The region of the rat somatosensory cortex devoted to processing information from the whiskers is much larger than that for other body parts.

Nonconscious Versus Conscious Somatosensation

The information from proprioceptors in nonconscious pathways helps the cerebellum coordinate movements. For example, the swinging of a baseball bat requires a motor plan (contraction of different muscles at different points in time) that is likely stored in the cerebellum once learned. In order to properly execute this motor plan, the cerebellum must have information about the starting point of the limbs. This information and continual feedback about the current location of the limbs is provided by the somatosensory system.

The same proprioceptive information is also processed by the neocortex in conscious somatosensation pathways. Conscious knowledge of where your limbs are in space is necessary to make appropriate voluntary movements and reactions to stimuli.

Plasticity in the Somatosensory System

Even in adults, the nervous system has a tremendous capacity for plasticity. For instance, if part of the somatosensory cortex is damaged then adjacent cells can take over the function. In addition, if part of the periphery is damaged, for example, the amputation of the hand, the cortex that previously processed signals from the hand will begin to respond to stimulation of the arm instead. In this way, the neurons are rarely idle. Even under normal conditions, plasticity can be observed. For instance, practicing piano can increase the amount of cortex that processes information from the fingers.

Illness

Syringomyelia is gliosis and cavitation in the midline of the spinal cord. This can result from pressure within the cerebrospinal fluid system, causing an expansion of the central canal of the spinal cord. The symptom is bilateral loss of pain and temperature focally at the level of the lesion. This is due to lesion of the second-order pain and temperature axons as they cross the midline. After crossing, these axons would normally reach the lateral part of the spinal cord, where they ascend as the lateral spinothalamic tract.

Tabes Dorsalis is a degeneration of the myelinated afferent fibers of the dorsal columns within the spinal cord. This tract is carrying fine discrimination touch and proprioceptive information. The lesion is bilateral, resulting in bilateral loss of touch and position sense within the body at the level of the affected dermatome (area of skin innervated by the sensory fibers of a single spinal nerve) and down (including lower body dermatomes). Typically with this lesion, temperature and pain perception are intact.

Brown-Sequard syndrome is a hemisection of the spinal cord, often in the cervical region. The symptoms are (a) loss of fine discrimination touch and position sense ipsilaterally for body regions from the affected dermatome and down, (b) loss of pain and temperature contralaterally for body regions from the affected dermatome and down, (c) a small region of bilateral loss of pain and temperature at the level of the lesion and 2 segments below, and (d) ipsilateral spasticity and weakness. Symptom (a) is due to lesion of the dorsal columns. Symptom (b) is due to lesion of the lateral spinothalamic tract. Symptom (c) is due to lesion of the primary afferent pain and temperature fibers entering the spinal cord to synapse in the dorsal horn. Symptom (d) is due to lesion of the lower motor neurons in the ventral horn of the spinal cord.

Wallenberg's syndrome, also called Lateral Medullary syndrome, is a lesion of the lateral medulla. This can be due to occlusion of the posterior inferior cerebellar artery which wraps around the medulla. The symptoms are (a) loss of pain and temperature in the head and face ipsilateral to the lesion due to lesion of the primary afferent fibers in the spinal trigeminal tract and the second-order cell bodies in the spinal trigeminal nucleus, (b) loss of pain and temperature in the body contralateral to the lesion due to lesion of the lateral spinothalamic tract, and (c) Clumsiness bilaterally due to lesion of the spinocerebellar tracts.

Lesions due to strokes, tumors, or head trauma may damage the ventral posterior nuclei of the thalamus or the postcentral gyrus. In both of these cases, the result will be a loss of all forms of somatosensation (touch, pressure, pain, temperature, proprioception) contralateral to the lesion. The location on the body of the loss of sensation is dependent on where within the body map the lesion occurs. If the lesion were in the ventral posterior medial nucleus of the thalamus or the lateral and ventral portion of postcentral gyrus, the deficit would be on the face or head. If the lesion were in the lateral portion of the ventral posterior lateral nucleus of the thalamus or the medial portion of the postcentral gyrus, then the deficit would be in the legs and feet.

Cross References

- ▶ Brodmann's Areas of the Cortex
- ▶ Neocortex
- ▶ Somatosensory Cortex
- ▶ Somatosensory Evoked Potentials
- ▶ Somatotopic Organization

References and Readings

Bear, M. F., Connors, B. W., & Paradiso, M. A. (2007). *Neuroscience: Exploring the brain* (3rd ed.). Baltimore, MD: Lippincott Williams & Wilkins.

Fix, J. D. (2008). *Neuroanatomy* (4th ed.). Baltimore, MD: Lippincott Williams & Wilkins.

Haines, D. E. (2008). *Neuroanatomy: An atlas of structures, sections, and systems* (7th ed.). Baltimore, MD: Lippincott Williams & Wilkins.

Kandel, E. R., Schwartz, J. H., & Jessel, T. M. (2000) *Principles of neural science* (4th ed.). New York: McGraw-Hill.

Nelson, R. J. (Ed.). (2001). *The somatosensory system: Deciphering the brain's own body image*. Boca Raton, FL: CRC Press.

Snell, R. S. (2006). *Clinical neuroanatomy*. Baltimore, MD: Lippincott Williams & Wilkins.

Urban, L. (Ed.). (1994). *Cellular mechanisms of sensory processing: the somatosensory system*. Berlin/New York: Springer.

Somatotopic Organization

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Definition

The relative distribution of somatosensory or tactile sensation for the different areas of the body as represented along the postcentral gyrus and other areas of the central nervous system.

Current Knowledge

The organization of the somatosensory cortex provides somatosensory "maps," also known as the somatosensory homunculus, that correspond to the anatomical areas along the postcentral gyrus, which receive tactile information from different regions of the body via the ventral posterolateral (limbs and trunk) and ventral posteromedial (facial areas) nuclei of the thalamus. The topography of the somatosensory receptive areas follows the same basic layout that is present in the primary motor cortex of the adjacent precentral gyrus. The organization of both the motor and somatosensory cortices seem to follow three basic principles and those are: (1) contralateral and inverted representation, (2) adjacent parts of the body lying along contiguous regions of the gyri, and (3) areas of greatest sensory sensitivity or discriminability (or in the case of motor function, the capacity for finer, discrete

movements) having the greater relative degree of cortical representation. Thus, the primary somatosensory and motor cortices for the right side of the body lie on either side of the central sulcus in the left hemisphere. The cortical areas for the face, mouth, and lips are located in the ventral portions of the gyri, near the lateral sulcus. The areas representing the arms and the hands are found more dorsally, followed by the trunk and the legs, with the upper part of the legs lying close to the longitudinal fissure that separates the two cerebral hemispheres, and the lower leg and foot being found on the medial surface of the hemispheres. In the case of the somatosensory system, because the lips, the tongue, and the hands are capable of finer tactile discriminations as a result of more dense populations of receptors at the periphery, there is greater relative amount of somatosensory cortex devoted to the processing of these inputs, especially compared to the truncal areas that are relatively less sensitive. A similar situation is found within the motor system. Although when thinking about somatotopic organization, there is a tendency to consider only the cortex, similar patterns of organization can be found in other regions of the central nervous, including the distribution of fibers in the spinal cord, brainstem pathways, thalamus, and thalamocortical radiations.

Cross References

► Homunculus

References and Readings

Carpenter, M. B., & Sutin, J. (1983). *Human neuroanatomy.* Baltimore: Williams & Wilkins.

Haines, D. E. (Ed.). (1997). *Fundamental neuroscience.* New York: Churchill Livingstone.

Nolte, J. (2002). *The human brain: An introduction to its functional anatomy.* St. Louis: C.V. Mosby.

Somesthesia

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Synonyms

Somesthesis; Tactile perception

Definition

Somesthesia is the perception of bodily sensations that include the skin, muscles, joints, and tendons. Such sensations would encompass the perception of pain, temperature, light touch, deep touch, vibration, proprioception (recognition of the relative position of a body part), and kinesthesia (awareness of movement of a limb or joint). While the visceral organs can also respond to certain types of stimuli, such as pains associated with the gut, these are not typically included in this definition.

Cross References

► Kinesthesia
► Pain Perception
► Proprioception
► Somatosensory System
► Stereognosis

References and Readings

Haines, D. E. (Ed.). (1997). *Fundamental neuroscience* (Chapters 16 and 17). New York: Churchill Livingstone.

Somesthesis

► Somesthesia

Source Memory

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Synonyms

Contextual memory

Definition

Can be thought of as a form of incidental memory and involves memory misattribution. Source memory refers

to recalling the source of learned information, such as knowledge of when or where something was learned. Often, memories are triggered by contextual information (i.e., time and place). Source memory failure may be associated with old age, stress, distractibility, or intoxication and is a phenomenon in which a person retrieves fragments of a memory without remembering how or when the fragment was acquired. Source memory impairments have been shown to be disproportionately impaired in patients with frontal lobe lesions. For instance, when asked to learn two separate lists of items, frontal lobe patients are impaired at determining if a word was on the first or second list (i.e., source), and not on the actual recall or recognition of the items. In contrast, medial temporal amnesics are impaired in recall of the actual items but not its source (e.g., first or second list).

Cross References

- ▶ Incidental Memory
- ▶ Memory
- ▶ Memory Impairment

References and Readings

Baldo, J. V., & Shimamura, A. P. (2002). Frontal lobes and memory. In A. D. Baddeley, M. D. Kopelman & B. A. Wilson (Eds.), *The handbook of memory disorders* (2nd ed.). London: Wiley.

Kolb, B., & Whishaw, I. Q. (2008). *Fundamentals of human neuropsychology.*

Span of Apprehension

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Synonyms

Apprehension span; Attention span; Span of attention; STM transfer rate

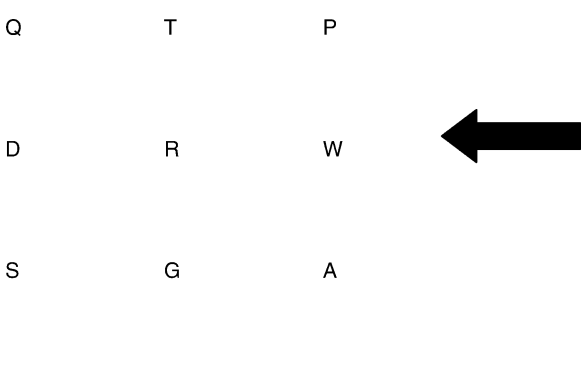
Definition

The maximum number of stimuli or units of information that can be processed at one time after brief presentation. Span of apprehension directly determines short-term memory capacity. The term span of apprehension is sometimes used interchangeably with attention span, though technically span of apprehension refers to a more basic processing capacity that influences attention span.

Historical Background

The construct of span of apprehension arose out of efforts in the field of cognitive psychology to demonstrate the processes by which information is transferred from sensory storage to short-term memory, and the constraints on this transfer. Several converging lines of research supported its existence. George Sperling's classic work on iconic memory demonstrated the existence of very short-term sensory storage or buffer, by presenting arrays of 12 letters, and showing that while people reported seeing all 12 letters after a brief presentation, they were only able to report 3 or 4 of the letters immediately following the presentation, though this number decreases to 2 then 1 then 0 relatively quickly (Fig. 1).

This sensory store decays rapidly compared with short-term memory (STM), which often lasts 30 to 40 s. The decay rate is governed by the amount of information that can be transferred between the sensory store and STM, which is in effect the span of apprehension. This span represents a disparity between what is perceived



Span of Apprehension. Figure 1 An example of the type of visual presentation used in studying span of apprehension. Visual stimuli are presented in an array. An arrow points to the row of letters that the subject will attempt to recall following presentation of the stimuli. Task demands can be modified by varying the duration between stimulus presentation and recall, the number of stimuli in the array and the way cueing to location is performed

at a more basic sensory level, and what is available for retrieval. This disparity has direct relevance to distinctions between serial and parallel processing, with the times limits imposed by the decay process providing a capacity limitation on serial processing.

Other methods, such as forced choice recognition were subsequently used to demonstrate that this span is largely independent of STM and longer-term memory capacity, and also motivational influences. Accordingly, it can be distinguished from attention span, which varies as a function of momentary changes in arousal, motivation, other attentional influences, such as interference. However, because span of apprehension is essentially the maximal transfer rate between sensory and STM, it has been viewed as an index of the maximal attention span. This maxima is strongly influenced by intrinsic neurobiological constraints, most notably the information processing speed capacity for particular individuals.

Historically, span of apprehension had its first major impact on the cognitive science of attention in studies involving dichotic listening, in which it was shown that for the most part only information that is attended to is transferred into STM. This was demonstrated in dichotic listening experiments, in which information presented to one ear that was attended was more easily recognized and recalled, whereas stimuli going to the unattended channel was not. Performance on dichotic listening paradigms was constrained however by the span of apprehension.

Current Knowledge

Span of apprehension is not routinely assessed in a direct way during standard clinical neuropsychological assessments, though a span of apprehension test (SPAN) does exist for clinicians and researchers attempting to do so. Span of apprehension paradigms continue to be employed in research studies that aim to characterize underlying information processing speed capacity, and optimal attention span. For example, studies of attention deficit disorder (ADD) have shown that visual attention span is similar for children with and without ADD when the amount of visual distraction is experimentally controlled, suggesting that the core deficit in this disorder is not span of apprehension, or more broadly attention span.

Span of apprehension has been studied extensively in severe psychiatric conditions, in particular schizophrenia and to a lesser extent bipolar illness. These studies have shown reduced span of apprehension in the presence of major symptoms, but less impairment when symptoms are not present. However, alterations in span of apprehension among schizophrenics occur as a function of length of asynchrony in stimulus onset and also masking (i.e., interference), indicating that the reduction in span is a function of attentional influences, as well as clinical factors such as schizophrenia subtype and also symptomatology.

While relatively few systemic neuropsychological studies of span of apprehension exist, some research has demonstrated reduced span in patients with brain disorders. Some generalizations can be made based on what is known about the determinants of this span, and also limited clinical evidence to date. Neurological conditions that reduce the overall processing speed for information transmission and transfer across functional brain systems are likely to reduce this span. For example, reduced span of apprehension is evident among patients with HIV-associated brain dysfunction. There is also evidence of decreased span in Alzheimer's disease, the bases for these effects are clear at this point.

Future Directions

There continues to be a need for studies aimed at distinguishing between the short-term sensory storage capabilities of posterior brain systems involved in early perceptual processing and information transmission and transfer speed constraints on span of apprehension. Within clinical neuropsychology there may be value in routinely incorporating specific measures of span of apprehension, though currently speed of processing estimates are typically derived from other tests.

Cross References

► Dichotic Listening
► Information Processing Speed

References and Readings

Denton, C. L., & McIntyre, C. W. (1978). Span of apprehension in hyperactive boys. *Journal of Abnormal Child Psychology, 6*(1), 19–24.

Estes, W. K. (1975). The locus of inferential and perceptual processes in letter identification. *Journal of Experimental Psychology: General, 104*(2), 122–145.

Sperling, G., Budiansky, J., Spivak, J. G., & Johnson, M. C. (1971a). Extremely rapid visual search: The maximum rate of scanning letters for the presence of a numeral. *Science, 174*(6), 307–311.

Sperling, G., Budiansky, J., Spivak, J. G., & Johnson, M. C. (1971b). Extremely rapid visual search: The maximum rate of scanning letters for the presence of a numeral. *Science, 174*(4006), 307–311.

Sperling, G., & Melchner, M. J. (1978). The attention operating characteristic: Examples from visual search. *Science, 202*(4365), 315–318.

Treisman, A., & Geffen, G. (1968). Selective attention and cerebral dominance in perceiving and responding to speech messages. *The Quarterly Journal of Experimental Psychology, 20*(2), 139–150.

Span of Attention

► Span of Apprehension

Span Test

► Digit Span

Spastic Bulbar Palsy

► Pseudobulbar Palsy

Spastic Gait

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Synonyms

Stiff-legged gait

Definition

Spastic gait is caused by increased muscle contraction of the limbs. As a result, the individual must drag a leg if the spasticity is on one side or waddle if both of the legs have increased tone. A spastic gait can be seen after stroke, with multiple sclerosis and other neurodegenerative diseases.

Cross References

► Circumduction
► Multiple Sclerosis
► Stroke

References and Readings

Hallett, M. (2007). Stiffness syndrome. In S. Fahn, & J. Jankovic (Eds.), *Principles and Practice of Movement disorders* (pp. 295–299). Philadelphia: Churchill Livingstone Elsevier.

Spatial Competence

► Route Finding

Spatial Dyscalculia

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Definition

Impaired arithmetical problem-solving due to difficulties handling the two-dimensional or spatial arrangement of the numbers, especially when multi-digit figures are involved. In performing certain mathematical operations involving multi-digit numbers, where one starts from the rightmost column and works leftward, it is essential that the positions of the numbers be respected with regard to their columnar arrangement. Thus, solving such problems requires precise spatial organization and meticulous alignment. This applies not only to written problems but to purely mental calculations as well, as these are also vulnerable to spatial disruption. An additional problem can occur in cases of left-sided neglect where numbers on the left side of the equation are ignored. Such disturbances are typically the result of right hemispheric lesions, particularly these involving the parietal-occipital cortices.

References and Readings

Denberg, N. L., & Tranel, D. (2003). Acalculia and disturbances of the body schema. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (pp. 161–184). New York: Oxford University Press.

Hartje, W. (1987). The effect of spatial disorders on arithmetical skills. In G. Deloche & X. Seron (Eds.), *Mathematical disabilities: a cognitive neuropsychological perspective* (pp. 121–135). Hillsdale, NJ: Lawrence Erlbaum Associates.

Spatial Filtering

► Spatial Frequency Analysis

Spatial Frequency Analysis

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Synonyms

Spatial filtering

Definition

Spatial frequency analysis is a specialized form of systems analysis. It is used to study the spatial filtering properties of optical imaging devices. A filter in systems analysis is thought of as a stimulus–response mechanism that responds better to some stimuli than others. A linear system is one that accepts an input and produces an output. The input and output can be one-dimensional (time), two-dimensional (space), or higher dimensional. Spatial frequency analysis is a way in which one can predict the response of a linear spatial filter to any spatial pattern or complex waveform. Sinusoidal (sine-wave) gratings are used to determine the spatial filtering characteristics under study. This is because a sine function is the one spatial function that can pass through a linear filter unchanged in form. A sine-wave grating of specified orientation, spatial frequency, amplitude, and spatial phase is thought of as a Fourier component. All visual images can be described as a set of Fourier components that when added together recreate the original image. Spatial filters are selective for passing on some Fourier components and filtering out others. A high-pass filter transmits whatever input is present above a particular spatial frequency, a low-pass filter transmits what is impinging below a certain spatial frequency, and a band-pass filter rejects spatial frequencies at both ends. For example, a microscope is a three-dimensional low-pass linear system designed to both magnify the image and filter out some high spatial frequencies (Braddick, 1981; DeValios & DeValios, 1988; Messe, 2000; Shapley & Lennie, 1985).

Current Knowledge

Spatial frequency analysis has extended to the study of the optical properties of the eye, imaging characteristics of single neurons in electrophysiology, and the visual system as a whole in psychophysics. In biological systems, it can provide a description of how spatial information is processed or how neurons behave when signals are summed linearly. Studies suggest that early spatial vision consists of multiple spatial filters. Spatial frequency analysis cannot, however, be a comprehensive model of a biological system because of its assumption of linearity. It is unable to describe the behavior of neurons that combine signals in a nonlinear fashion.

The way in which a nonbiological optical system (lens, camera, microscope, etc.) passes spatial information can be determined by its modulation transfer function (MTF). It is an index of the degree to which each spatial frequency of an image (with known amplitude or related contrast) is transferred by the system by measuring the modulation (i.e., amplitude) of each spatial frequency in the output image. The MTF of a spatial filter is characterized by: (1) its preferred spatial frequency, orientation, and spatial phase (i.e., those properties that produce the greatest output) and (2) its bandwidth (the range of Fourier components to which it responds). The output image contains only those Fourier components from the input image that the filter is selectively tuned (or within its passband).

The MTF for single cells in the visual system can be determined in electrophysiological studies, where a cell's discharge rate is measured for a range of spatial frequencies of constant contrast. These studies suggest that cells in early spatial vision act, in some ways, as spatial filters in that they are selective for passing on some Fourier components and filtering out others. Moreover, that the filtering becomes increasingly refined as information is further transferred from the eye to the visual cortex. For example,

the optical properties of the eye are characterized as a low-pass isotopic filter because its output image contains low spatial frequencies but no high spatial frequencies at any orientation. Retinal cells and cells within the lateral geniculate nucleus pass only a band of spatial frequencies at any orientation, referred to as band-pass isotropic filters; and some cells within the visual cortex pass bands of spatial frequencies at only specific orientations (oriented band-pass filters).

The MTF of an entire biological system is not directly measurable. The spatial frequency response or contrast sensitivity function (CSF) provides an estimate of the MTF for human vision. It is the contrast that is required by an observer for the detection of each of a variety of different spatial frequencies (the number of sinusoidal luminance cycles per degree of visual angle [c/deg]). In psychophysical studies, the contrast value is varied until the pattern is at the visibility threshold. Methods include two-interval forced choice, single interval seen/not seen, and method of adjustment. Sensitivity is defined as 1/threshhold. The procedure is repeated across a range of spatial frequencies (e.g., 0.5 to 32 c/deg) and the CSF is a plot of sensitivity versus spatial frequency on logarithmic axes. It is an inverted U-shaped function with, for humans, the highest sensitivity in the mid-spatial frequency range (2–6 c/deg). Sensitivity decreases for both higher and lower frequencies with a sharper drop in sensitivity to higher spatial frequencies. The attenuation to both high and low spatial frequencies is thought to reflect both optical filtering (especially for high spatial frequencies because of the optics of the eye, receptor spacing) and neural processes. The spatial frequency CSF also varies with changes in luminance level, retinal location, and temporal frequency.

The CSF is a way to describe the spatial frequency tuning or selectivity of the human visual system. It represents multiple spatial filters or "channels" tuned for a range of different spatial frequencies. Psychophysical evidence of multiple spatial frequency channels come from studies of frequency-specific adaptation, spatial frequency specific aftereffects, spatial frequency selective masking, and subthreshold summation. For example, in frequency-specific adaptation studies, the subject is exposed to a high contrast sine-wave grating for several minutes before their CSF is measured. Post-adaptation aftereffects are observed which consist of increased detection thresholds (reduced sensitivity) for gratings at and close to the spatial frequency that they had adapted to, with no effect on the detection of higher or lower spatial frequencies. Adapting to different spatial frequencies results in reduced sensitivity at different locations in the CSF but is always close to the spatial frequency that the subject adapted to. This suggests that adaptation desensitizes the filter tuned to the adapting stimulus and that there are multiple spatial filters tuned for different spatial frequencies.

The brief summary of spatial frequency analysis presented here is not meant to imply that the visual system is strictly a Fourier analyzer because most of the requirements for a rigorous linear spatial filter are not met in biological systems. Requirements include linearity, spatial delocalization, narrow bandwidth, spatial homogeneity, and encoding of amplitude and phase. Spatial frequency analysis is, however, usual as a descriptive measure; has predictive power when the requirement of linearity is met; and in early vision, the visual system appears to perform, at least crudely, spatial frequency analysis of patterns into frequency components (Braddick, 1981; DeValios & DeValios, 1988; Messe, 2000; Shapley & Lennie, 1985).

Cross References

▶ Fourier Analysis

References and Readings

Braddick, O. (1981). Spatial frequency analysis in vision. *Nature, 291*, 9–11.

DeValois, R. L., & DeValois, K. K. (1988). *Spatial vision*. New York: Oxford University Press.

Meese, T. S. (2000). *A tutorial essay on spatial filtering and spatial vision*. www.aston.ac.uk/downloads/lhs/Meese%20Intro%20Advanced.pdf

Shapley, R., & Lennie, P. (1985). Spatial frequency analysis in the visual system. *Annual Review of Neuroscience, 8*, 547–583.

Spatial Inhibition

▶ Lateral Inhibition

Spatial Neglect

▶ Hemiinattention
▶ Hemispatial Neglect
▶ Neglect
▶ Neglect Syndrome
▶ Visual Neglect

Spatial Orientation

► Route Finding

Spatial Processing

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Synonyms

Auditory spatial processing; Visual-spatial processing

Definition

Spatial processing is sensing and integrating information pertaining to a location in space.

Historical Background

Lashley (1948) was among the early researchers who commented on the neural pathways involved in visual-spatial perception, suggesting that these systems do not extend beyond the striate cortex. Subsequent neurobehavioral, physiological, and anatomical studies demonstrated that these pathways extend beyond striate cortex to include parietal and temporal lobes. In 1969, Schneider proposed an anatomical separation between the identification of a visual stimulus and its location in space. He postulated that spatial information was coded by the retinotectal pathway, whereas stimulus identification was processed by the geniculostriate system. Investigations by Mishkin, Ungerleider, and Macko (1983) later provided evidence to support two separate cortical visual pathways: one specialized for "object vision" and the other for "spatial vision," although the streams that process this information are different from Schneider's propositions. Instead, spatial location was thought to be subserved by an occipitoparietal projection system, which followed the course of the superior longitudinal fasciculus. This pathway provided interconnections for the striate, prestriate, and inferior parietal areas. It was also proposed that links between this pathway and dorsal limbic and frontal cortex allowed for the construction of cognitive spatial maps. As originally conceptualized, pathways for object and spatial vision were quite sharply separated, with more modern views emphasizing interconnections among these two streams at various processing levels. Goodale and Milner (1992) extended the model of the dorsal stream to include the posterior parietal region, an area that plays an important role in sensorimotor transformations for visually guided actions directed at objects.

Current Knowledge

Spatial processing has been used broadly to encompass a number of different cognitive domains. Animal work has focused on the well-known hippocampal "place cells" (O'Keefe & Dostrovsky, 1971), while topics such as spatial attention, spatial orientation, spatial construction, and spatial imagery have commanded ongoing debates in cognitive psychology and cognitive neuroscience (for a review, see Mast & Lutz, 2007)]. Information related to the spatial location of objects is distributed in complex cortical and sub-cortical structures and includes the processing of both visual and auditory information.

Visual information is processed in two functionally distinct pathways: the ventral and dorsal streams. The ventral stream, also known as the "what" pathway, is principally responsible for object recognition. It receives its main input from the parvocellular layer of the lateral geniculate nucleus of the thalamus, projecting from primary visual cortex (V1), through V2 and V4, to eventually terminate in the inferior temporal region. The dorsal stream, also known as the "where" or "how" pathway, processes information about the spatial location of objects in space. The dorsal stream projects from V1 to the parietal lobe. The dorsal and ventral streams are interconnected in various respects (Kaas & Lyon, 2007). Area V3 is a cortical region, which appears to be involved in both the dorsal and ventral streams. The ventrolateral nucleus of the lateral pulvinar and nuclei of the inferior pulvinar relate to occipital and temporal areas of the dorsal and ventral streams. These pulvinar regions contain precise representations of the contralateral visual hemifield.

Auditory spatial processing is accomplished via distinct neural pathways (Nelken, 2008). Auditory space perception involves a combination of binaural disparities, spectral cues, and temporal factors. Changes in the spatial location of a sound involve the planum temporal, which is

a cortical region just posterior to the primary auditory cortex, as well as other more posterior regions. Evidence from animal studies with the macaque delineates two anatomical pathways involved in processing auditory spatial information. The "where" pathway originates from caudal to primary auditory cortex, whereas the "what" pathway begins rostrally. Neurons in the ventral prefrontal cortex also appear to be selectively attuned to the spatial location of auditory information. Determining the location of a sound is additionally reliant on the detection and integration of monaural and binaural cues generated by the interaction of sound waves and the head and external ears (King et al., 2007). Localization of sounds in the horizontal plane is based primarily on binaural discrepancy cues. Spectral cues generated by each external ear allow for localization in the vertical plane, discrimination between the front and back, and localization of sounds using one ear alone. The processing of monaural and binaural localization cues begins in the brainstem and continues through the inferior colliculus in the midbrain. Spectral cues are processed in the midbrain and superior colliculus. While much auditory spatial processing occurs at the subcortical level, there is emerging evidence that implicates the involvement of cortex in the localization of sounds in the presence of other competing sources.

Future Directions

Modern views of the dorsal stream suggest that the inferior parietal cortex appears to be involved in other functions in addition to spatial processing (Husain & Nachev, 2007). These include the detection of salient new items embedded in a sequence of events and in maintaining or controlling attention over time. Data from lesion studies and functional brain imaging investigations support this view, extending the role of inferior parietal cortex to other functionally important processes. Recordings of cell populations in the cortex of monkeys is illuminating the functional divisions of parietal cortex for cognitive spatial versus sensorimotor processing (Chafee, Crowe, Averbeck, & Georgopoulos, 2005).

Sex differences in visuospatial functions have also been hotly debated and remain an area requiring further research. A male advantage has been shown on tests of spatial orientation (e.g. Hiscock, 1986), object location memory (Postma, Izendoorn, & De Haan, 1998), and learning spatial placement by touch, although others have also refuted these findings.

Cross References

- ▶ Auditory Discrimination
- ▶ Auditory Processing
- ▶ Visual-Spatial Ability
- ▶ Visual System

References and Readings

Chafee, M. V., Crowe, D. A., Averbeck, B. B., & Georgopoulos, A. P. (2005). Neural correlates of spatial judgement during object construction in parietal cortex. *Cerebral Cortex, 15*, 1393–1413.

Goodale, M. A., & Milner, A. D. (1992). Separate visual pathways for perception and action. *Trends in Neurosciences, 15*, 20–25.

Hiscock, M. (1986). On sex differences in spatial abilities. *American Psychologist, 41*, 1011–1018.

Husain, M., & Nachev, P. (2007). Space and the parietal cortex. *Trends in Cognitive Sciences, 11*, 30–36.

Kaas, J. H., & Lyon, D. C. (2007). Pulvinar contributions to the dorsal and ventral streams of visual processing in primates. *Brain Research Reviews, 55*, 285–296.

King, A. J., Bajo, V. M., Bizley, J. K., Campbell, R. A. A., Nodal, F. R., et al. (2007). Physiological and behavioural studies of spatial coding in the auditory cortex. *Hearing Research, 229*, 106–115.

Lashley, K. S. (1948). The mechanism of vision; Effects of destroying the visual associative areas of the monkey. *Genetic Psychology Monographs, 37*, 107–166.

Mast, F., & Lutz, J. (Eds.). (2007). *Spatial processing in navigation, imagery and perception*. New York: Springer.

Mishkin, M., Ungerleider, L. G., & Macko, K. A. (1983). Object vision and spatial vision: Two cortical pathways. *Trends in Neurosciences, 6*, 414–417.

Nelken, I. (2008). Processing of complex sounds in the auditory system. *Current Opinion in Neurobiology, 18*(4), 413–417.

O'Keefe, J., & Dostrovsky, J. (1971). The hippocampus as a spatial map. Preliminary evidence from unit activity in the freely-moving rat. *Brain Res., 34*, 171–175.

Postma, A., Izendoorn, R., & De Haan, E. H. (1998). Sex differences in object location memory. *Brain Cogn, 36*(3), 334–345.

Schneider, G. E. (1969). Two visual systems. *Science, 163*, 895–902.

SPD

▶ Schizotypal Personality Disorder

SPE

▶ Reitan–Klove Sensory Perceptual Examination

Special Education

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Definition

Special education refers to those programs or services offered for students identified with educational exceptionalities. This umbrella term may include both gifted and talented students and students with disabilities. Most frequently, however, it refers to students who are failing to meet the curricular requirements in a general education classroom setting. It is important to note that special education no longer refers to a place or a particular classroom. Instead, it refers to a variety of services that are offered to help children be successful in the least restrictive environment.

Historical Background

Special education ideals were evidenced in 1905 when Alfred Binet and Théodore Simon created the Binet–Simon Scale to predict which students were most likely to be successful in a classroom. Those students with low scores would be placed into a special classroom to receive remedial instruction. Since their time, many separate schools and classrooms were created to educate students who were resistant to regularly utilized teaching techniques in the general classroom. Unfortunately, students with disabilities had little legal protection. Some students were frequently excluded from education, while others were placed in classrooms or institutions that provided substandard facilities and resources. In the USA, the Individuals with Disabilities Educational Act (IDEA) provided a federal legal mandate for the free and appropriate public education (FAPE) in the Least Restrictive Environment (LRE) for students with disabilities. Originally passed in 1975, several reauthorizations of IDEA have taken place. In 1997, reauthorization of IDEA directed schools to provide services to help transition those students from special education settings to adulthood by identifying necessary training and supports for a student to develop a career, to have a place to live, the access community resources, and to take care of themselves. The 2004 reauthorization authorized schools to use a response to intervention (RTI) model to aid in the determination of a student's eligibility for a learning disability. In the USA as of 2005, it was estimated that 6.5 million school-aged children were receiving special educational services while 200,000 infants and toddlers were receiving early intervention programs and services.

Rationale or Underlying Theory

Special education legislation is designed to improve the educational and vocational outcomes for students with disabilities that hinder their progress in the general education classroom. The goal of any educational team is to provide a student with the highest quality education in the LRE, or the environment that maximizes the amount of interaction a student with a disability spends with grade-level peers and teachers.

Goals and Objectives

Special education most directly addresses educational needs of children from birth to 21 years of age. In particular, the deficits these children and youth display must have an impact on educational outcomes such as reading, math, written expression, or, in some cases, socialization. Some children may have been identified as having a disability by a physician or other specialist. However, if that disability has not been judged to have a significant educational impact, the child will not qualify for special education services.

Neuropsychologists must also realize that special education is not an intervention. Instead, it provides the legal means to access resources that ensure a child will receive needed interventions. Thus, a practitioner of neuropsychology in the community will need to consult with members of a school to include needed accommodations, modifications, and interventions on a student's individualized education plan (IEP). Practitioners of neuropsychology have expertise that may be useful at many levels of the special education process. While a student is being evaluated for special education, a neuropsychologist provides expertise at analyzing and synthesizing broad arrays of assessment data and turning that assessment data into effective recommendations for interventions. In particular, neuropsychological expertise may be required to better understand the services that child needs who has experienced a brain injury, or who has a pernicious learning difficulty or emotional/behavioral problem. Likewise, a neuropsychologist must work closely with a

school district to implement a treatment plan that can help the child be successful in the school setting. Whether a child is in need of close supervision, frequent memory cueing, a quiet space when they become over stimulated, or social skills instruction, the neuropsychologist will need to provide careful and deliberate consultation services to the members of the school district. In some cases, the neuropsychologist may need to teach a particular intervention to the school personnel while, in other cases, the neuropsychologist may need to come up with a creative treatment plan that optimizes the resources available to the school.

Treatment Participants

There are a variety of reasons a student may qualify for special education services. These categories include learning disability, an emotional or behavioral disability, a physical disability or other health impairment, mental retardation, autism, or a speech language disability. It is important to note that special education is designed for individuals with educational disabilities. For those who have disabilities that need accommodations, the Americans with Disabilities Act (ADA) legislates 504 plans. These accommodations frequently involve questions of access such as wheelchair ramps for individuals with physical problems, enlarged print to those who are visually impaired, or interpreters who may sign lectures. Occasionally, students with mental health disorders such as attention-deficit/hyperactivity disorder (ADHD) or depression may receive accommodations through section 504 of the ADA. However, the curriculum demands cannot be modified through a 504 plan.

Treatment Procedures

Special education supports cover a broad array of both direct and related services. These services may include a separate classroom, a separate teacher, separate school, physical and occupational therapy, speech therapy, school psychological or social work services, or consultative services. There are three broad types of special education services that students may receive: accommodations, modifications, and interventions/specialized instructional techniques.

Accommodations and modifications have to do with the curriculum the student is accessing. An accommodation is something that does not change the curricular expectations, but allows a student to access the curriculum. Glasses, wheelchair ramps, or allowing students to take tests in other places or have portions of the test read to them are all accommodations. A modification entails changing the curriculum to better meet the student's needs. An extreme example may be not teaching Shakespeare to a student because that student should instead work on life skills or reading for survival.

An intervention is something done to change a behavior. It may include teaching, rehabilitation, altering environments (antecedent control), or manipulating the consequences of a behavior. It is important to note that behavior may be a social interaction or action, may be an academic interaction, or may simply be a thought or thought process.

In order to receive special education services, a specific eligibility requirement must be met. While federal law provides a framework for identifying students with disabilities, each state and school district may interpret the federal law slightly differently. For a student to receive special education services, an eligibility evaluation must take place to determine if a student meets state and federal criteria for an exceptionality. The evaluation must address a student's abilities in some or all of the following realms: academic achievement, cognitive, communicative, social/emotional, physical motor, and adaptive. For students above the age of 16, evaluations must also focus on a student's ability to transition from school settings to postsecondary education or work settings. As a result of this evaluation, a student may meet the eligibility criteria for special education. Typically, a student may qualify for special education services due to a learning disability, an emotional or behavioral disability, a physical disability or an other health impairment, mental retardation, autism, or a speech language disability. Once a child has been found eligible, an IEP meeting takes place. The IEP document that is created during this meeting is a legally binding agreement that stipulates the special education services a child will receive.

Efficacy Information

Special education has long been criticized for its failure to address the needs of students. In general, critics contend that students in special education are denied opportunities to learn from curricular and pedagogical experts when they are removed from the general classroom. In February, 2010, an analysis of students who had received special education for the previous 10 years found that special education had either a statistically insignificant

or a negative effect on student outcome data when compared to similarly functioning peers.

While the effect is certainly different based on the types of services a student is receiving, the most frequent problems include a shortage of qualified service providers as well as a reluctance to use evidence-based intervention to meet the needs to students. IEPs with unclear objectives and generic educational techniques have also been blamed for preventing students with disabilities from succeeding in special education.

Outcome Measurement

Because special education services are provided for a broad range of disabilities, outcome measures are varied. However, each child has IEP goals that must be observable and measurable indicators of success. Parents must receive reports on these goals as frequently as students in the general education receive grades. These goals may be reported as observation data, curriculum-based measurements or assessments, norm-referenced assessments, portfolio reviews or other indicators of a child's progress.

Qualifications of Treatment Providers

There are a variety of individuals who provide services to students with disabilities. Primarily, special education teachers, school psychologists, school social workers, counselors, occupational therapists, speech language therapists, and physical therapists provide services to students with disabilities. These individuals are most frequently employed by a school district, and are credentialed through a state department of education. In some states, service providers are licensed through a department of regulatory agencies. It is not uncommon for a school district to hire a consultant to provide services – especially if the district is small or has a small number of students in need to specialized care. While all providers must hold a bachelor's degree, the majority hold graduate degrees.

Cross References

► 504 Plan
► Educational Testing
► Individualized Education Plan
► Individuals with Disabilities Education Act

References and Readings

Bjoraker, K. J. (2005). Sample neuropsychological IEP. In R. C. D'Amato, E. Fletcher-Janzen, & C. R. Reynolds (Eds.), *Handbook of school neuropsychology.* Hoboken, NJ: Wiley.

Individuals with Disabilities Improvement Act of 2004. Downloaded from http://idea.ed.gov/download/statute.html on March 20, 2010.

Morgan, P. L., Frisco, M. L., Farkas, G., & Hibel, J. (2010). A propensity score matching analysis of the effects of special education services. *Journal of special education, 43*, 236–254.

U.S. Department of Education (2008). U.S. Department of Education, Office of Special Education Programs' (OSEP's) Website. Retrieved November 12, 2008, from http://idea.ed.gov/explore/home.

Specialty Guidelines

► Specialty Guidelines for Forensic Psychology

Specialty Guidelines for Forensic Psychology

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Synonyms

Practice development; Practice guidelines; Specialty guidelines

Definition

The Specialty Guidelines for Forensic Psychologists were originally developed in 1991. They were informed by the Ethical Principles of Psychologists (APA, 1990) and were meant to be consistent with them, were designed to provide more specific guidance to forensic psychologists in monitoring their professional conduct when acting in assistance to courts, parties to legal proceedings, correctional and forensic mental health facilities, and legislative agencies. The primary goal of the Guidelines was to improve the quality of forensic psychological services offered to individual clients and the legal system with the goal of enhancing forensic psychology as a discipline and profession. The specialty guidelines are undergoing revision with the most recent iteration disseminated on 2/13/2005.

Historical Background

The 2/13/2005 document (and it subsequent interactions) is intended to replace the 1991 *Specialty Guidelines for Forensic Psychologists* that was approved by the American Psychology-Law Society, Division 41 of the American Psychological Association (APA) and the American Board of Forensic Psychology.

Current Knowledge

Forensic psychology refers to all professional practice by any psychologist working within any subdiscipline of psychology (e.g., clinical, developmental, social, and cognitive) when the intended purpose of the service is to apply the scientific, technical, or specialized knowledge of psychology to the law and to use that knowledge to assist in solving legal, contractual, and administrative problems. Application of the *Guidelines* does not depend on the practitioner's typical areas of practice or expertise, but rather on the service provided in the case at hand. The *Guidelines* are intended to apply in all matters in which practitioners provide forensic psychological expertise to judicial, administrative, and educational systems including, but not limited to examining or treating persons in anticipation of or subsequent to legal, contractual, administrative, or disability determination proceedings; offering expert opinion about psychological issues in the form of *amicus* briefs or testimony to judicial, legislative or administrative bodies; acting in an adjudicative capacity; serving as a trial consultant or otherwise offering expertise to attorneys, the courts, or others; conducting research in connection with, or in the anticipation of, litigation; or involvement in educational activities of a forensic nature.

According to the Guidelines, psychological practice is not considered forensic solely because the conduct takes place in, or the product is presented in, a tribunal or other judicial, legislative, or administrative forum. Similarly, when a party (such as a civilly or criminally detained individual) or another individual (such as a child whose parents are involved in divorce proceedings) is ordered into treatment with a practitioner, that treatment is not necessarily the practice of forensic psychology. The provision of forensic services and functions may include a wide variety of psycholegal roles and functions. Psychologists may function as researchers, advisors, consultants, examiners, treatment providers, mediators, or negotiators, and as arbiters, serving parties, attorneys, and the courts.

The *Specialty Guidelines* are regarded as aspirational in nature and recommend professional behavior and conduct for forensic practitioners. As such, they differ from practice standards and other required codes of conduct that are mandatory and may be accompanied by an enforcement mechanism. Guidelines reflect aspirations for accomplishment and are not accompanied by an enforcement mechanism; they are intended to inform the judgment of forensic psychologists and not replace it. As such, they are advisory in areas in which the forensic practitioner has discretion to exercise professional judgment that is not prohibited or mandated by the American Psychological Association Ethical Principles of Psychologists and Code of Conduct (EPPCC: APA, 2002) or by applicable law, rules, or regulations. The *Guidelines* neither add obligations to nor eliminate obligations from the EPPCC, but provide additional guidance for psychologists. The *Guidelines* are not intended to serve as a basis for disciplinary action or civil liability. The standard of care is established by a competent authority (e.g., state licensing board) not by the *Guidelines.* The *Guidelines* may assist in establishing standards of care in their attempt to identify the best possible practice, but they do not, in and of themselves, identify what other conduct may also be competent practice and what may be the standard of care in a particular case.

The current iteration of the Specialty Guidelines includes a number of areas that are relevant to the practice of forensic psychology, including the various responsibilities (e.g., integrity, impartiality, trust, and respect) of forensic psychologists. The following areas are emphasized: competence; diligence; relationships; fees; notification, assent, consent and informed consent; conflicts in practice; privacy, confidentiality, and privilege; methods and procedures; assessment; documentation; and professional and other public communications.

Cross References

► American Academy of Clinical Neuropsychology (AACN)
► American Psychological Association Ethical Principles of Psychologists and Code of Conduct

References and Readings

American Academy of Clinical Neuropsychology (2007). American Academy of Clinical Neuropsychology AACN Practice Guidelines for

Neuropsychological Assessment and Consultation. *The Clinical Neuropsychologist, 21*, 209–231.
American Psychological Association (2002a). Ethical Principles of Psychologists and Code of Conduct. *American Psychologist, 57*, 1060–1073.
American Psychological Association (2002b). Criteria for practice guideline development and evaluation. *American Psychologist, 57*, 1048–1051.
American Psychological Association (2005). Determination and documentation of the need for practice guidelines. *American Psychologist, 60*, 976–978.
Committee on Ethical Guidelines for Forensic Psychologists (1991). Specialty guidelines for forensic psychologists. *Law and Human Behavior, 15*, 655–665.
Updated (2/13/05) version of the Specialty Guidelines can be found at: http://www.ap-ls.org/links/SGFP%20version%202.0%20of%2002-14-05%20for%20posting%20to%20the%20discussion%20list.pdf

Specific Reading Disability

► Dyslexia

Specificity

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Definition

A clinical outcome statistic concerned with evaluating the accuracy of a test/measure to predict what the test/measure purports to predict. Specificity is the *true-negative* rate for a test/measure. It answers the question, knowing that the person *does not belong* to a certain group (e.g., patients exhibiting poor effort), what percentage of these people will actually be correctly identified as *not belonging* to that group by a particular test/measure (e.g., ► Test of Memory Malingering [TOMM]). For example, a specificity of 0.45 indicates that 45% of the people who *do not belong* to the poor effort group (i.e., adequate effort group) were correctly identified by the TOMM as providing adequate effort. Specificity is calculated by dividing the number of people accurately identified by the test as *not belonging* to the target behavior group (i.e., adequate effort group or Test Negative, Target Behavior Absent) by the number of people actually known to be in the adequate effort group (Total Target Behavior Absent).

Cross References

► Negative Predictive Power
► Outcome/Outcome Measurement
► Positive Predictive Power
► Receiver Operating Characteristics Curve (ROC)
► Sensitivity

References and Readings

Retzlaff, P. D., & Gibertini, M. (1994). Neuropsychometric issues and problems. In R. D. Vanderploeg (Ed.), *Clinician's guide to neuropsychological assessment* (pp. 195–209). Hillsdale, NJ: Lawrence Erlbaum.
Slick, D. J. (2006). Psychometrics in neuropsychological assessment. In E. Strauss, E. M. S. Sherman, & O. Spreen (Eds.), *A compendium of neuropsychological tests: Administration, norms, and commentary* (pp. 3–43). New York: Oxford University Press.

SPECT

► Single Photo Emission Computer Tomography

Speech

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Definition

Speech is the external acoustic representation of language. Speech is produced by the coordinated movements of the articulators, including the tongue, lips, jaw, oro- and nasopharynx, larynx, and respiratory system. Speech production results in both acoustic products and perceptual experiences. As described by Hixon, Weismer, and Hoit (2008), speech production may be observed at different levels of observation: (1) the neural includes motor planning and execution as well as sensory input to speech production; (2) the muscular level focuses on mechanical forces and movements of individual muscles; (3) the structural level focuses on the displacements, velocities, and accelerations or decelerations of individual articulatory structures such

as the tongue, lips, or jaw; (4) the observation of aero-mechanical events including air pressures, volumes, and flow as they rapidly change over time; and (5) the acoustic level or, as the authors describe it, "the buzz-like, hiss-like, and pop-like sounds that result from the speaker's valving of the airstream in different ways and at different locations within the speech production apparatus" (p. 3).

Speech disorders may be acquired, as in the case of *dysarthria* or *apraxia* associated with neurological disorders, or developmental, in which case the terms *articulation disorders* or *speech sound disorders* are used.

Cross References

► Articulation Disorder
► Dysarthria
► Phoneme

References and Readings

Hixon, T. J., Weismer, G., & Hoit, J. D. (2008). *Preclinical speech science: Anatomy, physiology, acoustics, perception.* San Diego, CA: Plural.

Speech and Language Disabilities

► Speech/Communication Disabilities

Speech Sound Disorder

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Short Description or Definition

A speech sound disorder is a failure to acquire the speech sounds and/or speech sound rules of a particular language by the expected normative age. Speech sound errors can include substitutions e.g. *wabbit* for *rabbit*, omissions e.g. *ouse* for *house*, distortions, or additions. Problems with speech sound rules include omission of final consonants (*do/dog*, *ca/cat*) or deleting parts of blends in words (*poon/spoon*, *bown/brown*). While some of these sound changes are common for toddlers and early preschoolers, children should master the sounds and basic rules of speech sounds by the age of eight. The effect of a speech sound disorder on speech understandability can range from mildly to totally unintelligible. However, individuals whose sound substitutions or omissions reflect a dialectic variation are not considered to have a speech sound disorder.

Categorization

Speech sound disorders are divided into two categories: articulation disorders and phonological (pattern) disorders. An articulation disorder is associated with a motoric inability to produce a speech sound or sounds. A phonological disorder is viewed as a failure to perceive the difference between speech sounds, resulting in patterns of errors such as final consonant deletion (*ba* for *bat*, *bee* for *bees*), or velar fronting (*tat* for *cat*).

Epidemiology

Prevalence of speech sound disorders in children is higher than in adults. Figures cited for preschoolers are 8–9% with approximately 5% still demonstrating a disorder by first grade. Some preschoolers will outgrow their errors while others will require treatment from a speech-language pathologist to develop understandable speech (see Evaluation).

During the process of speech development, speech sound disorders occur more often in children with:

- Genetic syndromes such as Down syndrome or other syndromes associated with cognitive delays
- Developmental disorders such as autism
- Hearing loss (sensorineural or conductive loss associated with frequent ear infections in early childhood)
- Childhood apraxia of speech
- Neurological disorders such as cerebral palsy
- Orofacial anomalies such as cleft palate

Speech sound disorders may also run in families. However, in some cases no definitive etiological factor will be found.

For school-aged children, speech sound disorders can be a continuation of an earlier phonological or articulation problem or the result of some type of neurological injury. Similarly, in adults, a speech sound disorder can consist of residual errors of an earlier disorder or a new disorder owing to a variety of neurological causes (► Dysarthria and Apraxia for further information on these adult causes of speech sound disorders).

Speech Sound Disorder. Table 1

By age 3:	p m h w b (emerging between ages 1 ½ and 3)
By age 3 ½:	k g d t f y (girls only) (emerging between ages 2 and 3 ½)
By age 4:	y (boys)
By age 5:	s-blends (emerging between ages 3 ½ and 5)
By age 5 ½:	v
By age 6:	sh ch j (girls)
By age 7:	sh ch j (boys) th (as in *th*at)
By age 8:	r s ng l z th (as *in* thumb) zh (as in mea*s*ure)

Speech Sound Disorder. Table 2

Final Consonant Deletion:	2 ½–3
Liquid Gliding (w/r, w/l)	7–8
Velar Fronting (tat for cat, dum for gum)	3–3 ½
Stopping (too/Sue, pan/fan)	3 for f and v
	3 ½ for s, z, sh
	4 ½ for ch, j, *th* sounds

Natural History, Prognostic Factors, Outcomes

A child's acquisition of the speech sounds and rules is a gradual process. Correct articulation can depend not only on motor skills and perceptual development but also the sound make-up and length of a word. Nevertheless, research indicates that the following sounds should be produced correctly by the ages indicated:

- *Vowels*: should all be acquired by age 3 with the exception of the *er* sound in words like *bird* and *hammer*.
- *Consonants*: these represent ages of *mastery*; prior to these ages correct production will vary.

For speech sound rules or patterns, the following age levels have been identified for *loss* of the early patterns. Up to these ages, these patterns are considered normal. For children who do not meet these milestones, testing and possible treatment by a certified speech-language pathologist is required. Some patterns *never* occur in the speech of typically developing children and should always be evaluated: deletion of initial consonants (*ome/home*; *ee/bee*) and backing (*kee/tee*, *gum/dum*). Preschool children with intelligibility problems are at risk for later reading problems. Early intervention is crucial in these cases.

Neuropsychology and Psychology of Disorder

While some speech sound disorders may be associated with neurological damage (see Epidemiology), many are not. The effect of a speech sound disorder on emotional behavior will generally depend on the age of the patient, etiology, and severity of disorder. Children who have limited verbalization or are highly unintelligible speech may refuse to communicate, even nonverbally. Other children may show no emotional response. Still others may develop other ways of communication, including gestural systems (particularly those with childhood apraxia of speech). Children with mild speech sound disorders generally are not emotionally affected by their speech errors.

For adults with speech sound disorders, etiology and age of onset will affect the patient's response to communication difficulties. Adult-onset speech problems are generally associated with neurological damage such as stroke or traumatic brain injury. The degree of emotional response to the problem may depend on extent and location of neurological damage as well as the degree of disruption of communication (► Apraxia and Dysarthria for further information).

Evaluation

Evaluation of speech sound disorders is designed to determine:

1. Existence of a problem
2. Nature of the problem (sounds in error, patterns in error, intelligibility)
3. Possible etiology(ies) of the problem (such as hearing loss, neurological disorder)
4. Probable course of treatment
5. Prognosis

To meet these goals, the following components should be included in an evaluation for speech sound disorders:

1. Case history
2. Hearing screening: to determine adequacy of hearing for speech
3. Oral mechanism evaluation: to determine if speech structures and functions are adequate for speech purposes
4. Phonemic sound-by-position tests and/or phonological tests designed to determine the phonological patterns used
5. Language testing
6. Other tests as appropriate

Treatment

The type of treatment will depend on the type of disorder and pertinent etiological factors. For patients with simple articulation disorders, a traditional, phonetic approach can be successful. For patients with articulation disorders associated with motor weakness, motoric approaches can be useful. For pediatric patients with phonological disorders, a rule-based treatment approach is best, depending on etiology. For children whose etiology is unknown or associated with chronic ear infections, a rule-based treatment approach is recommended. In every case, treatment should be performed by a certified speech-language pathologist with experience in speech sound disorders. Phonological disorders associated with childhood apraxia of speech require an eclectic treatment approach. ► Phonological Disorders and ► Articulation Disorders for more information on treatment.

Cross References

► Apraxia
► Articulation Disorder
► Dysarthria
► Phonological Disorder
► Phonology

References and Readings

American Speech-Language-Hearing Association. (2007). *Speech sound disorders: Articulation and phonological disorders.* www.asha.org/public/speech/disorders/SpeechSoundDisorders.

American Speech-Language-Hearing Association. (2008). *Incidence and prevalence of communication disorders and hearing loss in children* (2008 ed.). www.asha.org/members/research/reports/children.

Speech/Communication Disabilities

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Synonyms

Communication disorders; Speech and language disabilities

Definition

Speech communication disabilities are a broad category that includes disorders of articulation, phonation, fluency, and language.

Speech production involves four primary steps or processes that include respiration, phonation (vibration of the vocal folds due to air pressure that builds as it moves through the larynx), resonance, (modification of the air waves in the pharynx, nasal cavity, or oral cavity that were produced as the air moved through the vocal cords), and articulation (movements of the speech structures in order to produce the speech sounds). Disruption at any level is considered a speech defect.

The articulators include the pharynx, soft palate, hard palate, mandible, teeth, tongue, lips, and cheeks. Articulation disorders can include single articulation errors, such as substitution, omission, or addition of a sound. Or, it can refer to multiple errors that have a pattern in production or location, which is often considered a phonological disorder. Dysarthria is an articulation disorder that is related to poor muscle control. Because of disease or injury, the individual may be unable to produce speech sounds with any precision. Their slurred and weak speech is considered dysarthric. Speech apraxia is a motor speech disorder in which there are inconsistent speech errors. The individual has difficulty with volitional production of sounds and words.

Phonation is the process that occurs at the level of the larynx and involves vibration of the vocal cords. Voice disorders can be biological, such as growths on the vocal folds, or functional, due to psychological factors. The most prevalent voice problem can develop as a result of abusive vocal practices, such as coughing or prolonged yelling.

Fluency includes the rapid production of speech in a smooth and fluent manner. Stuttering is "disfluent" speech and is often characterized by the production of repetitions or prolongation of a sound or syllable. This disorder can also involve "blocks" as when speech is halted and the individual struggles to produce a word or sound. Stuttering can also involve "circumlocution." This is an attempt by the speaker to disguise his or her stuttering. He or she may rearrange the sentence or use unusual synonyms, or may decline to participate in a conversation. More severe stuttering may include secondary characteristics, such as tics or unusual behaviors that "start" his or her speech. If the disorder is untreated, secondary characteristics can develop into a behavioral chain that is very distracting to the speaker and listener. Cluttering is an associated fluency disorder. It is defined as rapid and disorganized speech combined

with disfluency and disorganized thought and disorganized language.

Language includes receptive and expressive language skills. Children may experience language delay if they fail to acquire language skills in a prescribed time frame. A language based learning disorder may develop in children with early language difficulties. This is characterized by difficulty in learning to read, write, and spell but may manifest in younger children as listening or speaking difficulties.

Acquired language disorders can be classified as forms of aphasia and may result from insult to the part of the brain that is responsible for language. Aphasia can involve disruption of speaking, listening, reading, or writing skills.

Pragmatic language is social language, such as greetings, maintaining eye contact with a listener, taking turns in a conversation, etc. A disorder with pragmatics is often diagnosed when the speaker lacks these interpersonal communication skills and lacks awareness of the skills.

Categorization

The American Speech Language Hearing Association groups communication disorders into disorders of children and adult onset disorders. There can be overlap, as when a childhood disorder continues to afflict the individual as they get older. Disorders of children include articulation, speech apraxia, phonological processing disorders, voice, fluency, and language disorders. Adult speech and language disorders include apraxia, dysarthria, stuttering, voice, and aphasia.

Epidemiology

The incidence of stuttering is reported to be 1% of the United States population with a 3:1 male to female ratio (www.asha.org). The lifetime prevalence of voice disorders is reported to be 30% or approximately 23 million US workers (Roy, Merrill, Gray, & Smith, 2005). Approximately one million Americans are reported to have aphasia (www.nidcd.htm).

Evaluation

Evaluation of speech articulation disorders first involves examination of the oral mechanism to determine whether the muscles and structures of the oral cavity are working properly. In addition, the speech and language pathologist will conduct an articulation evaluation to identify the type of error, such as distortion, omission or substitution. There are several standardized instruments that can quantify the degree of disorder.

Voice disorders are evaluated through perceptual and instrumental methods. When a voice disorder is suspected, a physician should always be involved because of the possibility of serious health concerns (e.g., papilloma, tumor, nodule). The physician will often use some sort of instrument (laryngeal mirror, for example) to examine the larynx and vocal folds. A perceptual evaluation is conducted by the speech and language pathologist who will determine the perceptual features of the voice (pitch, volume, and quality – hoarseness, harshness, breathiness, etc.).

Fluency is evaluated by requesting the individual to read or speak, sometimes using stress producing tasks, such as speaking on the phone. The number and types of repetitions are counted and quantified. An evaluation of fluency may also involve a thorough interview and the assessment of language skills. Children often experience a normal period of disfluency from the ages of two to four. A trained speech and language pathologist can identify risk factors, such as a family history of stuttering, prolonged period of stuttering, and whether there is concomitant speech and language disorder.

The evaluation of language involves measuring the individual's comprehension (receptive language) and expressive language in areas of vocabulary, morphology, and syntax. A thorough language evaluation will also investigate the individual's pragmatic language capabilities (social language). The speech/language pathologist will first determine whether a significant problem exists. Then, the SLP will identify how to address the problem using evaluation information.

An evaluation for aphasia will often include examination by a number of medically related professionals, such as speech/language pathologists, doctors, nurses, neuropsychologists, occupational therapists, physical therapists, and social workers. The communication domains (listening, speaking, reading, writing, and social language) will be evaluated informally or formally, using standardized test instruments.

Treatment

The treatment of articulation disorders involves learning to recognize when the sound is produced correctly

(discrimination), followed by production of the sound in isolation, in words, in short utterances, and finally by generalizing the sound to connected speech.

Fluency therapy is often based on behavioral interventions. The individual is taught to identify, monitor, and modify their rate of speech, breath control, or the physicality of their speech (softer speech contacts). They learn these strategies in single words, phrases, sentences, and eventually in lengthier connected speech.

The treatment of voice disorders involves identifying the nature of the problem first. If there is a medical problem, this must be remediated before any therapy. Laryngectomee patients will be retrained to speak using prosthetic speaking devices. Individuals with neurological disorders will be taught compensatory strategies to improve breath control and articulation. If there is an issue with abusive vocal practices, the goal will be to reestablish healthy vocal habits (Colton, Casper, & Leonard, 2005).

The treatment of language disorders with children should include parent training, especially with young children. Service delivery models can include individualized treatment, small group treatment, whole class lessons, or collaboration with parents or teachers. The type of intervention will depend on the student's needs (Roseberry-McKibbon & Hedge, 2000).

The treatment of aphasia can involve specialized programs, collaboration with other professionals, or informal intervention. The type of therapy is dependent on the type and severity of the individual's needs with a goal of establishing effective communication strategies.

Cross References

- ▶ Aphasia
- ▶ Apraxia of Speech
- ▶ Articulation Disorders
- ▶ Dysarthria
- ▶ Language
- ▶ Learning Disabilities

References and Readings

Colton, R. H., Casper, J. K., & Leonard, R. (2005). Understanding voice problems: A physiological perspective for diagnosis and treatment. Lippincott, Williams, & Wilkins: NY.

National Institute on Deafness & Other Communication Disorders. *Aphasia.* Retrieved May 8, 2007, from http://www.nidcd.nih.gov/health/voice/aphasia/htm. NIH Pub. No. 97–4257.

Roseberry-McKibbon, C., & Hegde, M. N. (2000). *An advanced review of speech-language pathology.* San Antonio, TX: Pro-Ed.

Roy, N., Merrill, R. N., Gray, S. D., & Smith, E. M. (2005). Voice disorders in the general population: Prevalence risk factors, and occupational impact. *Laryngoscope, 115*(11), 1988–1995.

Speech-Language Intervention

▶ Speech-Language Therapy

Speech-Language Pathology

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Synonyms

Speech-language therapy

Definition

Speech-language pathology refers to both clinical specialty in and the study of the science, development, and disorders of speech, language, and communication, specifically in the areas of articulation, fluency, voice and resonance, receptive and expressive language, and hearing. It includes the impact of these processes on speech and language, swallowing, cognitive aspects of communication, social aspects of communication, and communication modalities (American Speech-Language-Hearing Association, 2003).

Current Knowledge

Certificate of Clinical Competence

The American Speech-Language-Hearing Association (ASHA) is the professional, scientific, and credentialing association for speech-language pathologists. Individuals who have met academic and clinical criteria (i.e. a master's degree in speech-language pathology or its equivalent and a minimum of 400 h of supervised clinical practicum)

and demonstrate knowledge and skills in the profession are granted the ASHA CCC-SLP (Certificate of Clinical Competence in Speech-Language Pathology; American Speech-Language-Hearing Association, 2005a).

Scope of Practice

Speech-language pathology assessment and intervention services are delivered within the *Scope of Practice in Speech-Language Pathology* (American Speech-Language-Hearing Association, 2007) which includes "a statement of purpose, a framework for research and clinical practice, qualifications of the speech-language pathologist, professional roles and activities, and practice settings." The ASHA Practice Policy Documents provide detailed information on topics such as Preferred Practice Patterns and Clinical Guidelines, and also technical reports about communication disorders (ASHA, 2008).

Evidence-Based Practice

Clinical application of diagnostic and intervention methods should be guided by principles of evidence-based practice "in which current, high-quality research evidence is integrated with practitioner expertise and client preferences and values into the process of making clinical decisions" (American Speech-Language-Hearing Association, 2005b). ASHA policy requires that clinical activities are conducted in a manner that considers the impact of culture and linguistic background and uses the best available evidence for practice to ensure optimal outcomes for persons with speech, language, communication, and/or swallowing disorders or differences.

Cross References

- ▶ American Congress of Rehabilitation Medicine
- ▶ American Psychological Association (Division 40)
- ▶ American Speech-Language-Hearing Association (ASHA)
- ▶ Apraxia of Speech
- ▶ Articulation
- ▶ Articulation Disorders
- ▶ Cognitive-Communication Disorder
- ▶ Communication Ability
- ▶ Dysphagia
- ▶ Dysphonia
- ▶ Language
- ▶ National Institute of Neurological and Communication Disorders and Stroke
- ▶ National Institute on Disability and Rehabilitation Research
- ▶ National Institutes of Health
- ▶ Phonological Disorders
- ▶ Phonology
- ▶ Pragmatic Communication
- ▶ Speech
- ▶ Speech/Communication Disabilities
- ▶ Speech-Language Therapy

References and Readings

American Speech-Language-Hearing Association (2003). *Knowledge and skills acquisition (Kasa) summary form for certification in speech-language pathology.* http://www.asha.org/NR/rdonlyres/3B9B6A8F-4AA7-4203-8447-BCFD754D64F9/0/KASASummaryFormSLP.pdf

American Speech-Language-Hearing Association. (2005a). *Membership and certification handbook of the American- speech-language-hearing association.* http://www.asha.org/about/membership-certification/handbooks/slp/slp_standards.htm

American Speech-Language-Hearing Association. (2005b). *Evidence-based practice in communication disorders* [Position Statement]. Available from www.asha.org/policy

American Speech-Language-Hearing Association. (2007). *Scope of practice in speech-language pathology* [Scope of Practice]. Available from www.asha.org/policy

ASHA (2008). *American speech-language-hearing association practice policy documents.* http://www.asha.org/policy/about.htm

Speech-Language Therapy

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S

Synonyms

Speech-language intervention

Definition

Speech-language therapy is the variety of processes employed by speech-language pathologists who work with the full range of human communication and its disorders. Treatment areas include speech, language, cognitive communication, or swallowing disorders in

individuals of all ages, from infants to the elderly. Speech-language therapy includes multiple techniques that induce change in communication skills of patients. Therapy can be impairment-based, focusing on the management of contingent relations between antecedents, responses, and consequences of the communication attempt, or participation-based, focusing on manipulating pragmatic communication interactions among the speaker, listener, and environment. For example, if working from an impairment-based model, the therapy would be directed toward helping the individual formulate better-sounding words and sentences or increasing reading or writing skills. If working from a participation-based model, the therapy would include significant communication partners in real environments and would focus on helping the client be understood in any environment by any communication means possible. Compensatory strategies such as assistive technology could also be employed.

Cross References

► Speech-Language Pathology

References and Readings

Roth, F., & Worthington, C. (2005). *Treatment resource manual for speech-language pathology.* New York: Cengage/Thompson Learning.

Speechlessness

► Anarthria

Speed–Accuracy Tradeoff

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Definition

The complex relationship between an individual's willingness to respond slowly and make relatively fewer errors compared to their willingness to respond quickly and make relatively more errors is described as the speed–accuracy tradeoff.

Current Knowledge

In experimental studies of human performance, both the speed at which an individual completes a task and the accuracy of their response rates are important considerations in methodological study design as well as in the interpretation of findings. Ideally, an individual attempts to maximize performance on both factors. In some situations, however, an individual may increase his or her response time at the cost of reducing the accuracy of his or her responses, while in other situations an individual may find it necessary to slow his or her response time in order to increase his or her overall accuracy level (Proctor & Vu, 2003). In experimental research, illustrative schematics of speed–accuracy tradeoff data reveal consistent relationships across many different tasks. Following the basic principal that it takes time for an individual to process information accurately, consideration of the speed–accuracy tradeoff is often undertaken to determine the minimum amount of time that is required to produce a correct response on a given task (Bullinaria, 2000). This information has critical implications for overall experimental design as well as interpretation of results.

The implications of the speed–accuracy tradeoff for clinical neuropsychological assessment are implicit in test procedures which are timed or have time limits. When the patient is aware of the time component of the scoring system, he or she may attempt to maximize either the speed or the accuracy. Careful behavioral observation as well as a debriefing interview process may help elucidate the patient's strategy, thereby aiding in the interpretation of the test results.

References and Readings

Bullinaria, J. A. (2000). Free gifts from connectionist modeling. In R. Baddeley, P. J. B. Hancock, & P. Foldiak (Eds.), *Information theory and the brain*(pp. 221–240). Cambridge: Cambridge University Press.

Proctor, R. W., & Vu, K.-P. L. (2003). Action selection. In A. F. Healy & R. W. Proctor (Eds.), *Experimental psychology* (pp. 293–316). Vol. 4 in I. B. Weiner (Editor-in-Chief) Handbook of psychology. New York: Wiley.

Sperry, Roger Wolcott (1913–1994)

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Major Appointments

- National Research Council Fellow, Harvard University (1941–1942)
- Biology Research Fellow, Harvard University, Yerkes Laboratories of Primate Biology (1942–1946)
- Assistant Professor, Department of Anatomy, University of Chicago (1946–1952)
- Associate Professor of Psychology, University of Chicago (1952–1953)
- Section Chief, Neurological Diseases and Blindness, National Institutes of Health (1952–1953)
- Hixon Professor of Psychobiology, California Institute of Technology (1954–1984)
- Fellowship Committee, National Science Foundation (1963–1964)
- Experimental Psychology Study Section, National Institutes of Health (1966–1970); Chairman of Section (1969–1970)
- Corporate Visiting Committee for Psychology, Massachusetts Institute of Technology (1969–1976)
- Board of Trustees Professor Emeritus, California Institute of Technology (1984–1994)

Major Honors and Awards

- Amos C. Miller Scholarship from Oberlin College (1931–1935)
- National Research Council Fellowship (1941–1942)
- Distinguished Alumni Citation, Oberlin College (1954)
- Elected National Academy of Sciences (1960)
- Elected American Academy of Arts and Sciences (1963)
- Howard Crosby Warren Medal, Society of Experimental Psychologists (1969)
- Distinguished Scientific Contribution Award, American Psychological Association (1971)
- Corecipient, William Thomson Wakeman Research Award, National Paraplegia Foundation (1972)
- California Scientist of the Year Award (1972)
- Honorary Doctor of Science Degree, Cambridge University (1972)
- Passano Award in Medical Science (1973)
- Elected Honorary Member American Neurological Association (1974)
- Co-recipient Claude Bernard Science journalism Award (1975)
- Karl Lashley Award of American Philosophical Society (1976)
- Elected Foreign Member of Royal Society (1976)
- Honorary Doctor of Science Degree, University of Chicago (1976)
- Elected Member Pontifical Academy of Sciences (1978)
- Honorary Doctor of Science Degree, Kenyon College (1979)
- Wolf Prize in Medicine (1979)
- Ralf Gerard Award of the Society for Neuroscience (1979)
- International Visual Literacy Association Special Award for 1979 (1979)
- Albert Lasker Medical Research Award (1979)
- Honorary Doctor of Science Degree, Rockefeller University (1980)
- American Academy of Achievement Golden Plate Award (1980)
- Shared the Nobel Prize in Medicine/Physiology (1981)
- Honorary Doctor of Science Degree, Oberlin College (1982)
- California State Psychological Association Award for Distinguished Scientific Achievements in Psychology
- Realia Award of the Institute for Advanced Philosophic Research (1986)
- Mentor Society Award (1987)
- Elected Foreign Member USSR Academy of Sciences (1988)
- National Medal of Science (1989)
- Elected William James Fellow, American Psychological Society (1990)
- Distinguished Centennial Address Award, American Psychological Association (1991)
- Editorial advisory boards for the following journals: Experimental Neurology, Experimental Brain Research,
- Neuropsychologia, The International Journal of Neuroscience, Behavioral Biology, Zygon, Perspectives in Biology and Medicine, Synapse, and Journal of Neural Transplantation

Landmark Clinical, Scientific, and Professional Contributions

- Famed as one of the few psychologists to receive the Nobel Prize, Sperry produced groundbreaking work in the fields of neuroscience, neurophilosophy, neuropsychology, philosophy, and psychology. Even laypeople have heard of cerebral hemispheric differences as pioneered by Sperry's discoveries in split-brain patients. Heralded as one of the intellectual giants of the century, Sperry's contributions are best described by what Sperry himself termed "the four turnarounds" of his lifelong scientific career (Puente, 2002): (1) Nerve Regeneration Research (1937–1975), (2) Studies Involving Equipotentiality (1952–1955), (3) corpus callosum/split-brain (1950–1985), and (4) consciousness/values (1962–1994). Throughout each phase of his research, Sperry sought to address the two questions that emerged during his first psychology class with Raymond Stetson at Oberlin: (1) Where does behavior come from? (2) What is consciousness?

Nerve Regeneration Research (1937–1975)

Sperry's earliest investigations sought to address the nature versus nurture question and determine how brain nerves form appropriate connections. Under Paul Weiss, his doctoral mentor and a well-known zoologist, Sperry began his studies through muscle transplantation in rats, later expanding his research to examine nerve crossing and the sensory functions of olfaction and vision. Animal research first started with rats and extended to amphibians, fish, and monkeys. By the 1940s, Sperry had concluded that nerve fibers were not interchangeable (Puente, 2002).

Through his rigorous investigations, Sperry became convinced by the early 1950s that motor and sensory functions across multiple animals were limited in scope (Puente, 2002). Sperry's doctoral dissertation, "Functional results of crossing nerves and transposing muscles in the fore and hind limbs of the rat", offered an alternate view to Weiss's interpretation, and actually disproved much of Weiss's research. He also spent considerable time investigating the optic nerves of newts. After severing optic nerves, manipulating the eyeballs' orientation, and then replacing the eyeballs in their original position, he found that the nerves reestablished original connections (Wade, 1995). His 20-year devotion to this research endeavor successfully challenged Weiss's ideas and highlighted the power of nature over nurture (Puente, 2002).

Studies Involving Equipotentiality (1952–1955)

During his postdoctoral years at Harvard and in the Yerkes primate laboratories under the supervision of Karl Lashley, Sperry devised experiments to challenge Lashley's theory of equipotentiality during his "second turnaround." Equipotentiality, the theory that brain lesions have nonspecific effects, was consistent with the common belief at that time that lesion location is unrelated to the nature of the resulting behavioral deficit (Puente, 2002). As such, Sperry sought to explore the concept of neuronal integration, particularly function. He investigated how electrical fields played a crucial role in neocortical processes. He placed various insulating (e.g., mica plates or subpial scarring) or short-circuiting elements (e.g., tantalum pins) in the cortex and subsequently examined the function of the system directly affected by the stimulation (Doty, 1994). As such, he systematically tested Lashley's theory and after approximately 3 to 4 years, Sperry concluded that synapse formation is well-organized, prearranged, and highly regimented (Finger, 2000). His research findings laid the foundation of his assertion that chemicals are instrumental in attracting axons to specified targets (Finger, 2000).

Split-Brain Studies (1950–1985)

Sperry decided to examine the enigma of the corpus callosum which was sweeping the scientific field in the 1950s. The medical arena had asserted that the complete surgical cutting of the corpus callosum (also known as the largest bundle of brain connections) caused no definite symptoms (Adler, 1990). He started his research by examining in cats and monkeys the transfer of sensory information across this bundle of fibers connecting the hemispheres. Through surgically splitting the corpus callosum and optic nerve in adult cats, he found that visual tasks could be presented and actually learned by a single hemisphere by closing one of the animal's eyes so that, when tested, the other hemisphere had no knowledge of the visual discrimination learned by the first eye/hemisphere (Puente, 2002). By conducting various experiments, he had learned that a fully intact corpus callosum was required to effectively transfer information across hemispheres. He presented his early findings from his corpus callosum experiments for the first time to a group of psychologists at the American Psychological Association 1960 convention. In the journal *Science*, he published an article entitled "Cerebral Organization

and Behaviour" purporting how separate hemispheres actually behave like individual brains.

Joseph Bogen, a neurosurgeon who had followed Sperry's initial work on sectioning of the corpus callosum in animals, believed such experiments could likely be applied to humans. At Caltech, Bogen, Peter Vogel, another neurosurgeon, Sperry, and a few graduate students initiated a series of experiments to determine the effects of callosal sectioning on neurocognitive functions. In collaboration with Sperry, Bogen performed his first commissurotomy on a seemingly hopeless case of post-traumatic intractable epilepsy in a war veteran. This landmark case marked the first extension of Sperry's split-brain effect from animals to humans. A promising graduate student in Sperry's laboratory, Michael Gazzaniga, played a key role in the research program designed to examine the specificity of the cerebral hemispheres which was conducted over the following 20 years.

Sperry and his colleagues at Caltech made numerous groundbreaking discoveries over the next few years. They defined the role of the corpus callosum as providing a means of communication between and a venue for integration of the knowledge acquired by each of the two cerebral hemispheres. He found that postsurgically, cognition and perception were modified such that one hemisphere was completely oblivious to the information processing of the other hemisphere. Although the widely accepted view was that the left hemisphere was dominant, he sought to empirically and systematically investigate hemispheric specialization, especially in the right hemisphere (Wade, 1995). He and his colleagues learned that although the left hemisphere is more analytical than the right, the right hemisphere was actually superior to the left in many ways, particularly in intuitive information processing, appreciation of a gestalt, and comprehension of emotional behavior (Puente, 2002). In the late 1960s, he and his colleagues started publishing technical papers on his split-brain findings. His most significant contribution to science was in his meticulous approach to understanding hemispheric functioning and the function of the corpus callosum across three species (cats, monkeys, and humans) which lead to his receipt of the Nobel Prize in 1981. Overall, his split-brain research advanced two cutting-edge notions: (1) the corpus callosum not only physically connects but also transmits information between the two cerebral hemispheres and (2) the right hemisphere has a consciousness of its own and is superior to the left hemisphere in a few specific ways (Puente, 2002).

Consciousness Research (1962–1994)

Sperry's final turnaround focused on consciousness and values systems. He investigated each hemisphere's unique contribution to consciousness and the superiority of the concerted effort of both hemispheres working in unison for optimal purposeful and goal-directed behavior (Puente, 2002). The interplay between the emergence of consciousness from brain activity and consciousness's influence and control on the brain's activity was also a focus of his research during this time. His second focus was directed toward two value-driven questions: what thoughts ought to arise in consciousness and which values can be deemed the most useful? (Puente, 2002). Sperry proposed that application of scientific, empirically driven methodology to the value system may in turn augment the development of consciousness. He also believed that the values deemed most useful to human beings would ultimately be determined by nature and time (Puente, 2002). Throughout this "turnaround" and well into the final decades of his life, Sperry addressed the philosophical questions of ethics and morality, commenting "In my scheme, values are perceived to be organized in a complex of nested manifolds involving value hierarchies within hierarchies" (Wade, 1995, p. 195).

Sperry's interest in the brain was well-grounded from the start, and he sincerely believed that a more accurate understanding of brain–behavior relationships was a determining factor in the development of what he called the "consciousness revolution" (Puente, 2002). Throughout his 65-year commitment to neuroscience, Sperry published over 300 articles in the most rigorous and prestigious scientific journals. His research was funded continuously for almost 50 consecutive years. His work has been translated into several languages including Spanish, Russian, Chinese, and Japanese. During his career, he was influenced by his interactions with numerous well-known scientists including: Brenda Milner, Robert Galambos, William Burbanck, Jerry Kollros, and Kao Liang Chow (Puente, 2002).

Short Biography

Roger Wolcott Sperry was born in Hartford, Connecticut on August 20, 1913 as the first son of Florence Kraemer Sperry, a banker and Francis Bushnell, who was trained in business school. He attended local public schools in the small suburb of Elmwood. He grew up in a family known for valuing education. In fact, Roger was first introduced

to the William James's (1890) *Principles of Psychology* when his father brought the book home from the public library (Puente, 2002). During grade school, Roger excelled in academics, fostered a love of biology, truly enjoyed sports, and in his spare time collected and raised large American moths. He gathered wild pets and actively searched out dead animals for dissection well into his junior high school years (Puente, 2002). Unfortunately, the family's income drastically changed when, at age 11, his father died. Consequently, his mother obtained a position as a high school assistant principal and Roger and his younger brother, Russell Loomis, moved to East Hartford. Roger attended William Hall High School in West Hartford, Connecticut and was captain of a few of his high school sports teams.

When applying to colleges, Roger listed athletic coaching as his primary interest and medical research as secondary. Roger attended Oberlin College in Ohio on a 4-year Amos C. Miller Scholarship. His brother also attended Oberlin and chose a career in chemistry. Upon arrival at Oberlin, Roger continued his love of sports and was active in basketball, baseball, and track. Academically, he chose English as his focus and obtained his Bachelor of Arts in English in 1935. Near the end of his undergraduate studies, his interests started shifting toward psychology. In fact, he remained at Oberlin for 2 additional years, completing his Master's degree in psychology in 1937 under the mentorship of Professor R. H. Stetson. Stetson particularly impressed Roger in that he obtained his Ph.D. with William James. Puente (2002) highlighted the impact that Stetson had on Roger Sperry's career in the field. It is known that Roger's notes from his first class with Stetson "served as a foundation" for his entire future research program. Sperry left his papers to Oberlin College upon his death. Following his master's degree work, he wanted to further his training and decided to spend time at the University of Chicago with Paul Weiss in the Department of Zoology. He received his Ph.D. in zoology in 1941 from the University of Chicago. He then completed a year of postdoctoral research as a National Research Council Fellow at Harvard University where Professor Karl S. Lashley served as his mentor.

At age 36 on December 28, 1949 Roger Sperry married Norma Gay Deupree. Unfortunately, that same year, a routine chest x-ray revealed evidence of his tuberculosis. He and his new bride consequently spent winters in Bimini and summers at Saranac Lake in the Adirondack Mountains. Fortunately, he was offered a position of associate professorship at California Institute of Technology in Pasadena. He quickly accepted and found that his health dramatically improved once away from the windy city.

Finger (2000) noted that Sperry was a Renaissance man with formal training across various disciplines (English literature, experimental psychology, and zoology) and a plethora of hobbies. He enjoyed fishing, boating, snorkeling, camping, collecting fossils, and actively exploring the arts (drawing, watercolor, ceramic, and sculptures).

Sperry and his wife had two children: a son, Glenn Tad Sperry (born October 13, 1953) and a daughter Jan Hope Sperry (born August 18, 1963).

In the early 1980s, Sperry's speech was starting to diminish secondary to a form of lateral sclerosis/Lou Gehrig's disease. By 1980, he stopped lecturing due to speech impairments (e.g., slowed and slurred speech) (Puente, 2002). In 1984, at age 71, he retired from Caltech (Finger, 2000). Ten years later, at age 80, on April 17, 1994, in Pasadena, California, he died of cardiac arrest. It was said that he had been suffering from loss of motor control for close to 3 decades. Colleagues have said that he worked until his death, revising a paper during his final days in the hospital. Finger (2000) noted that upon his death, Sperry was eulogized as a gentleman of "strong personal convictions and exceptional clarity of thought (p. 300)" who had a gift for asking thought-provoking questions, designing methodologically robust experiments, and always maintaining a keen focus on the overarching goal of exploration into the workings of the mind.

Cross References

- ▶ Corpus Callosum
- ▶ Hemispheric Specialization
- ▶ Gazzaniga, M. S. (1939–)
- ▶ Split Brain

References and Readings

Adler, T. (1990). Sperry wins science medal. *Monitor*, 5–6.

Anonymous (1994). In Memoriam: Roger W. Sperry. *Monitor* of the American Psychological Association, June 1994.

Doty, R. (July/August 1994). Obituary: Physiological psychologist Roger Wolcott Sperry (1913–1994). *American Psychological Society Observer of the American Psychological Society*, 34–35.

Finger, S. (2000). Roger W. Sperry and Rita Levi-Montalcini: From neural growth to "split brains". In *Minds behind the brain: A history of the pioneers and their discoveries*. New York, NY: Oxford University Press.

Miller, J. G. (1994). Roger Wolcott Sperry. *Behavioral Science, 39*(4), 265–268.

Mook, D. (2004). Roger Sperry and the bisected brain. Chapter in *Classic experiments in psychology*. Westport, CT: Greenwood Press.
Puente, A. E. (November 1995). Obituary: Roger Wolcott Sperry (1913–1994). *American Psychologist*, *50*(11), 940–941.
Puente, A. E. (2002). Roger W. Sperry: From neuro-science to neuro-philosophy. In A. Y. Stringer, E. L. Cooley, & A-L. Christensen (Eds.), *Pathways to prominence in neuropsychology: Reflections of twentieth-century pioneers* (pp. 63–75). New York, NY: Taylor and Francis Books, Inc.
Wade, N. (1995). Split brain: Roger Walcott Sperry. In: *Psychologists in word and image* (pp. 194–195). Cambridge, MA: Massachusetts Institute of Technology.
www.rogersperry.info

Spina Bifida

► Myelomeningocele

Spinal Cord

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Synonyms

Spinal neuraxis

Definition

The spinal cord is the caudal continuation of the brain stem medulla. It is a component of the central nervous system that functions to relay information between the brain and periphery.

Current Knowledge

Anatomy

The spinal cord is a thin tubular neural structure that weighs 30–35 g and is 42–45 cm long in the human adult (Mccormick & Stein, 1990). It is invested in meninges which is continuous with the meninges of the brain and is contained within the spinal canal of the vertebral column. In the adult, the caudal termination of the spinal cord is at the lower border of the first lumbar vertebrae and is termed the conus medullaris. A thin fibrous extension of pia mater from the conus forms the filum terminale which becomes enveloped in dura and extends to the posterior surface of the coccyx as the coccygeal ligament. There is a large bundle of lumbosacral roots emerging from the conus that surrounds the filum, known as the cauda equina. The spinal cord is traditionally divided into 31 segments: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal. Dorsal and ventral root fibers emerge from the spinal cord segments and exit the spinal canal as the spinal nerves via the intervertebral foramen. Dorsal root fibers are absent in the coccygeal and first cervical segments. There are two enlargements of the cord: cervical and lumbosacral, corresponding to the levels which give rise to the nerve roots that form the brachial plexus and the lumbosacral plexus.

The vascular supply of the spinal cord is primarily from branches of the vertebral artery and multiple radicular arteries. The anterior spinal artery originates from descending branches of the intracranial vertebral arteries and descends in the anterior medial sulcus. The continuity of the vessel is dependent upon anastomoses with numerous segmental vessels at various levels (Gillilan, 1958). This artery supplies the anterior two thirds of the cord. The paired posterior spinal arteries descend on the posterior surface of the cord just medial to the dorsal roots. These arteries supply the posterior third of the cord. Radicular arteries pass through the intervertebral foramina providing anastomotic connections with either the posterior or anterior spinal arteries. The radicular arteries arise from numerous segmental vessels throughout the length of the cord. One important radicular artery of note is the artery of Adamkiewicz which most frequently originates on the left side from vertebral segments T9–12. Infarct of this vessel can have devastating neurologic consequences.

Ascending and Descending Pathways

In transverse section, the spinal cord consists of an H-shaped central gray matter enveloped in a mantle of white matter. The two posterior portions of the H are termed the dorsal horns and the two anterior portions are termed the ventral horns. The gray matter is composed of numerous neuron cell bodies while the white matter is composed of bundles of ascending and descending myelinated fibers originating from either brain or periphery. A full discussion of each of these pathways is beyond the

scope of this text but some of the major pathways are described.

Anterolateral System

This ascending pathway mediates the sensations of pain, temperature, itching, and simple touch. Afferent fibers from the Dorsal Root Ganglia (DRG) synapse in nuclei are located in the dorsal horns. Most of the axons projecting from these neurons then project to the contralateral anterior and lateral funiculi by crossing the midline through the ventral white commissure. These fibers then ascend and synapse in the Ventral Posterolateral (VPL) nucleus of the thalamus. A unilateral cord lesion of this pathway would therefore result in contralateral loss of pain and temperature sensation one to two segments below the level of the lesion.

Spinal Lemniscal System

This ascending pathway carries information regarding proprioception, vibration sense and tactile discrimination. Afferent fibers from the DRG enter the spinal cord and ascend in the posterior funiculi without synapsing in the spinal cord. These fibers synapse in the dorsal column nuclei of the lower medulla. Fibers from these nuclei then send projections to the contralateral medial lemniscus and ascend to synapse in the VPL nucleus of the thalamus. A unilateral cord lesion of this pathway would therefore result in ipsilateral loss of proprioception, vibration sense, and tactile discrimination.

Corticospinal Tract

This descending pathway controls skilled motor movements of the limbs. Efferent fibers originate in various nuclei of the cortex (Brodmann's areas 4, 6 and 3, 1, 2) and pass through the posterior limb of the internal capsule. Fibers travel through the brain stem and the majority of fibers cross in the lower medulla. Fibers descend in the ventral and lateral corticospinal tracts in the ventral and lateral funiculi. These fibers then synapse in the ventral horn.

Neoplastic Lesions of the Spinal Cord

In describing pathologic lesions of the spinal cord, especially neoplasms, it is useful to categorize them by anatomic location with respect to the meninges. Neoplastic lesions can be (a) extradural, (b) intradural extramedullary, or (c) intradural intramedullary. A brief description of these entities is outlined. Clinical presentation of these lesions is highly variable and is dependent on the size, location, and growth rate of these tumors.

Extradural

The majority of extradural spinal cord tumors arise in the vertebral bodies or epidural tissues and are metastatic. Most cause some element of bony vertebral body destruction. The most common lesions result from primary cancers in the lung, breast, and prostate. In men, prostate cancer is the most common and in women, breast cancer is the most common.

Intradural Extramedullary

The majority of tumors in this compartment are schwannomas, neurofibromas, and meningiomas. Other rarer tumors are ependymomas and metastases. Ependymomas of the filum terminale are usually of the myxopapillary type and represent 50% of all spinal ependymomas (Barone & Elvidge, 1970). Complete surgical resection of myxopapillary ependymomas is usually curative.

Intradural Intramedullary

These tumors are relatively rare and are predominately tumors of glial origin. In adults, ependymomas are the most common followed by astrocytomas and hemangioblastomas (Miller, 2000). Intramedullary tumors may be found over the entire length of the spinal cord. Patients with von Recklinghausen's neurofibromatosis are predisposed to the development of these tumors. Similarly, patients with von Hippel–Lindau syndrome are predisposed to having multiple intramedullary hemangioblastomas.

Cross References

- ▶ Gray Matter
- ▶ Meninges
- ▶ Spinal Cord Injury
- ▶ White Matter

References and Readings

Barone, B., & Elvidge, A. (1970). Ependymomas: A clinical survey. *Journal of Neurosurgery, 33*, 428–438.

Gilman, S., & Newman, S. (2003). *Essentials of clinical neuroanatomy and neurophysiology* (10th ed.). Philadelphia, PA: F.A. Davis.

Gonzalez, L. F., & Dickman, C. A. (2006). *Spinal cord and spinal column tumors.* New York: Thieme.

Gillilan, L. A. (1958). The arterial blood supply of the human spinal cord. *Journal of Comparative Neurology, 110*, 75–103.

McCormick, P. C., & Stein, B. M. (1990). Functional anatomy of the spinal cord and related structures. *Neurosurgery Clinics of North America, 1*, 469–489.
Miller, D. C. (2000). Surgical pathology of intramedullary spinal cord neoplasms. *Journal of Neurooncology, 47*, 189–194.
Parent, A. (1996). *Carpenter's human neuroanatomy.* Media, PA: Williams & Wilkins.

Spinal Cord Dysfunction

► Spinal Cord Injury

Spinal Cord Independence Measure (SCIM)

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Brief Description

The Spinal Cord Independence Measure (SCIM) is a comprehensive functional assessment and rating scale for patients with spinal cord lesions (SCL), created in 1994.

SCIM current version (SCIM III) covers 19 activities of daily living grouped into four areas of function (subscales): Self-care (scored 0–20), Respiration and Sphincter Management (0–40), Mobility in Room and Toilet (0–10), and Mobility Indoors and Outdoors (0–30) (Catz et al., 2007; Itzkovich et al., 2007). The domains of each subscale were determined by a consensus of experienced professionals to enable the unidimensionality of SCIM subdivisions. Unidimensionality was verified by Rasch and factor analyses (Catz et al., 2007).

The SCIM scoring is based on the observation of the patient performing the various tasks and assigning the most compatible score from among those that appear in the SCIM evaluation sheet. Scoring criteria are listed next to each task. For tasks such as sphincter management, which may not be observable, the rater is advised to consult the patient's records or a professional who observed the patient performing the task. Each subscale is best scored by someone experienced in assessment and treatment in the domain of the subscale. But, the SCIM can also be scored by a single team member or by interview, which decreases somewhat the accuracy of the score (Catz et al., 2002; Itzkovich et al., 2003).

Historical Background

Several valid rating scales are in use for the functional assessment of SCL patients (Catz & Itzkovich, 2003; Dodds, Martin, Stolov, & Deyo, 1993; Morganti, Scivoletto, Ditunno, Ditunno, & Molinari, 2005). Some, however, measure the burden of care rather than functional achievements according to their importance for the SCL patient. Certain scales measure only some daily activities, use unweighted scoring of items, have relatively low sensitivity to change in relevant functions, or require a manual and extensive training for proper use. The SCIM was designed to overcome the shortcomings of previous scales. The first version of SCIM was presented in 1996 and published in 1997 (Catz, Itzkovich, Agranov, Ring, & Tamir, 1977). A refined second version (SCIM II) was published in 2001 (Catz et al., 2001). Both versions were sensitive to functional changes in patients with SCL (Catz et al., 1977, 2002), reliable, valid, and user friendly (Catz et al., 1977, 2001, 2002; Itzkovich et al., 2002). A third version (SCIM III) was formulated in 2002 to overcome intercultural differences (Catz et al., 2007; Itzkovich et al., 2007).

Current Knowledge

SCIM, the only comprehensive functional assessment instrument designed specifically for SCL, measures independence in every aspect of primary daily activities relevant to SCL patients. Tasks are scored according to their value for the patient, and every task or area of function is scored according to its relative weight in the total relevant daily function (Catz et al., 1977, 2001, 2002, 2007; Itzkovich et al., 2002, 2007). SCIM items are weighted based on a combination of (a) the items' value for the patient, (b) predicted difficulty, and (c) time required for performance. The value of each item for the patient and the relative weight of each tasks or area of function were initially based on a consensus of Israeli professionals. Following the findings of a Rasch analysis and consultations with European and American experts, these values were later modified for SCIM III (Itzkovich et al., 2002).

The SCIM is easy to use. Total score ranges from 0 to 100 (Catz et al., 1977, 2001, 2002, 2007; Itzkovich et al., 2002, 2007). The SCIM requires no manual (which would place a burden on raters), and all the instructions for scoring are detailed on the evaluation sheet.

Because it is patient oriented and user friendly, the SCIM can be useful for measuring the status or improvement of everyday functions relevant for SCL patients. The SCIM can also serve as a compact guide for determining treatment goals. In addition to assessing functional status, the SCIM can be used to define quantitative goals for functional restoration of primary daily activities. The SCIM form (Catz et al. 2007; Itzkovich et al., 2007) can help caregivers determine which of the patient's primary daily tasks should be improved, and how the performance of that task must change for the patient to achieve better function.

The SCIM can also be used for quantitative functional outcome assessment after interventions (Grijalva et al., 2003; Popovic, Thrasher, Adams, Takes, Zivanovic, & Tonack, 2006).

SCIM III has undergone a multicenter international evaluation with participants from Canada, Germany, Denmark, England, Italy, and Israel. Findings showed that the total agreement between raters was 74.5–96.2% for all SCIM tasks, with κ values between 0.631 and 0.823 ($p < 0.001$) and intraclass correlation coefficient values of 0.94–0.97. Cronbach's α was above 0.7 (Itzkovich et al., 2007). The coefficient of Pearson's correlation between the Functional Independence Measure (FIM) and SCIM III was 0.79 ($p < 0.01$) (Itzkovich et al., 2007). The McNemar test showed that the SCIM was significantly more sensitive than the FIM to changes in total score between the first and second examination (changes > 1 raw score point, the least detectable) on the Respiration and Sphincter Management and on the Mobility Indoors and Outdoors subscales ($p < 0.001$). Differences between the two scales were nonsignificant on the other two subscales, Self-care and Mobility in Room and Toilet (Itzkovich et al., 2007).

Most SCIM III items showed an acceptable fit to the Rasch model (mean square fit values of 0.6–1.4), indicating that categories that represent higher difficulty are passed by fewer people and by people with higher abilities. According to the Rasch analysis, the raters scored only patient ability and not other patient properties, used item categories in correct and hierarchical order, and differentiated well between levels of patient ability and item difficulty. Item hierarchy was found to be stable across most clinical subgroups and across countries (Catz et al., 2007). The findings support the validity and reliability of SCIM III despite intercultural differences, demonstrate its superior sensitivity to changes in function compared with FIM, and justify its use in clinical research, including cross-cultural trials. Currently, the SCIM is being used for clinical purposes in spinal cord units in North America, Europe, East Asia, and the Middle East.

Future Directions

Following comments received from users in various countries, the findings of the international study, (Catz et al., 2007; Itzkovich et al., 2007) and the findings of a current multicenter study in the USA, the intention is to develop a fourth version of the SCIM. To assess the potential and actual contribution of rehabilitation to changes in the function of SCL patients, another instrument is also being developed, combining SCIM scores with the Americans Spinal Injury Association (ASIA) motor scores (Catz, Greenberg, Itzkovich, Bluvshtein, Ronen, & Gelernter, 2004).

Cross References

- ► Functional Assessment
- ► Spinal Cord Injury

References and Readings

Catz, A., Itzkovich, M., Agranov, E., Ring, H., & Tamir, A. (1977). SCIM-spinal cord independence measure: A new disability scale for patients with spinal cord lesions. *Spinal Cord, 35*, 850–856.

Catz, A., Itzkovich, M., Steinberg, F., Philo, O., Ring, H., Ronen, J., et al. (2001). The Catz-Itzkovich SCIM: A revised version of the spinal cord independence measure. *Disability & Rehabilitation, 23*, 263–268.

Catz, A., Itzkovich, M., Steinberg, F., Philo, O., Ring, H., Ronen, J., et al. (2002). Disability assessment by a single rater or a team: A comparative study with the Catz-Itzkovich spinal cord independence measure. *Journal of Rehabilitation Medicine, 34*, 226–230.

Catz, A., Itzkovich, M., Tamir, A., Philo, O., Steinberg, F., Ring, H., et al. (2002). SCIM-spinal cord independence measure (version II): Sensitivity to functional changes [Hebrew]. *Harefuah, 141*, 1025–1031.

Catz, A., & Itzkovich, M. (2003). Trends in the assessment of functional outcomes after spinal cord lesions. In N. Soroker & H. Ring, (Ed.), *Advances in physical and rehabilitation medicine* (pp. 123–127). Bologna (Italy): Monduzzi.

Catz, A., Greenberg, E., Itzkovich, M., Bluvshtein, V., Ronen, J., & Gelernter, I. (2004). A new instrument for outcome assessment in rehabilitation medicine: Spinal cord injury ability realization

measurement index (SCI-ARMI). *Archives of Physical Medicine and Rehabilitation, 85*, 399–404.

Catz, A., Itzkovich, M., Tesio, L., Biering-Sorensen, F., Weeks, C., Laramee M. T., et al. (2007). A multi-center international study on the Spinal Cord Independence Measure, version II: Rasch psychometric validation. *Spinal Cord, 45*, 275–291.

Dodds, T. A., Martin, D. P., Stolov, W. C., & Deyo, R. A. (1993). A validation of the functional independence measurement and its performance among rehabilitation inpatients. *Archives of Physical Medicine and Rehabilitation, 74*, 531–536.

Grijalva, I., Guizar-Sahagun, G., Castaneda-Hernandez, G., Mino, D., Maldonado-Julian, H., Vidal-Cantu, G., et al. (2003). Efficacy and safety of 4-aminopyridine in patients with longterm spinal cord injury: A randomized, double-blind, placebo-controlled trial. *Pharmacotherapy, 23*, 823–834.

Itzkovich, M., Tripolski, M., Zeilig, G., Ring, H., Rosentul, N., Ronen, J., et al. (2002). Rasch analysis of the Catz-Itzkovich spinal cord independence measure. *Spinal Cord, 40*, 396–407.

Itzkovich, M., Tamir, A., Philo, O., Steinberg, F., Ronen, J., Spasser, R., et al. (2003). Reliability of the Catz-Itzkovich spinal cord independence measure assessment by interview and comparison with observation. *American Journal of Physical Medicine & Rehabilitation, 82*, 267–272.

Itzkovich, M., Gelernter, I., Biering-Sorensen, F., Weeks, C., Laramee, M. T., Craven, B. C., et al. (2007). The spinal cord independence measure (SCIM) version III: Reliability and validity in a multi-center international study. *Disability & Rehabilitation, 29*, 1926–1933.

Morganti, B., Scivoletto, G., Ditunno, P., Ditunno, J. F., & Molinari, M. (2005). Walking index for spinal cord injury (WISCI): Criterion validation. *Spinal Cord, 43*, 27–33.

Popovic, M. R., Thrasher, T. A., Adams, M. E., Takes, V., Zivanovic, V., & Tonack, M. I. (2006). Functional electrical therapy: Retraining grasping in spinal cord injury. *Spinal Cord, 44*, 143–151.

Spinal Cord Injury

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Synonyms

Spinal cord dysfunction

Short Description

Spinal cord injury refers to injury due to trauma, ischemia, or disease to the portion of the central nervous system that conducts impulses between the body and the brain and is located inside the vertebral canal.

Categorization

Spinal cord injury (SCI) can be subdivided into traumatic and non-traumatic SCI. Traumatic SCI occurs when an outside force is applied to the spinal cord. This force is typically from a vertebra or vertebral disc that is displaced due to trauma and consequently presses on the spinal cord and its vascular supply. Less commonly, an object such as a bullet or knife blade directly injures the spinal cord. Non-traumatic SCI etiologies include vascular insufficiencies or malformations, infections, disc prolapse or bony stenosis, demyelinating disease, neoplasms, and the effects of radiation.

The standard classification system developed by the American Spinal Cord Injury Association (www.asia-spinalinjury.org/publications/2006_Classif_worksheet.pdf) is based on functional outcomes depending on the variables of neurological level and extent of injury. The neurological level is the lowest segment of the spinal cord with normal movement and feeling. Tetraplegia involves sensory and motor loss throughout the body due to SCI at the cervical level. Paraplegia involves sensory and motor loss in the legs and trunk due to injury at or below the first thoracic vertebra. Injuries are further subdivided into complete injuries where sensory and motor function is absent below the level of the injury and incomplete injuries where some residual function below the level of the injury remains. SCI can be further subdivided depending on the location of the injury within the spinal cord. For example, incomplete SCI can be further classified into such categories as ventral, Brown–Sequard, and central cord syndromes.

Epidemiology

Approximately two-thirds of SCI is due to trauma. Those with traumatic SCI have been extensively studied in the United States through the SCI Model Systems since the early 1970s and represent a distinct population compared with those with non-traumatic onset SCI. The National Spinal Cord Injury Statistical Center database (www.spinalcord.uab.edu) is focused on individuals with traumatic SCI. This database reveals an annual incidence rate of traumatic SCI in the USA of 11,000 cases per year. The current US prevalence is about 250,000 persons. The current mean age at onset is 36 years, with an increasing number of individuals older than 60 years incurring traumatic SCI. Males represent about 80% of those with traumatic SCI. The primary causes of traumatic SCI include motor vehicle crashes (50%), falls (24%), violence

(11%), and sports or recreation (9%). Traumatic SCIs are frequently accompanied by other injuries including loss of consciousness (28%) and head injury (12%). Thus, routinely assessing for the possibility of brain injury is important. The causes of SCI vary based on such demographic factors as gender, ethnicity, age, and rural versus urban setting. For example, falls are the most common cause of SCI among older individuals and violence accounts for 46% of SCIs among African Americans.

Unlike those with traumatic SCI, those with non-traumatic SCI typically do not enter major trauma centers and are not tracked in standard SCI databases. McKinley and colleagues (1998) reported that non-traumatic SCI is primarily due to either spinal stenosis (narrowing of the spinal canal) or spinal cord compression due to tumors. Individuals with non-traumatic SCI are more likely to be older (greater than 50 years), female, married, and retired compared with those with traumatic SCI. While the focus of this synopsis is on traumatic SCI, the psychological issues faced by those with SCI due to the effects of aging or disease are equally demanding from a psychological standpoint. Fear of loss of independence, stress placed on care providers, and reduced quality-of-life are but a few of the multiple issues faced by these persons.

Psychology of Spinal Cord Injury

Spinal cord injury is arguably one of the most distressing and psychologically disorganizing events that anyone could experience. Losing the ability to walk and having to rely on a wheelchair for mobility represents one of the most feared events in life. Psychological adjustment to SCI was initially explained using a simple stage model. Current conceptualizaions have evolved into more sophisticated models, which describe coping and adaptation as a dynamic process that changes over time and is affected by intrapersonal, biological, environmental, and social factors. The reader is referred to the classic text of Trieschmann (1988) for a historical perspective and the chapter by Richards and colleagues for a contemporary overview of the field.

While there are many issues that might be discussed in regard to SCI, three issues are noteworthy. First, researchers have long recognized that males who incur traumatic SCI do not represent a random sample of the population. More than half of these injuries occur in the 16–30-year-olds and 80% are male. Rohe (1996) reviewed the research on personality and SCI and summarized that males with traumatic SCI are typically conventional, socially retiring, practical, physically oriented individuals who may have trouble expressing feelings, avoid physical closeness, and can be interpersonally distant. They prefer to work with things (tools and machines) rather than with ideas or people. They are visual and kinesthetic learners, who prefer to learn by discovering environmental contingencies. They tend to be less intellectually curious, persistent, and achievement-oriented. While this personality description does not apply to males with non-traumatic SCI, if SCI onset occurs secondary to behavior, then this personality description is likely to apply. These data suggest that the frequency of males with SCI with Axis II disorders is similar to that of the general population; however, assessment for Axis I mood disorders such as anxiety and depression is particularly important. Depression in those with SCI has been the subject of significant research with widely varying rates reported across studies. Bombardier and colleagues (2004) found a prevalence rate of 11.4% in a community dwelling sample of persons with SCI using the Patient Health Questionnaire 9, about twice the rate in the general population. When assessing mood in persons with SCI, it is vital to strictly adhere to diagnostic criteria. Assessing depression during acute rehabilitation is especially complicated due to such factors as confusing depression with grief/mourning and the impact of trauma, hospital routine, and medications on energy, appetite, and sleep.

Second, studies indicate that between a third and a half of traumatic SCI occurs in conjunction with alcohol or drug use. A total of 35–57% of this population shows evidence of alcohol abuse before injury. The implications of these high rates of chemical health problems pre-injury include the need to routinely assess chemical health after injury, developing an intervention plan with the patient and advising medical providers about possible iatrogenic chemical health problems when they prescribe medications for SCI-related problems of pain and spasticity.

Third, the literature suggests that as many as 50% of persons with traumatic SCI experience either loss of consciousness or post-traumatic amnesia. Since inpatient rehabilitaiton is focused on learning the skills necessary to live successfully with an SCI, assessment for potential cognitive impediments to learning may prove essential. Subsequent adaptation to SCI is dependent on intact cognitive functioning.

Evaluation

Evaluation of persons with SCI begins by establishing a collaborative therapeutic relationship through carefully

taking a comprehensive social history. Topics in the comprehensive social history may include family of origin, religious background, educational and occupational attainment, social adjustment, stressors at injury onset and prior history of mental or chemical health diagnoses and treatment, and the patient's most pressing concerns, to name a few. The primary goal of social history taking is to see the SCI through the eyes of the patient. This is done by understanding the person in the context of their developmental history with particular attention to prior learning experiences that may affect attitudes and behaviors associated with disability. The most critical question to be answered is: What is the meaning of the SCI for the patient and their life? The patient's understanding of their disability may be incomplete either due to lack of understanding of what has been communicated or disbelief that the SCI is permanent. Sensitively taking a comprehensive social history is vital to the work of the psychologist, whether it occurs during acute rehabilitation or years after the onset of the injury.

The comprehensive social history provides insight regarding what additional assessment is needed. If the initial interview raises the question of a possible mood disorder, subsequent evaluation may include administration of brief mood screening instruments such as the Beck Anxiety Inventory (BAI), the Beck Depression Inventory Fast Screen (BDIFS), or the Patient Health Questionnaire 9 (PHQ-9). The BAI contains 21 items that reflect the cognitive, affective, and physiological aspects of anxiety. The BDIFS was specifically designed to assess depression in medical patients and excludes physical symptoms of depression, focusing instead on the affective and cognitive aspects of depression. The PHQ-9 (www.phqscreeners.com) is a nine-item scale whose content specifically parallels the diagnostic criteria for depression. The caveat when administering it to individuals with SCI is that any positive item responses that are primarily due to non-mood factors are not included in the scoring. Frequently used measures of personality in persons with spinal cord injury include the Minnesota Multiphasic Personality Inventory 2, the NEO Personality Inventory Revised, and the Personality Assessment Inventory. Psychologists who work with individuals with SCI are frequently trained in career assessment, as this is vital to social reintegration. Frequently used measures of vocational interests are the Strong Interest Inventory and the Career Assessment Inventory.

Assessment of chemical health begins by determining if there are laboratory data available on the presence of alcohol or non-prescribed medications at the time of injury onset. Presence of such substances highlights the need to include a careful history of past and current use of all substances including alcohol, nicotine, caffeine, and street drugs. Two helpful alcohol-screening measures are the CAGE questions and the Alcohol Use Disorders Identification Test.

Assessment of cognitive status typically begins with a screening instrument followed by more in-depth assessment if indicated. If the person with SCI has a documented head injury, assessment may begin with the Galveston Orientation and Amnesia Test. This is then followed by a brief cognitive status examination such as the Montreal Cognitive Assessment (www.mocatest.org.). Timing of additional cognitive assessment is influenced by a variety of factors including the relevance of the testing to post-discharge planning, including questions of safety. Given the current short hospital lengths of stay, administering brief cognitive test batteries such as the Repeatable Battery for the Assessment of Neuropsychological Status or the Cognistat is typical. If acquired brain injury is identified, the patient is typically seen for comprehensive outpatient neuropsychological assessment after discharge. Assessment of persons with tetraplegia requires selection of a non-motorically mediated test battery and test interpretation that considers the impact of compromised physical function on test results.

Treatment

Psychological interventions for persons with SCI vary depending on the issues identified and whether this is occurring during acute rehabilitation or after discharge. The most pressing initial issue is to establish a trusting relationship with the patient. Since males with traumatic SCI are typically less than enthused about meeting with a psychologist, countering stereotypical beliefs about mental health providers is essential. This can be accomplished during the initial interview by explaining that you are a regular member of the rehabilitation team and routinely meet to discuss such things as coping with the "hassles" of SCI, managing mood, discussing sexuality, and providing vocational planning. A first step is to have the patient identify what is most concerning to them, and let that topic provide the focus of discussion. The vast majority of those with new SCI cope effectively. The more important role for the psychologist falls in the realm of psycho-educational interventions. Providing SCI educational materials such as books (e.g. Roll Models; Spinal Cord Injury, A Guide for Living) or video materials are typical interventions. Linking the patient with peer mentors is a frequent psycho-educational intervention. Meeting with

the patient's family, spouse, or significant other is also common. If disorders of mood or personality are identified, explanation to the patient is provided and appropriate treatment initiated. The same is true if chemical health or cognitive problems are identified. The goal is to provide an appropriate intervention while engaging the patient and their family in the treatment planning and intervention process. For example, mood interventions may include initiation of pharmacotherapy couple with cognitive behavior therapy. Chemical health interventions may include use of motivational interviewing techniques coupled with a written plan on next steps to improving chemical health. Cognitive impairment interventions may include discussion with rehabilitation team members and family members about helpful potential interventions.

Cross References

- ▶ Cognistat
- ▶ Cognitive Behavioral Therapy
- ▶ Galveston Orientation and Amnesia Test
- ▶ Interdisciplinary Rehabilitation Team
- ▶ Mental Status Exam
- ▶ Motivational Interviewing
- ▶ Rehabilitation Psychology
- ▶ Repeatable Battery for the Assessment of Neuropsychological Status
- ▶ Spinal Cord
- ▶ Traumatic Brain Injury
- ▶ Vocational Rehabilitation

References and Readings

Bombardier, C. H., Richards, J. S., Krause, J. S., Tulsky, D., & Tate, D. G. (2004). Symptoms of major depression in people with spinal cord injury: Implications for screening. *Archives of Physical Medicine & Rehabilitation, 85*(11), 1749–1756.

Holicky, R. (2004). *Roll models.* Vancouver, Canada: Trafford.

Kirschblum, S. (2005). Rehabilitation of spinal cord injury in physical medicine and rehabilitation principles and practices. In J. A, DeLisa (Ed.), (pp. 1715–1751). Philadelphia, PA: Lippincott.

McKinley, W. O., Hardman, J., Seel, R. (1998). Nontraumaic spinal cord injury: Incidence, epidemiology and functional outcome. *American Journal of Physical Medicine & Rehabilitation, 79*, 1186–1187.

Palmer, S., Kriegsman, K. H., Palmer, J. B. (2008). *Spinal cord injury: A guide for living* (2nd. ed.). Baltimore, MD: The Johns Hopkins University Press.

Richards, J. S., Kewman, D. G., Richardson, E., & Kennedy, P. (2009). Spinal cord injury. In R. G. Frank, M. Rosenthal, & B. Caplan (Eds.), *Handbook of rehabilitation psychology* (2nd edn). American Psychological Association.Washington, DC:

Rohe, D. E. (1996). Personality and spinal cord injury. *Topics in spinal cord injury, 2*(9), 1–10.

Trieschmann, R. B. (1988). *Spinal cord injuries: Psychological, social and vocational rehabilitation* (2nd ed.). New York: Demos.

Spinal Ganglia

- ▶ Dorsal Root Ganglia

Spinal Neuraxis

- ▶ Spinal Cord

Spinal Nucleus of V

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Synonyms

Spinal trigeminal nucleus

Definition

Brainstem nucleus that extends from the lateral pons, through the medulla and into the upper cervical cord. The largest of all cranial nerve nuclei, the spinal nucleus of V is one of the three paired sensory nuclei associated with the trigeminal or fifth cranial nerve. The spinal nucleus of V receives input from the spinal tract of V which carries information regarding pain, temperature, and crude touch from the face and forehead. In turn, it gives rise to the ventral trigeminothalamic tract which transmits this information to the ventral posterior medial nucleus of the thalamus and eventually to the somatosensory cortex. Brainstem lesions involving this nucleus (or tract) would be expected to result in ipsilateral loss of pain and temperature in the face and/or forehead.

Cross References

- ▶ Spinal Tract of V

References and Readings

Wilson-Pauwek, L., Akesson, E. J., Stewart, P. A., & Spacey, S. D. (2002). *Cranial nerves in health and disease.* Hamilton, Ontario, Canada: B.C. Decker.

Spinal Puncture

► Lumbar Puncture

Spinal Tap

► Lumbar Puncture

Spinal Tract of V

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Synonyms

Spinal trigeminal tract

Definition

Fiber tract associated with the trigeminal or fifth cranial nerve which extends from the pons to the upper cervical cord. The tract carries sensations of pain, temperature, and crude touch from the face and forehead and in this sense it is the brainstem equivalent of the anterolateral system of the spinal cord. The spinal tract of V lies lateral and adjacent to the spinal nucleus of V with which it synapses. Brainstem lesions involving this tract would result in ipsilateral loss or disturbances in the perception of pain and temperature in the face and/or forehead.

Cross References

► Spinal Nucleus of V

References and Readings

Wilson-Pauwek, L., Akesson, E. J., Stewart, P. A., & Spacey, S. D. (2002) *Cranial nerves in health and disease.* Hamilton, Ontario, Canada: B.C. Decker.

Spinal Trigeminal Nucleus

► Spinal Nucleus of V

Spinal Trigeminal Tract

► Spinal Tract of V

Spinocerebellar Tracts

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Definition

Spinal cord pathways that carry somatosensory information from the trunk and extremities to the cerebellum.

Current Knowledge

The spinocerebellar pathways are divided into a *dorsal* (posterior) and *ventral* (anterior) *spinocerebellar tract, both of* which lie in the lateral portion of the cord. These latter tracts supply proprioceptive and other somatosensory information to the cerebellum from muscle spindles, Golgi tendon organs, and cutaneous touch and pressure receptors. The information carried by these tracts to the cerebellum is not consciously perceived but nevertheless is critical to the spinal-cerebellar-cortical-spinal feedback loops essential for coordination and balance. The anatomical distinctions between the dorsal and ventral spinocerebellar tracts are clearer than their functional distinctions. The spinal input for both tracts is via the dorsal nerve roots. The dorsal spinocerebellar tract is

proximately derived from axonal fibers generated in the dorsal nucleus of Clarke that extends from the lumbar to lower cervical cord. These fibers remain ipsilateral in the cord and enter the cerebellum through the inferior cerebellar peduncle. By contrast, the ventral spinocerebellar tract is made up of fibers that synapse more diffusely in the gray matter of the cord and the majority of which ascend in the contralateral cord. Again in contrast to the dorsal fibers, these ventral tracts enter the cerebellum through the superior cerebellar peduncle, after the majority once again cross the midline in the brain stem. Thus, for the most part, each half the cerebellum receives ipsilateral input from the spinal cord. The *cuneocerebellar* and *rostral spinocerebellar* tracts are thought to represent the respective equivalents of the dorsal and ventral spinocerebellar tracts for the upper extremities.

Cross References

► Cerebellum

References and Readings

Haines, D. E., Mihailoff, G. A., & Bloedel, J. R. (1997). The cerebellum. In D. E. Haines (Ed.), *Fundamental neuroscience* (pp. 379–398). New York: Churchill Livingstone.

Spinoreticular Tract

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Definition

Phylogenetically old tract that likely carries pain and other somatosensory information from the trunk and extremities.

Current Knowledge

As the name implies, the tract originates in the spinal cord and terminates in the reticular formation (RF) in the brainstem. While most of the fibers appear to terminate in the RF of the medulla and lower pons, some may ascend as far as the midbrain. The major role of these fibers is thought to be general arousal rather than sensory discrimination per se. If the organism is sleeping, resting, or otherwise not paying particular attention to what is going on around it, there is potentially great survival value in being alerted to things that unexpectedly come into contact with the body. Whether it is something that bites, stings, brushes up against the skin or hair, or simply causes the ground or perch on which it is resting to vibrate, the organism may need to rapidly change its level of arousal and attend to its surroundings to avoid being eaten, injured, or otherwise harmed. Think of someone lying in bed at night, drowsy and ostensibly alone when they feel something gently brush up against their leg. Without taking the time to think about what it could be, the initial response is likely to immediately become fully alert and quickly withdraw their leg, if not actually jump up out of the bed. This is an example of the nonspecific early warning system of the spinoreticular tract at work.

References and Readings

André P. (1996). *Carpenter's human neuroanatomy* (9th ed.). Baltimore: Williams and Wilkins.

Spinothalamic Tract

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Synonyms

Lateral spinothalamic tract; Ventral spinothalamic tract

Definition

See ► Anterolateral System.

Cross References

► Anterolateral System

Splenium

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Definition

Term for the bulbous, posterior portion of the corpus callosum which lies above the quadrigeminal cistern. This portion of the corpus callosum is supplied by the posterior cerebral artery and consists of commissural fibers interconnecting the posterior parietal, posterior temporal and occipital cortices. Lesions in this area are associated with the well-known neurobehavioral disconnection syndrome of *alexia without agraphia*. This syndrome can result from infarction of the left posterior cerebral artery. Here, in addition to suffering a right homonymous hemianopia (secondary to left occipital lobe infarction), visual input (i.e., written words) to posterior language area of the left hemisphere from the intact right occipital lobe is disrupted by the infarction of the splenium.

Cross References

► Alexia Without Agraphia

Split-Brain

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Synonyms

Callosal disconnection syndrome; Commissurotomy; Hemispheric disconnection

Definition

Split-Brain is a condition resulting from surgical lesioning of all or a substantial portion of the corpus callosum, with or without sectioning of other commissures, thus interrupting the normal flow of information between the two hemispheres of the brain. While carried out experimentally in animals, in humans such relatively drastic surgical procedures are quite rare. The most common indication is to help control otherwise intractable generalized tonic–clonic (*grand mal*) seizures.

Historical Background

Around the turn of the twentieth century, Hugo Liepmann, a German neurologist, described a right-handed, right-hemiplegic patient who had difficulty executing verbal commands, writing, or correctly demonstrate the use of tools with his left hand. Upon subsequently discovering the patient had a lesion involving the corpus callosum, Liepmann postulated that the memory for skilled movements must have resided in the left hemisphere and normally communicated with the right hemisphere (controlling the left hand) via the corpus callosum. Although later ablation studies initially failed to confirm his theory, this was due to the fact that animal subjects were used and the experimenters likely failed to ask the right questions.

Studies of humans with callosal section conducted by Akelatis (1944/1945) produced negative findings as well, possibly, in part, because the anatomical disconnection was incomplete, but certainly because once again, the investigative techniques were insufficiently sensitive – or perhaps more fairly, insufficiently ingenious.

It required the thoughtful and clever methods of Geschwind and Kaplan (1962) to begin to elucidate callosal function. They discovered, for example, that their patient produced lucid writing with the right hand (left hemisphere) but "aphasic" writing with the left hand (right hemisphere). They also reported anomia for objects placed in the left hand but not the right. Their combination of neuroanatomical insight and clinical inventiveness also characterized the work of Roger Sperry and his colleagues, to whom the field owes the greatest debt for their detailed studies of split-brain patients.

By the middle of the 20th century, Sperry and his associates noted that if both the corpus callosum and the optic chiasm were lesioned in animals (thus, restricting visual information from each eye to the ipsilateral hemisphere), dramatically different results were obtained. Not only could the animal not carry out a discrimination task previously learned by one eye (cerebral hemisphere) when only the opposite eye was left uncovered, but each eye (cerebral hemisphere) could learn conflicting tasks, depending on which eye was used. In 1961, using a similar preparation in monkeys, Downer demonstrated differences in affective responses to visual stimuli following

unilateral temporal lesions, again depending on which eye was employed. Although naturally occurring, callosal lesions in humans, or even more surprisingly in cases of callosal agenesis, there is typically a failure to evidence signs of a disconnection syndrome. However, in their classic series of studies in the 1960s, Gazzaniga, Bogen, and Sperry clearly demonstrated the effects of split-brain preparations in human subjects.

Current Knowledge

Both cerebral hemispheres are believed to make significant contributions to most routine daily functions, including language-based activities being carried out primarily by the left hemisphere and the right hemisphere apparently being the more important for certain perceptual and emotional functions. Somewhat surprisingly perhaps, following commissurotomy, behavioral deficits were not always obvious. This is probably due to the extensive cross-cueing that normally takes place, especially through vision where the right hand sees what the left one is doing and vice versa.

However, when studied under more stringent experimental conditions where sensory input was limited to one hemisphere, the consequences of callosal disconnection readily become apparent. For example, when deprived of visual feedback, the commissurotomized participant is unable to name an object held in the left hand because the left (naming) hemisphere no longer has access to information in the right hemisphere. For the same reason, when deprived of visual input, one hand is unable to select an object held by the other. While the left hand is unable to write an intelligible sentence, it may surpass the efforts of the right in copying a complex geometric design. If emotionally charged images are restricted to the left visual field, a split-brain patient may evidence an appropriate affective response; however, because the speaking left hemisphere is disconnected from the hemisphere that saw the image, he or she may be at a loss to explain their reaction. On rare occasions, patients have been dismayed that one hand (usually the left) might be carrying out some activity of which the left (speaking hemisphere) is unaware, or find that one hand is working in a counterproductive fashion to the other (e.g., one hand pulling pants up and the other pushing them down).

In addition to providing insights into how the two hemispheres normally interact and their respective strengths, studies of split-brain patients have raised some intriguing questions about the presumed unitary nature of consciousness.

It should be noted that the disconnection effects typically observed after surgical section are not found in cases of callosal agenesis.

Cross References

- ▶ Akelaitis, Andrew John Edward ("A.J.") (1904–1955)
- ▶ Alexia Without Agraphia
- ▶ Corpus Callosum
- ▶ Disconnection Syndrome
- ▶ Gazzaniga, M. S. (1939–)
- ▶ Kaplan, Edith (1924–2009)
- ▶ Norman Geschwind
- ▶ Sperry, Roger Wolcott (1913–1994)

References and Readings

Akelatis, A. J. (1944/1945). Studies on the corpus callosum, IV: Diagnostic dyspraxia in epileptics following partial and complete section of the corpus callosum. *American Journal of Psychiatry, 101*, 594–599.

Gazzaniga, M. S., Ivry, R. B., & Mangun, G. R. (2002). *Cognitive neuroscience: The biology of the mind* (2nd ed.). New York: W.W. Norton & Company.

Geschwind, N., & Kaplan, E. (1962). A human cerebral deconnection syndrome: A preliminary report. *Neurology, 12*, 675–685.

Sperry, R. W. (1982). Some effects of disconnecting the cerebral hemispheres. *Science, 217*, 1223–1226.

Zaidel, E., Iacoboni, M., Zaidel, D. W., & Bogen, J. E. (2003). The callosal syndromes. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (pp. 347–403). New York: Oxford University Press.

SPM

- ▶ Standard Progressive Matrices

SPM-C

- ▶ Standard Progressive Matrices

Spongiform Encephalitis

- ▶ Kuru

Spongiform Encephalopathy

► Creutzfeldt-Jakob Disease

Spontaneous Recovery

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Synonyms

Plasticity

Definition

The natural redevelopment of or improvement in function following insult or injury to the nervous system.

Current Knowledge

Spontaneous recovery generally occurs following acute, nonprogressive, neurological insults such as strokes (either hemorrhagic or occlusive), closed head trauma, tumor resection, and anoxia. The mechanisms underlying behavioral or functional improvements depend, in large part, on the nature of the original pathology.

The many theories about how the brain recovers from such insults may be thought of as falling into one of two broad categories, for example, those that attempt to explain either acute (short-term) or long-range recovery. Some common mechanisms associated with acute recovery likely involve edema and other forms of increased pressure causing temporary suppression of function. Edema can result from a breakdown of intracellular processes or *cytotoxic edema* (as is common in infarcts), a disruption of the blood-brain barrier or *vasogenic edema* (as often occurs in brain tumors and traumatic brain injuries), or as a result of disruption of CSF circulation (*interstitial edema*). Pressure can also be exerted on brain structures by the accumulation of blood, either directly inside brain tissue or simply within the cranial cavity as a result of hemorrhagic lesions. Such lesions may be primary, as might result from hypertension, or secondary, as seen in closed head injury (e.g., subdural or intraparenchymal hematomas). Whether as a result of edema, hemorrhage or brain tumor, the resulting pressure on brain tissue, either local or generalized, can disrupt normal neuronal function. While extensive increased intracranial pressure can be fatal, in nonfatal cases it resolves over time, resulting in at least partial restoration of function. In addition to exerting local pressure on brain tissue, intraparenchymal blood has a toxic effect on neurons themselves until reabsorbed.

Immediately following an injury to the brain, acute changes in metabolic functions and a disruption of neurochemical processes can contribute to behavioral disturbances. In addition to a disturbance of normal neurochemical pathways, an excess release of glutamate can lead to increases in intracellular calcium and production of oxygen free radicals, all of which can contribute to excitotoxicity and subsequent cell death. The eventual stabilization of these processes can also provide a basis for recovery.

Another example of short-term spontaneous recovery is that provided by transient ischemic attacks (TIAs) where return of function is thought to result from spontaneous resolution (in whole or in part) of circulation to the affected brain area. While perhaps not strictly constituting "spontaneous recovery," similar effects can often follow the prompt administration of thrombolytic drugs following ischemic strokes.

Functional recovery can continue for days or weeks, possibly for years. The mechanisms behind these long-term changes are less well studied. In the case of strokes, anoxia, or other types of brain injury, it is suspected that different levels of damage may occur. For example, at the nidus of an infarction, there may be a total loss of blood supply (e.g., glucose and oxygen) to the neurons resulting cell death, eliminating all possible recovery utilizing those cells. However, surrounding the area of total infarction, an incomplete loss of circulation may result in cells that are rendered temporarily dysfunctional. This region is often referred to as the *ischemic penumbra* and the phenomenon as the "*idling neuron hypothesis*."

Another theory behind long-term recovery is that over time some revascularization occurs that enables damaged neurons to function more efficiently. The concept of "dendritic sprouting" and the resulting establishment of new synaptic connections that has been offered to explain recovery in such damaged areas, although new, long axonal connections are not thought to develop.

The practices of rehabilitation medicine and cognitive rehabilitation appear to be largely based on other premises. It is believed that, following damage to a portion of

the brain, at least two things might happen. The first is that other areas of the brain may assume the function(s) of the damaged cortex or that new neuronal pathways are recruited to carry out a particular function. The consequences of early loss of language or sight appear to support this idea. When language is lost as a result of childhood brain injury, the recovery or reemergence of language skills is usually much better than when such loss occurs later in life. Similarly, individuals who lose their sight appear to develop enhanced acuity of other sensory modalities, allowing them, for example, to become proficient in Braille. Such recoveries or enhanced proficiencies are attributed to a phenomenon known as *neuroplasticity.*

A second premise behind rehabilitation programs is that the patient learns new techniques, strategies, or other compensatory mechanisms to carry out functions impaired by the brain lesion. While these latter two mechanisms of recovery likely occur spontaneously, repeated, facilitated, or guided practice may be useful.

Cross References

► Diaschisis
► Ischemic Penumbra

References and Readings

Carmichael, S. T., & Chesselet, M. (2002). Synchronous neuronal activity is a signal for axonal sprouting after cortical lesions in the adult. *The Journal of Neuroscience, 22*, 6062–6070.

Dobkin, B. (2003). *The clinical science of neurologic rehabilitation.* New York: Oxford University Press.

Finger, S., Koehler, P. J., & Jagell, C. (2004). The Monakow concept of diaschisis: Origins and perspectives. *Archives of Neurology, 61*, 283–288.

Fisher, M. (2004). The ischemic penumbra: Identification, evolution and treatment concepts. *Cerebrovascular Diseases, 17*(Suppl. 1), 1–6.

Kennard, M. A. (1938). Reorganization of motor function in the cerebral cortex of monkeys deprived of motor and premotor areas in infancy. *Journal of Neurophysiology, 1*, 477.

Kertesz, A., & Gold, B. T. (2003). Recovery of cognition. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (pp. 617–639). New York: Oxford University Press.

Kohn, B., & Dennis, M. (1974). Patterns of hemispheric specialization after hemidecortification for infantile hemiplegia. In M. Kinsbourne & W. Lynn Smith (Eds)., *Hemispheric disconnection and cerebral function.* Springfield, IL: C.C. Thomas.

Kolb, B., & Gibb, R. (2007). Brain plasticity and recovery from early cortical injury. *Developmental Psychobiology, 49*, 107–118.

Kolb, B., Gibb, R., & Robinson, T. E. (2003). Brain plasticity and behaviour. *Current Directions in Psychological Science, 12*, 1–5.

Reilly, J. S., Levine, S. C., Nass, R., & Stiles, J. (2008). Brain plasticity: Evidence from children with perinatal brain injury. In J. Reed & J. Warner-Rogers (Eds.), *Child Neuropsychology: Concepts, theory, and practice* (pp. 58–91). Malden, MA: Wiley-Blackwell.

Robertson, I. H., & Murre, J. M. J. (1999). Rehabilitation of brain damage: Brain plasticity and principles of guided recovery. *Psychological Bulletin, 125*(5), 544–575.

Sport-Related Concussion

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Synonyms

Concussion; Mild traumatic brain injury

Short Description or Definition

A sport-related concussion is synonymous with a mild traumatic brain injury (MTBI) sustained during a sports event such as football, soccer, or hockey. As discussed in the MTBI entry, concussion is a transient alteration in a neurological function caused by a direct blow to the head (i.e., blunt force) or by biomechanical forces (i.e., rapid rotation or deceleration) acting on brain tissue.

Diagnosis and Further Categorization of Concussion

Sport concussions, like all brain injuries, are diagnosed by identifying an event with enough force to cause concussive injury and by the athlete's acute injury characteristics. Although all concussions are MTBIs (► mild traumatic brain injury entry for a full listing of current MTBI definitions), attempts have been made to refine the diagnostic nomenclature for concussion to more accurately portray the heterogeneity of injuries within the category MTBI (► American Academy of Neurology [AAN], 1997; Aubry et al., 2002; Cantu, 1986, 2001; McCrory et al., 2005). Many different grading systems have been discussed in the literature as an attempt to better capture the severity of an individual's concussive injury, with a higher grade equating to a more severe concussion. Most systems differentiate between various grades or complexities of concussion by acute injury

variables, such as length of confusion, length of amnestic period, or presence or absence of loss of consciousness. There is considerable agreement in the diagnostic criteria among the various grading (diagnostic) systems.

Natural History, Prognostic Factors, Outcomes

Scientific Advances in Study of MTBI from Sports Concussion Research

The sport-concussion research paradigm created in the mid-1980s by Barth et al. (1989) has proven ideal for studying the acute effects of MTBI, recovery from MTBI, and the outcome following MTBI. Benefits of this methodology include access to a population at high risk for concussion (prospective study of concussion), opportunity for immediate evaluation and intervention of highly motivated persons following concussion, and easy access to matched controls (Freeman, Barth, Broshek, & Plehn, 2005; McCrea, 2008).

Epidemiology of Sport-Related Concussion

Available data indicate that an individual has approximately a 3 to –10% risk of concussion per year if he/she participates in football, and the risk for concussion appears to be greater during games than practice (Guskiewicz, Weaver, Padua, & Garrett, 2000; Macciocchi, 2006). Risk for concussion in other contact sports such as hockey approaches a comparable level of risk, but has not received as much empirical investigation. Please see Macciocchi (2006) for a comprehensive discussion of epidemiology of concussion during athletics.

Acute Symptoms and Natural Recovery from Sports Concussion

Acute symptoms of concussion include alteration in consciousness (confusion), loss of consciousness, focal neurological abnormalities (speech disturbance, weakness), cognitive deficits (attention problems), vision disturbance, balance problems, behavioral changes (irritability, inappropriate affect), and somatic complaints (headache, fatigue). Vegetative (sleep disturbance) and mood changes (anxiety or depression) may also occur, but represent examples of symptoms that may not be noticeable immediately after the injury. All symptoms associated with the injury are expected to slowly and gradually resolve with time.

Regarding natural recovery following a single concussion, most athletes will be symptom-free within 7 to 10 days, with very few individuals being symptomatic beyond 3 months (Belanger & Vanderploeg, 2005; Iverson, 2005; McCrea, 2008). Nevertheless, clinicians need to consider individual factors and medical history in modifying the recovery-and-outcome expectations for athletes. As discussed in greater detail in the MTBI entry, data suggest that (1) the vast majority of individuals recover fully from a single concussion, (2) individuals may be at a greater risk for repeat concussive injury if they are still recovering, (3) individuals with a history of recurrent concussion may have a slower recovery and (4) sustaining many concussions over the course of one's life may increase the risk for persisting or worsening neurological or cognitive abnormalities later in life.

Evaluation and Management of Sport-Related Concussion

Over the past two decades, the healthcare and sports communities have come to understand the public health concerns associated with MTBI, which has resulted in more sophisticated and standardized (i.e., less idiosyncratic) methods being established for evaluation, diagnosis, and management of sport-related concussion (AAN, 1997; Kelly & Rosenberg, 1997, McCrea, 2008). To this end, neuropsychologists have joined sports medicine physicians and other specialists (e.g., athletic trainers) to help with evaluation and management of athletes postconcussion. Neuropsychology provides a means for identifying cognitive residuals of concussion, objectively monitoring cognitive recovery post-injury, and informing decision-making regarding return-to-play.

Baseline Testing

Although frequently not feasible due to expense and time constraints, performing baseline neurocognitive evaluations on all athletes prior to the season can be very helpful in evaluating and managing an athlete post-concussion. Having a cognitive baseline on athletes allows for a more precise understanding of the athlete's acquired symptoms following concussion because baselines make it possible to compare an athlete's post-injury functioning to his or her pre-injury functioning. Because

athletes represent an accessible group at a considerable risk for concussion, baseline evaluations are possible and may be clinically beneficial. All baselines must be conducted with tests proven to have adequate temporal stability.

Acute Evaluation and Management Post-injury

Acute evaluation of sport-related concussion should theoretically be no different than evaluation of MTBI in other contexts. Athletes in organized sports who sustain a concussive injury, however, frequently receive immediate on-field and sideline evaluations of their injury, and also have access to comprehensive monitoring and management post-injury. On-field and sideline evaluations serve to diagnose the injury, rule out more devastating injury, and determine whether an athlete can return to play within the same practice or game. At a minimum, an athlete who sustains a concussion should immediately be evaluated by a medical specialist trained in emergency medicine protocols. If necessary, an athlete should be sent to an emergency department so that they can undergo evaluation using sensitive technologies such as computed tomography (CT) of the head. After a devastating central nervous system (brain and spine) abnormality or other injury to the body is ruled out, acute evaluation needs to include sideline assessment of mental status (cognition), neurological status including balance testing, and other symptoms status (e.g., headache, dizziness, vision changes). Protocols such as the Standardized Assessment of Concussion (SAC; McCrea et al., 1998) have been developed to assist athletic personnel in systematically evaluating the mental status following the concussion. If an athlete's acute symptoms resolve quickly at rest, the athlete needs to be evaluated for reemergence of symptoms under physical exertion. Increased intracranial pressure can lead to the reemergence of symptoms for individuals still recovering from concussion. Athletes need to be held out-of-play until they are symptom-free at rest and under exertion. As per sport-concussion management guidelines, the grade of concussion and the player's history of concussions will impact how long they should refrain from play. Immediate post-injury education about the injury, expected recovery, and outcome is recommended.

Post-acute Evaluation and Management

Following concussion, athletes are ideally evaluated over appropriate time intervals (minutes, hours, days, or weeks) by sports medicine specialists to monitor symptom recovery, and provide reasonable intervention for persisting symptoms, with the goal of determining when return-to-play is advisable.

Neuropsychological assessment protocols contribute information that is most pertinent to the postacute evaluation and management of concussed athletes. There is no better method than neuropsychological testing to evaluate and track cognitive status following concussion, and without psychometric data, sports medicine specialists must rely solely on an athlete's subjective report of cognitive symptoms postconcussion. Neuropsychological evaluation is a standardized method of quantifying a person's cognitive status for the purpose of determining if they are exhibiting any cognitive abnormalities (i.e., cognitive changes) in light of his or her history of concussion. These decisions are ideally based on intraindividual changes in cognition over time, – which is why baseline evaluations can be helpful, – but can also be made by comparing an individual's test performance relative to an estimated baseline and against appropriate normative groups (i.e., same-aged peers). At present, there are screening instruments (SAC; McCrea et al., 1998), a wide array of paper and pencil tests, and computerized measures (e.g., Immediate postconcussion assessment and cognitive testing (ImPACT; Lovell, Collins, Podell, Powell, & Maroon, 2005) or the Automated Neuropsychological Assessment Metrics (ANAM; Bleiberg, Cernich, & Reeves, 2006)) that can help inform management of an athlete following concussion.

In addition to the neuropsychologist's role in the evaluation and management of athletes following concussion, it is important for physicians or other qualified medical specialists to continue to monitor and treat any comorbid injuries or persisting symptoms (i.e., headache) associated with MTBI. As discussed above, athletes need to have their symptoms monitored at rest as well as under physical exertion, even in the postacute phase, as individuals who appear symptom-free at rest can experience an increase in symptoms during a physical exercise. Athletes should be considered symptomatic following concussion until they have resolution of all acquired symptoms at rest and under exertion, and until their cognitive profile is deemed commensurate with their baseline capacity.

An interdisciplinary approach to concussion management is ideal. Treatment for symptoms in the postacute recovery phase is generally a "symptom management" approach. In addition to pharmacological intervention, individuals may benefit from psychoeducation and cognitive-behavioral therapy, particularly athletes who are experiencing a prolonged recovery (Mittenberg, Canyock, Condit, & Patton, 2001; Mittenberg, Tremont, Zielinski, Fichera, & Rayls, 1996).

Return-to-Play Decision-Making

Following concussion, an athlete may be most concerned about when he or she will be cleared to safely return to play. Echemendia and Cantu (2004) highlight the complexity of the return-to-play decisions, and outline many variables that need consideration when making recommendations regarding when an individual should return to play. Areas that need to be carefully considered by treatment providers include concussion factors (i.e., severity of injury or history of concussion), player factors (age, style of play, career aspirations), and relevant medical factors. Neuropsychological data, as described above, can be very useful for identifying persisting cognitive symptoms following injury, particularly for individuals who are not subjectively experiencing cognitive residuals or who are minimizing symptoms to speed up the medical clearance for returning-to-play.

Return-to-play guidelines have been established to assist providers in making appropriate decisions. Such guidelines focus primarily on concussion factors and have not been empirically validated; instead, the available guidelines were generated by clinical consensus groups. Generally speaking, providers are encouraged to keep athletes from playing until they are symptom-free at rest and under exertion. Although the guidelines lack empirical validation, they appear to have clinical utility, and it is generally recommended that the guidelines be considered when managing concussed athletes until empirically derived algorithms that individualize return-to-play decisions become available to the clinician.

Cross References

► Automated Neuropsychological Assessment Metrics (ANAM)
► Immediate Post-Concussion Assessment and Cognitive Testing
► Postconcussion Syndrome

References and Readings

American Academy of Neurology. (1997). Practice parameter: The management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee. *Neurology, 48*, 581–585.

Aubry, M., Cantu, R., Dvorak, J., Graf-Baumann, T., Kelly, J., Lovell, M., et al. (2002). Summary and agreement statement of the first International Conference on Concussion in Sport, Vienna 2001. Recommendations for the improvement of safety and health of athletes who may suffer concussive injuries. *British Journal of Sports Medicine, 36*, 6–10.

Barth, J. T. Alves, W. M., Ryan, T. V., Macciocchi, S. N., Rimel, R. E., Jane, J. A., et al. (1989). Mild head injury in sports: Neuropsychological sequelae and recovery of function. In H. S. Levin, H. M. Eisenberg, & A. L. Benton (Eds.), *Mild head injury* (pp. 257–275). New York: Oxford University Press.

Belanger, H. G., & Vanderploeg, R. D. (2005). The neuropsychological impact of sport-related concussion: A meta-analysis. *Journal of International Neuropsychological Society, 11*, 345–357.

Bleiberg, J., Cernich, A., & Reeves, D. (2006). Sports concussion applications of the Automated Neuropsychological Assessment Metrics Sports Medicine Battery. In R. J. Echemendia (Ed.), *Sports neuropsychology* (pp. 263–283). New York: The Guilford Press.

Cantu, R. C. (1986). Guidelines for return to contact sports after a cerebral concussion. *Physician Sports Medicine, 14*, 75–83.

Cantu, R. C. (2001). Posttraumatic retrograde and anterograde amnesia: Pathophysiology and implications in grading and safe return to play. *Journal of Athletic Training, 36*, 244–248.

Echemendia, R. J. & Cantu, R. (2004). Return to play following cerebral head injury. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports: A neuropsychological and international perspective* (pp. 479–498). Linne, The Netherlands: Swets & Zeitlinger.

Freeman, J. R., Barth, J. T., Broshek, D. K., & Plehn, K. (2005). Sports injuries. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Textbook of traumatic brain injury* (pp. 453–476). Washington, DC: American Psychiatric Publishing, Inc.

Guskiewicz, K. M., Weaver, N. L., Padua, D. A., & Garrett, W. E. (2000). Epidemiology of concussion in collegiate and high school football players. *American Journal of Sports Medicine, 28*(5), 643–650.

Iverson, G. L. (2005). Outcome from mild traumatic brain injury. *Current Opinion in Psychiatry, 18*, 301–317.

Kelly, J. P. & Rosenberg, J. H. (1997). Diagnosis and management of concussion in sports. *Neurology, 48*(3), 575–580.

Lovell, M. (2006). The ImPACT neuropsychological test battery. R. J. Echemendia (Ed.), *Sports neuropsychology* (pp. 193–215). New York: The Guildford Press.

Lovell, M., Collins, M., Podell, K., Powell, J., & Maroon, J. (2005). *ImPACT: Immediate post-concussion assessment and cognitive testing.* Pittsburgh, PA: NeuroHealth Systems, LLC.

Macciocchi, S. N. (2006). Epidemiology of cerebral concussion: The extent of the problem. In R. J. Echemendia (Ed.), *Sports neuropsychology: Assessment and management of traumatic brain injury* (pp. 107–123). New York: The Guilford Press.

McCrea, M. (2008). *Mild traumatic brain injury and postconcussion syndrome: The new evidence base for diagnosis and treatment.* New York: Oxford University Press.

McCrea, M., Kelly, J. P., Randolph, C., Kluge, J., Bartolic, E., Finn, G. et al. (1998). Standardized assessment of concussion (SAC): On-site mental status evaluation of the athlete. *Journal of Head Trauma Rehabilitation, 13*(2), 27–35.

McCrory, P., Johnston, K., Meeuwisse, W., Aubry, M., Cantu, R., Dvorak, J., et al. (2005). Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. *British Journal of Sports Medicine, 39*, 196–204.

Mittenberg, W., Canyock, E. M., Condit, D., & Patton, C. (2001). Treatment of post-concussion syndrome following mild head injury.

Journal of Clinical and Experimental Neuropsychology, 23(6), 829–836.

Mittenberg, W., Tremont, G., Zielinski, R. E. Fichera, S., & Rayls, K. (1996). Cognitive-behavioral prevention of postconcussion syndrome. *Archives of Clinical Neuropsychology, 11*(2), 139–145.

Spotlight Hypothesis

► Searchlight Hypothesis

SPPB

► Short Physical Performance Battery

SR-FAI

► Frenchay Activity Index

SRS

► Supervision Rating Scale

SRT

► Seashore Rhythm Test

Stages of Adjustment

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Synonyms

Coping

Definition

Stage models of psychological adjustment posit a series of predictable emotional reactions following catastrophic injury such as a spinal cord injury. Survival of catastrophic injury, with significant psychological implications arising from limitations in movement, sensation, and neurological functioning, led psychologists to consider universal sequential aspects of adjustment similar to those proposed by Kubler-Ross in her work on adjustment to terminal illness. These reactions became known as "stages of adjustment." Though most often considered in terms of spinal cord injury, the concept of stages of adjustment has been widely applied to many catastrophic injuries including amputation, brain injury, and diseases such as multiple sclerosis (MS).

Historical Background

Over the last half of the twentieth century, medical advances have allowed individuals to survive catastrophic injuries that previously proved fatal. The prototypical example is spinal cord injury. As early as 1950, Nagel described seven types of reactions he observed in 500 patients over a 5-year span. His observations included categories from anxiety and depression to psychotic reactions and normal reactions (see Frank, Van Valin, & Elliott, 1987 for full review of work describing stages of adjustment). Almost all stage models of adjustment postulated a depressive phase that followed an anxiety phase. Siller (1969), in an oft-quoted paper, suggested all individuals sustaining spinal cord injury became depressed, even if the disorder could not be readily discerned. Siller encouraged rehabilitation staff to help the individual find meaning in their depression.

Current Knowledge

Elliott and Frank (1996) reviewed empirical studies to determine if support existed for mandatory depression or stage models of adjustment after spinal cord injury. They concluded: "In sum, these data-based studies systematically demonstrated a lack of objective, empirical support for stage models in our understanding of psychological adjustment following SCI. We found the empirical evidence warranted a greater clinical and theoretical appreciation of the individual differences, environmental characteristics, and physiological parameters associated

with depression in this population." (Elliott & Frank, 1996, p. 817).

It is now clear that the concept of stages of adjustment following catastrophic injury is an overly simplistic model of psychological functioning. As noted by Elliott and Frank (1996), psychological responses to catastrophic injury are complex interactions of individual variables. The emotional reactions for which the stages tend to be named certainly do occur following traumatic illness or injury. Indeed, a significant minority of individuals experiencing spinal cord injury evidence clinically significant anxiety (25%) or depression (27%) (North, 1999). However, no psychological reaction or sequence of reactions is invariant. In fact, the best "stage theory" may be the one that proposes the fewest stages, allowing for the greatest amount of individual variation (Caplan & Shechter, 1987). While the notion of "stages of adjustment" may some clinical value – in that it offers to hope to those struggling with newly acquired injuries or illnesses that their distress may eventually abate – rigid adherence can compromise optimal, individualized clinical intervention.

Future Directions

Current models of post-catastrophic injury adjustment emphasize coping strategies and attitudes rather than the occurrence of predictable, universal stages of adjustment. In a longitudinal study of coping 10 years after injury, Pollard & Kennedy (2007) provided more evidence for model of post-injury adjustment based upon problem-focused-active strategies (Buckelew, Baumstark, Frank, & Hewett, 1990). The authors concluded "The fact that two-thirds of the year 10 sample showed no signs or symptoms of depression 10 years post injury adds to the already substantial body evidence disconfirming the necessary existence of a 'stage' of depression after SCI." The authors conclude that many individuals cope effectively with catastrophic injury (spinal cord injury) without psychopathology, but their choice of coping strategies is critical to their long-term outcome.

References and Readings

Buckelew, S. P., Baumstark, K. E., Frank, R. G., & Hewett, J. E. (1990). Adjustment following spinal cord injury. *Rehabilitation Psychology, 35*, 101–109.

Caplan, B., & Shechter, J. (1987). Denial and depression in disabling illness. In B. Caplan (Ed.), *Rehabilitation Psychology Desk Reference.* Aspen Systems Corp.

Elliott, T. R., & Frank, R. G. (1996). Depression following spinal cord injury. *Achieves of Physical Medicine and Rehabilitation, 77*, 816–923.

Frank, R. G., Van Vallin, P. H., & Elliott, T. R. (1987). Adjustment to spinal cord injury: A review of empirical and nonempirical studies. *Journal of Rehabilitation, 53*, 43–48.

Nagel, B. (1950). Psychiatric aspects of spinal cord injury. *American Journal of Psychiatry, 107*, 49–56.

North, T. T. (1999). The psychological effects of spinal cord injury. *Spinal Cord, 37*, 671–679.

Pollard, C., & Kennedy, P. (2007). A longitudinal analysis of emotional impact coping strategies and post-traumatic psychological growth following spinal cord injury: A 10-year review. *The British Journal of Health Psychology, 12*, 347–362.

Siller, J. (1969). Psychological situation of the disabled with spinal cord injury. *Rehabilitation Literature, 30*, 290–296.

Stammering

► Stuttering, Developmental

Standard Antipsychotics

► Antipsychotics

Standard Error of the Mean

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Definition

If a test is given to a certain sample, a mean score can be calculated. If we then give the test to a group of samples and calculate mean scores for each sample, the standard error of the mean can be thought of as the standard deviation of the mean scores in all of the samples. The population mean can never be precisely known. The standard error of the mean gives some indication of the variability possible in various samples drawn from that population. As with using samples to estimate population values, the larger the sample, the more likely that the mean of that sample would approximate the population mean and the smaller would be the standard error of the mean.

In clinical neuropsychology, as in all of psychological measurement, test scores are never perfectly reliable.

There is always some unreliability. Many times in clinical neuropsychology, there may be multiple publications of mean scores drawn from different samples. For example, the Rey Auditory Verbal Learning Test has published "normative" data from older samples, VA samples, outpatient samples, and inpatient psychiatric samples, among others. These samples vary in size, and frequently the mean scores and standard deviations vary across the samples as well. Being able to calculate the standard error of the mean allows us to estimate how different these sample means are in relation to the population mean and thereby tell us how confident we can be in comparing our obtained scores to the mean scores.

References and Readings

Nunnally, J., & Bernstein, I. (1994). *Psychometric theory* (3rd ed). New York: McGraw Hill.

Standard Progressive Matrices

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Synonyms

SPM; SPM-C

Description

The standard progressive matrices (SPM) are test measures better known as the Raven's progressive matrices (RPM), referring to the original version of the test as the SPM began in the 1940s, when alternate versions of the RPM were created (colored progressive matrices and advanced progressive matrices). A parallel version of the test, the SPM-P, was developed later (Raven et al., 2000). A new extended plus version (SPM+) has also been developed to address ceiling effects that began to be seen on the SPM beginning in adolescence (Pind, Gunnarsdottir, & Johannesson, 2003), ostensibly due to the worldwide increase in intellectual ability since the test's inception in 1938 (Raven et al., 2000). The SPM+ has been coupled with the Mill Hill Vocabulary Scales, a measure that was specifically designed for use with the SPM+, to provide a brief nonverbal and verbal screening measure of general ability for children ages 7–18 years of age.

For a full description of the test, historical background, psychometric data, and clinical uses, please refer to the entry for RPM.

Cross References

▶ Advanced Progressive Matrices
▶ Colored Progressive Matrices
▶ Raven Progressive Matrices

References and Readings

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
Pind, J., Gunnarsdottir, E. K., & Johannesson, H. S. (2003). Raven's standard progressive matrices: New school age norms and a study of the test's validity. *Personality and Individual Differences, 34*, 375–386.
Raven, J. C. (1938, 1996). Progressive Matrices: A perceptual test of intelligence. Individual Form. Oxford: Oxford Psychologists Press, Ltd.
Raven, J., Raven, J. C., & Court, H. H. (2000). Raven manual: Section 3. Standard Progressive Matrices. Oxford: Oxford Psychologists Press, Ltd.
Raven, J., Raven, J. C., & Court, J. H. (2003). *Manual for Raven's progressive matrices and vocabulary scales. Section 1: General overview.* San Antonio, TX: Harcourt Assessment.
Strauss, E., Sherman, E. M. S., & Spreen, O. (Eds.). (2006). *A compendium of neuropsychological tests* (3rd ed.). New York: Oxford University Press.

Standard Scores

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Synonyms

Normal scores; Standardized scores; T-scores; Z-scores

Definition

A metric used to compare the distance of an observation (e.g., test score) from the population mean (or sample mean) measured in standard deviation units. Standard scores enable us to determine at what point under the

Standard Scores. Table 1 Standard score conversion table and percentiles

T_a	IQ_b	SS_c	%ile	−z|+z	%ile	SS_c	IQ_b	T_a
≤20	≤55	≤1	≤0.1	≤3.00≥	≥99	≥19	≥145	≥80
21–23	56–59	2	<1	2.67–2.99	≥99	18	140–144	77–80
24–27	60–67	3	1	2.20–2.66	99	17	133–139	73–76
28–30	68–70	4	2	1.96–2.19	98	16	130–132	70–72
31	71–72	-	3	1.82–1.95	97	-	128–129	69
32–33	73–74	-	4	1.70–1.81	96	-	126–127	67–68
34	75–76	5	5	1.60–1.69	95	15	124–125	66
-	77	-	6	1.52–1.59	94	-	123	-
35	78	-	7	1.44–1.51	93	-	122	65
36	79	-	8	1.38–1.43	92	-	121	64
-	80	6	9	1.32–1.37	91	14	120	-
37	81	-	10	1.26–1.31	90	-	119	63
-	-	-	11	1.21–1.25	89	-	-	-
38	82	-	12	1.16–1.20	88	-	118	62
-	83	-	13	1.11–1.15	87	-	117	-
39	84	-	14	1.06–1.10	86	-	116	61
-	-	-	15	1.02–1.05	85	-	-	-
40	85	7	16	0.98–1.01	84	13	115	60
-	-	-	17	0.94–0.97	83	-	-	-
41	86	-	18	0.90–0.93	82	-	114	59
-	87	-	19	0.86–0.89	81	-	113	-
-	-	-	20	0.83–0.85	80	-	-	-
42	88	-	21	0.79–0.82	79	-	112	58
-	-	-	22	0.76–0.78	78	-	-	-
-	89	-	23	0.73–0.75	77	-	111	-
43	-	-	24	0.70–0.72	76	-	-	57
-	90	8	25	0.66–0.69	75	12	110	-
-	-	-	26	0.63–0.65	74	-	-	-
44	91	-	27	0.60–0.62	73	-	109	56
-	-	-	28	0.57–0.59	72	-	-	-
-	-	-	29	0.54–0.56	71	-	-	-
-	92	-	30	0.52–0.53	70	-	108	-
45	-	-	31	0.49–0.51	69	-	-	55
-	93	-	32	0.46–0.48	68	-	107	-
-	-	-	33	0.43–0.45	67	-	-	-
46	94	-	34	0.40–0.42	66	-	106	54
-	-	-	35	0.38–0.39	65	-	-	-
-	-	-	36	0.35–0.37	64	-	-	-
-	95	9	37	0.32–0.34	63	11	105	-
47	-	-	38	0.30–0.31	62	-	-	53
-	96	-	39	0.27–0.29	61	-	104	-
-	-	-	40	0.25–0.26	60	-	-	-
-	-	-	41	0.22–0.24	59	-	-	-
48	97	-	42	0.19–0.21	58	-	103	52
-	-	-	43	0.17–0.18	57	-	-	-
-	-	-	44	0.14–0.16	56	-	-	-
-	98	-	45	0.12–0.13	55	-	102	-
49	-	-	46	0.09–0.11	54	-	-	51
-	99	-	47	0.07–0.08	53	-	101	-
-	-	-	48	0.04–0.06	52	-	-	-
-	-	-	49	0.02–0.03	51	-	-	-
50	100	10	50	0.00–0.01	50	10	100	50

[a]M = 50, SD = 10; [b]M = 100, SD = 15; [c]M = 3, SD = 1

Standard Scores. Table 2 Common ability classifications for standard scores and percentiles

Classification	Z	T	SS	IQ	%ile
Very Superior	≥ 2.00	≥ 70	≥ 16	≥ 130	≥ 98
Superior	1.37 to 1.95	64 to 69	14 to 15	120 to 129	91 to 97
High average	0.72 to 1.31	57 to 63	13	110 to 119	76 to 90
Average	−0.66 to 0.69	44 to 56	8 to 12	90 to 109	25 to 75
Low average	−1.26 to −0.70	37 to 43	7	80 to 89	10 to 24
Borderline/Unusually Low	−1.82 to −0.32	30 to 36	5 to 6	70 to 79	3 to 9
Extremely low	≤ −2.00	≤ 29	≤ 4	≤ 69	≤ 2

bell curve (i.e., frequency distribution) a given score falls relative to the comparison population/sample.

Current Knowledge

Type of Standard Scores: Standard scores can be expressed on a variety of scales. The most commonly used scales in neuropsychology are z-scores (M = 0, SD = 1), T-Scores (M = 50, SD = 10), IQ scores (M = 100, SD = 15), and scaled scores (M = 10, SD = 1). Standard scores can easily be converted from one scale to another by using Equation 1:

$$((\underline{X} - \underline{M}^1)/\underline{SD}^1) \times \underline{SD}^2 + \underline{M}^2) \quad (1)$$

where: X = the score you wish to change

M^1 = the mean of the score you wish to change from

SD^1 = the standard deviation of the score you wish to change from

M^2 = the mean of the score you wish to change towards

SD^2 = the standard deviation of the score you wish to change towards.

For example, to convert IQ scores to scaled scores, the following formula can be used: $((\underline{X} - 100)/15) \times 3 + 10)$. For convenience, a conversion table for common standard scores is presented in Table 1.

Interpretation of Standard Scores: Standard scores are used in norm-referenced assessment to compare one person's performance on a test to that of a group of persons with similar characteristics (e.g., age, education, gender, or ethnicity). One of the key features of standard scores is that raw scores from multiple tests can be converted to a common metric, which facilitates comparison across multiple measures. However, it is important to note that comparison of standard scores across multiple measures is only recommended (a) when the raw score distributions for the tests being compared are approximately normal, and (b) when the standard scores are derived from similar samples (or preferably the same sample).

Standard scores are routinely interpreted based on cut-off values that reflect the percentage of scores that fall at or below a given test score. For clinical interpretation, ability classifications are allocated to the range of scores that fall between the upper and lower cut-off scores. Although various classification systems have been proposed, one common ability classification system is presented in Table 2.

Calculation of Standard Scores: Standard scores are routinely provided for the vast majority of published tests by way of "raw score to standard score" look-up tables. However, in the absence of these tables, standard scores may be generated by calculating a *z-score* using the observed raw score and the mean and standard deviation from a comparison population/sample. A z-score is calculated by subtracting the population/sample mean (M) from an individual's raw score (X) and then dividing the difference by the population/sample standard deviation (SD): z = [X-M]/SD. A z-score can then be converted to any other standard score using Equation 1 or Table 1. However, it is important to note that this method of generating standard scores relies on the assumption that there is a normal distribution of scores in the comparison population/sample. In the absence of a normal distribution (i.e., positive or negative skew), we cannot be certain that the standard score is accurate and is therefore not recommended. The more highly skewed the distribution of scores in a population, the less accurate we expect the standard score to be.

Cross References

- ▶ Normal Curve
- ▶ Percentiles
- ▶ Standardized Tests

References and Readings

Slick, D. J. (2006). Psychometrics in neuropsychological assessment. In E. Strauss, E. M. S. Sherman, & O. Spreen (Eds.), *A compendium of neuropsychological tests: Administration, norms, and commentary* (pp. 3–43). New York: Oxford University Press.

Standardized Battery

▶ Fixed Battery

Standardized Measure

▶ Standardized Tests

Standardized Scores

▶ Standard Scores

Standardized Tests

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Definition

Standardized testing refers to the uniform administration and scoring of a test (as set forth by the author or publisher of the test) to assure that the results are comparable to the sample upon which the scores are based.

Historical Background

There is some evidence that standardized testing was used several thousand years ago in China for military purposes. However, for the purposes of psychology, the beginnings of standardized testing can be traced to the early and middle 1800s in France with the assessment of intelligence by Itard and Seguin, who developed some of the initial ideas concerning the education of children and adults with mental retardation and individual differences in intelligence. Several developments in the assessment of individual differences can be attributed to Wilhelm Wundt and his study of psychophysics. Through his interactions with Sir Francis Galton, James McKeen Cattell developed the use of "mental tests" and was one of the individuals whose work helped assure that the idea of mental testing was firmly rooted in North America. For a more thorough review of standardized testing history, and in particular intelligence tests, see Tulsky, Saklofske, and Ricker (2003).

Current Knowledge

Standardization specifically refers to the published instructions for administration of a test, the use of standardized test stimuli in the administration of the test, and the normative population to which the test results are compared. The normative population consists of the individuals on whom the test was administered and data was collected, usually stratified by certain demographic characteristics, such as age, gender, education status, and, sometimes, ethnicity, which individual test scores can be compared to. Thus, the normative data allows comparison of a single administration of a test to a similar group as that of the examinee. Test developers, whether the test is for publication or research, have a set of instructions and conditions under which the test was developed. For example, the Wechsler Intelligence tests have well-standardized instructions, including specific verbiage, placement of the testing materials, extra information to be given to the examinee, time limits, environmental conditions, etc. These administration methods are supposedly the same conditions under which the test was administered when the normative data was collected.

To the degree that normative conditions are met, the individual test data are comparable to the normative sample. For example, almost all neuropsychological tests indicate that tests should be administered under controlled conditions with little environmental distracters. If a test such as digit span – in which a string of numbers are read out to an examinee and he or she is asked to repeat them verbatim – were administered in a quiet environment with few external distracters in the standardized manner, then the results are likely comparable to similar normative data. If, however, the test was administered during a hockey game in a stadium, the results would likely vary a great deal from the normative data, making the results less comparable and compromising the meaning of normative data greatly. Lezak, Howieson, and Loring (2004) state "highly standardized test

administration is necessary when using norms of tests that have a fine-graded and statistically well standardized scoring system, such as the Wechsler intelligence tests. By exposing each patient to nearly identical situations, the standardization of testing procedures also enables the examiner to discover the individual characteristics of each patient's responses." Thus, to the degree that standardization is violated the ability of the obtained data to compare to the standardization normative data is compromised.

There is an approach in neuropsychological evaluation called the process approach that "stressed understanding each patient's unique cognitive and behavioral strengths and weaknesses at the expense of adhering to standardized testing practices" (Smith, Ivink, & Lucas, 2008). This "process" approach, which was an early development in neuropsychology, specifically adapts tests to the needs or conditions for individuals in order to attempt better understanding of how a patient completes a task rather than deriving a sum and comparing it to a normative group. The Boston Diagnostic Aphasia Exam is an example of this approach. There have been efforts made to codify the process by which a patient completes a task, such as in the Delis Kaplan Executive Functioning System or the NEPSY-II, which allows for a combination of a process approach and comparison to normative data.

A commonly used approach, which is a combination of standardized testing and the process approach, is testing the limits. Testing the limits allows for standard administration first, and second, alteration of the standardized administration, such as allowing more time or giving more instruction. This approach allows for a valid comparison to the standardized normative group and examination of the process by which the examinee completes a task, as well as allowing some flexibility in the administration for individuals who may not be adequately able to complete a task in the standardized manner. For example, a patient with an acute brain injury in a rehabilitation setting may be able to complete a task such as block design from the Wechsler Adult Intelligence Scales, which requires both visuospatial skills and speed, but cannot complete it in the time allotted due to processing speed difficulties. One may extend the time so that the patient can have as much time as he or she needs, which allows for a differentiation to be made between a difficulty with visuospatial processing and slow processing speed. Performance with the time limits may appear that the patient has poor visuospatial functioning leading to a false conclusion of visuospatial or constructional deficits, when in fact the patient may simply process information slowly, but have adequate visuospatial functioning. Of course, to the degree that testing the limits violates the standardized administration, the assessment data are less comparable to the normative group.

Standardization of tests is an important aspect of the practice of neuropsychology and is closely related to reliability of a test. The axiom that a test's reliability places an upper limit on validity is the issue of standardization. Of course, there are tests designed to measure states that are variable, such as depressed mood, that may intentionally have low test–retest reliability for example, but by and large neuropsychological tests of intelligence, attention, processing speed, visuospatial functioning, new learning and memory, executive functioning, personality, and others are assumed to be reasonably stable. For example, Slick (2006) states that "The process of neuropsychological assessment depends to a large extent on the reliability and validity of neuropsychological tests." Thus, validity, or the ability of a test to measure what it purports to or predict what it purports to, is necessary for a test and reliability of that test constricts the validity. Standardization allows for reliability, which allows neuropsychologists to eventually compare to normative data and make more data-driven diagnostic decisions and treatment recommendations. Furthermore, standardization facilitates the conduct of clinical research.

Vanderploeg (2000) suggests several principles that apply to testing in a multiple hurdles kind of format, which provides rules for application of standardization and when to alter it as well as the implications of altering standardization. At the core of these recommendations is to match the standardized administration as closely as possible.

Principle 1: Administer the test in the standardized manner; however, some examinees may have limitations that make standardized administration impossible. Refer to 2
Principle 2: The conditions necessary for each examinee to meet standardization are important. Thus, amplifying instructions by using pauses or speaking more slowly may be necessary to assure that the examinee understands the content and meets the standardized conditions. Alternate ways of responding may be necessary, such as pointing, if the examinee is not able to adequately communicate vocally. For example, if the examiner does not speak clearly or loudly enough for an examinee, then the examinee's performance on a test may be a reflection of random responding, because she did not understand the instructions, rather than true variance associated with the test.
Principle 3: Minimize environmental sources of distraction that may lead to error in the test data.

Principle 4: Ensure that the examinee is adequately alert and aroused for testing. For example, if an examinee is taking a high dose of pain medication, which tends to slow processing speed, finding the time when the examinee is most alert or using less medication is important to obtain valid results.
Principle 5: Present all materials midline, unless there is a visual field cut. If the examiner suspects that midline presentation may impair performance, such as in the case of visual neglect, complete the standardized administration then change the presentation as part of testing the limits.
Principle 6: For timed tasks, make sure to time and record all responses. The examiner can then use untimed administration as a part of testing the limits. Both the sources of information can be useful.
Principle 7: Provide enough help to assure that the examinee is engaged in the process without artificially enhancing performance.
Principle 8: Periodically review the examiner's manual to assure accurate administration.
Principle 9: There is a difference between assessment and testing. If the referral question conflicts with standardized administration for testing, then the referral question should take precedence and standardization should be altered. Similar to the above discussion, to the extent that the standardization is altered, the normative data are less useful and the performance becomes more of a behavioral observation than a standardized test.

Cross References

► Testing the Limits

References and Readings

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
Slick, D. J. (2006). Psychometrics in neuropsychological assessment. In E. Strauss, E. M. S. Sherman, & O. Spreen (Eds.), *A compendium of neuropsychological tests* (pp. 3–31). New York: Oxford University Press.
Smith, G. E., Ivnik, R. J., & Lucas, J. (2008). Assessment techniques, test batteries, norms and methodological approaches. In J. E. Morgan & J. H. Ricker (Eds.), *Textbook of clinical neuropsychology* (pp. 38–57). New York: Taylor & Francis.
Tulsky, D. S., Saklofske, D. H., & Ricker, J. (2003). Historical overview of intelligence and memory: Factors influencing the Wechsler scales. In D. S. Tulsky, D. H. Saklofske, G. J. Chelune, R. K. Heaton, R. J. Ivnik, R. Bornstein, A. Prifitera, & M. F. Leadbetter (Eds.), *Clinical interpretation of the WAIS-III and WMS-III*. San Diego: Academic.
Vanderploeg, R. D. (2000). *Clinicians guide to neuropsychological assessment* (2nd ed.). Mahway: Erlbaum.

Standards of Proof

► Burden of Proof

Standing Tremor

► Orthostatic Tremor

Stanford HAQ

► Health Assessment Questionnaire

Stanford–Binet 5

► Stanford–Binet Intelligence Scales and Revised Versions

Stanford–Binet Intelligence Scales and Revised Versions

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Synonyms

SB-5; Stanford–Binet 5

Description

The Stanford–Binet Intelligence Scales, Fifth Edition (SB-5) is the latest (2003) version of a well-established individually administered test of intelligence and cognitive

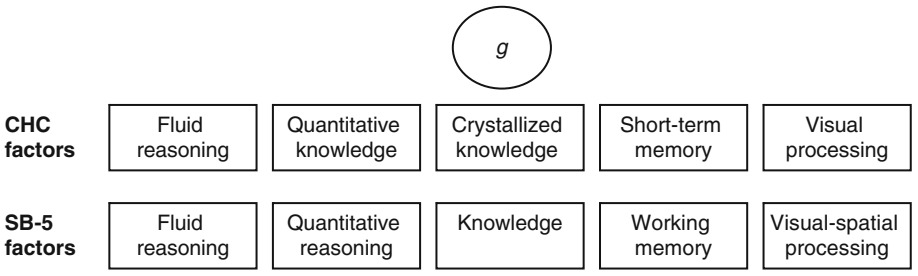
abilities. The SB-5 measures five factors from the Cattell–Horn–Carroll (CHC) (Carroll, 1993) theory of intellectual abilities including fluid reasoning, quantitative reasoning, crystallized knowledge, short-term memory, and visual processing. Each factor is assessed in both the verbal and nonverbal domains resulting in a total of ten subtests that yield factor index scores for each of the five CHC factors. The SB-5 also provides composite scores for Verbal IQ, Nonverbal IQ, and Full Scale IQ, which would correspond to the CHC's concept of general intelligence, termed *g*, residing at the highest level of hierarchy. Verbal (Vocabulary) and nonverbal (Object Series/Matrices) routing subtests determine the examinee's start point on the remaining subtests in each domain, and can be administered alone to generate an Abbreviated Battery IQ (Figure 1 and Table 1).

While the SB-5 routing subtests reflect a typical point scale format in which all items of a given type are presented continuously, the remaining subtests are organized by level of difficulty. The examinee rotates through "testlets" of three to six items per subtest at each level of the test so that each factor is measured at increasing difficulty levels. Testing is discontinued on a subtest-by-subtest basis when the examinee achieves a ceiling.

The SB-5 was normed on a stratified nationally representative sample of 4,800 individuals aged 2–85+ years (norms extend to 89 years 11 months). All items were reviewed for possible bias. Full Scale IQ scores range from 40 to 160 and supplemental test materials provide a means to calculate IQ scores from 10 to 225. Administration of the complete battery requires 60–75 min. An Abbreviated Battery IQ can be obtained in 15–20 min. Separate administration of either the Verbal or Nonverbal IQ section requires approximately 30 min. The test may be scored by hand, but the use of the SB-5 Scoring Pro software may be preferred due to the number and complexity of scores that can be calculated. Raw scores are converted to scaled scores, factor index scores, composite scores, percentile ranks, and age equivalents. All composite and factors scores have a mean of 100 and a standard deviation of 15 (in contrast to the standard deviation of 16 for scores from previous versions), which facilitates easier comparison with other cognitive and achievement measures. Subtest scores have a mean of 10 and a standard deviation of 3 (in contrast to a mean of 50 and a standard deviation of 8 for the Fourth Edition). In addition, change sensitive scores may be calculated to compare changes in an individual's ability level over time regardless of the IQ score obtained. Normative score difference data is provided to facilitate analysis of differences between and among the IQ and factor index scores, differences between individual subtest scores and the examinee's average subtest score, differences between pairs of subtest scores, and scatter among verbal and nonverbal subtests. Administration requires a highly trained examiner. The design of the SB-5 requires ample time for learning administration and scoring procedures even for experienced examiners.

Historical Background

The 1905 *Binet–Simon Intelligence Scale* was collaboratively designed by French psychologist, Alfred Binet, and physician, Theodore Simon, as a reliable system for identifying children aged 3–13 years with mental retardation. The scale consisted of 30 problems assessing language, auditory and visual processing, learning and memory, and judgment and problem solving. Items were presented in the order of increasing difficulty based on empirical performance data from typically developing children, as well as some cognitively impaired children and adults. The scale did not provide a precise method for determining overall scores. Binet and Simon's approach to assessing intelligence in a clinical interview format via complex tasks (i.e., tasks including both mental and physical components) represented a significant advance over the historical practice of measuring mental abilities using isolated laboratory tasks that were primarily sensory.



Stanford–Binet Intelligence Scales and Revised Versions. Figure 1 CHC factors measured by the SB-5

Stanford–Binet Intelligence Scales and Revised Versions. Table 1 Verbal and nonverbal tasks assessing SB-5 factors

	Verbal tasks	Nonverbal tasks
Fluid reasoning	• Early reasoning – *describe what is happening in a picture* • Verbal absurdities – *explain what is silly or impossible about statement read by the examiner* • Verbal analogies – *solve word problems orally*	• Object series/matrices (nonverbal routing subtest) – *select the best answer to complete a series or matrix*
Quantitative reasoning	• Quantitative reasoning, verbal – *respond verbally to problems presented orally and visually*	• Quantitative reasoning, nonverbal – *solve a problem and point to response*
Knowledge	• Vocabulary (verbal routing subtest) – *initial items require a pointing response, while later items progress from one-word answers to clear definitions*	• Procedural knowledge – *show what you typically do with a pictured object* • Picture absurdities – *point to and tell what is silly or impossible about a picture*
Working memory	• Memory for sentences – *recall orally resented sentences* • Last word – *listen to a question, answer the question, and then recall last word in the question*	• Delayed response – *remember which cup hides an object* • Block span – *tap blocks in the same sequence demonstrated by the examiner*
Visual-spatial processing	• Position and direction – *place a block on the part of the picture specified by the examiner or respond verbally to oral questions*	• Form board – *place shapes back into form board after seeing the examiner remove them* • Form patterns – *use blue plastic pieces to assemble a pictured stimulus*

Multiple revisions of the 1905 Binet–Simon Scale evidenced the international popularity of the instrument. In the USA, a revision published by Henry Goddard at the Vineland Training School facilitated acceptance of IQ testing among medical professionals. Another revision, the *Kuhlman–Binet* which extended the scale down to the age of 3 months marked the first attempt to assess intelligence in infants and preschool children. A 1908 revision by Binet provided a method to calculate an overall mental-level score based on the age of normal children, whose performance the examinee equaled. A second revision in 1911, the year of Binet's death, added items at higher levels to extend the test through adulthood. Both of Binet's revisions maintained the fundamental content and design of the test.

Binet's legacy was carried out in the USA by Lewis Terman. After the publication of a provisional revision developed with H.G. Childs in 1912, Terman published the *Stanford Revision and Extension of the Binet–Simon Intelligence Scale* in 1916. Terman sought to maintain Binet's age-scale format while addressing the tendency of original versions to overestimate the ability of young children and underestimate the ability of older children. Terman utilized a sample of more than 2,300 individuals from early childhood to mid-adolescence to revise old items and introduce new ones. He assigned items to a given age level when most children that age in the sample were able to pass them. The test yielded a derived IQ based on mental age divided by chronological age and provided detailed instructions for administration and scoring. The dominance of Terman's revision over other contemporaneous versions likely reflects the influence of his methodological rigor.

In 1937, Terman collaborated with Maud Merril to publish a parallel forms revision of the SB (Forms L & M). While both forms remained organized into functional age levels, the revision offered improved standardization, an extended floor and ceiling, broadened age range (18 months to 18 years), and the addition of an alternate form. While the sample size (3,184) and representativeness were improved over the 1916 version, the standardization sample was criticized as nonrepresentative, because the students represented higher than average SES were from primarily urban areas, and were all native-born whites. Other critics of the 1937 revision noted its failure to assess separate abilities, the inefficiency of all or none scoring, overemphasis on verbal abilities, and an inadequate ceiling. Additionally, the Wechsler scales' point-scale format led to criticism of the SB's continued use of an age-scale format.

In 1960, after Terman's death, Merril directed the publication of the SB L-M which created one test form for individuals aged 2–18 years from the most discriminating items of the separate L and M forms. The L-M introduced a deviation IQ with a mean of 100 and SD of 16. The 1960 revision was criticized for its lack of

restandardization, poorly constructed deviation IQ, and continued overemphasis on verbal abilities. In addition, the L-M provided only one overall score in contrast to the overall scores and individual subtest scores provided by the Wechsler scales.

Robert Thorndike directed a normative update of the L-M published in 1972 based on a sample of approximately 2,100 individuals that was more representative of the US population than the 1937 sample. The content remained unchanged from the 1960 L-M version.

The Stanford–Binet Intelligence Scales, Fourth Edition (Thorndike, Hagen & Sattler, 1986) represented a significant departure from earlier versions. The SB-4 answered critics' calls for a point-scale format and utilized a hierarchical four-factor model of general intelligence (verbal reasoning, abstract/visual reasoning, quantitative reasoning, and short-term memory). In the absence of an age-scale format, performance on the Vocabulary subtest was combined with chronological age to individualize subtest start points. The complete battery consisted of 15 subtests of which 8–13 were administered depending on the examinee's age. Abbreviated batteries could consist of two, four, or six subtests. Improvements included a large representative standardization sample, a broader age range from 2 to 23 years, and the inclusion of individual subtest scores (mean = 50, standard deviation = 8). The composite scores for each factor and Full Scale IQ were renamed Standard Age Scores, but continued to have a mean of 100 and standard deviation of 16. Limitations of the SB-4 included difficulty scoring responses, longer administration time than other commonly administered tests of intelligence, lack of comparable battery throughout age ranges covered, variable range of scores at different age levels, and limited support for four factors.

The Fifth Edition of the Stanford–Binet blends the use of routing subtests in the point-scale format of the 1986 edition along with the functional level design of the 1916 and 1960 editions. The SB-5 includes several returning subtests (e.g., Picture Absurdities, Matrices, Vocabulary, Memory for Sentences, Quantitative Reasoning, and Verbal Absurdities), as well as several new tasks. Specific improvements noted in the test manual include extensive high-end items to measure highest levels of gifted performance, improved low-end items for assessing young children or lower functioning or impaired children and adults, reintroduction of appealing toys, manipulatives, and colorful artwork and new tasks measuring working memory that can be used to assess adults and the elderly. In addition, the SB-5 includes a robust nonverbal domain which answers criticism that earlier versions overemphasized verbal ability. The scales for composite and subtest scores have been revised to be consistent with other commonly used assessment tools (i.e., mean of 100 and standard deviation of 15 or mean of 10 and standard deviation of 3). The SB-5 represents an attempt to maintain hallmark components (e.g., emphasis on developmental differences across age ranges) of the SB, while also utilizing up-to-date psychometric and clinically efficient techniques.

Psychometric Data

Development of the SB-5 took place over a 5-year period and included planning, pilot studies, a tryout edition, a standardization edition, and the final published edition.

Overall, the SB-5 exhibits excellent reliability. Mean split-half reliability for all 10 subtests ranges from 0.84 to 0.89 across all age groups. Test–retest reliability for most subtests ranges from 0.75 to 0.93 although some subtests have coefficients below 0.75 (e.g., 0.66 for nonverbal Working Memory in 21–59 age group). Mean reliability coefficients (sum of multiple tests) were above 0.95 for Full Scale, Verbal, and Nonverbal IQ scores, while test–retest reliability was 0.89 or above for these composite scores. Reliability also was acceptable for the Abbreviated Battery IQ (sum of multiple tests and test-retest from 0.84 to 0.91) and factor index scores (0.82–0.92).

The technical manual provides ample evidence of validity including professional judgment, empirical item analysis, and correlations with prior versions of the SB, Wechsler Scales, and Woodcock-Johnson III Tests of Cognitive Abilities. Additional discussion of validity includes studies of special groups, age trends, intercorrelations between subtest, factor index, and IQ scores, and confirmatory factor analysis.

Test design was informed by modern item response theory including the calibration of items and adaptive testing through the use of routing subtests.

Clinical Uses

The SB-5 Manual indicates that the test can be used as a part of diagnostic assessment for developmental disabilities, clinical and neuropsychological assessment, early childhood assessment, special education evaluation, and

adult social security and worker's compensation evaluations. The SB-5 is an appropriate tool for use in cases that may require regular assessment of cognitive abilities due to its excellent test-retest reliability and minimal practice effects. The novelty of the test tasks and variation in item types across difficulty levels also may be advantageous in cases requiring repeated testing.

Administration of a shortened battery may be most appropriate in specific clinical cases. For example, the Abbreviated Battery IQ can be used as an estimate of general intelligence when a battery of neuropsychological tests is used to assess other specific domains of cognitive functioning. However, as with other brief assessments of IQ, caution is warranted as the Abbreviated IQ may significantly misrepresent cognitive ability in some cases.

According to the SB-5 Manual, the verbal section may be used in isolation when the emphasis is on the oral presentation of verbal items. Such an administration may be appropriate for individuals with orthopedic or visual impairment. Similarly, the Manual indicates that the nonverbal section of the test may be used alone to assess individuals with communication disorders, hearing impairments, deafness, autism spectrum disorders, specific learning disabilities, limited English-language background, TBI, or other conditions where linguistic ability is limited such as aphasia or stroke. The test authors note that the SB-5 is unique in that the Nonverbal IQ covers all five cognitive factors. However, some of the SB-5 nonverbal tests present significant language processing demands prompting the suggestion that the nonverbal section is more appropriately viewed as "language-reduced."

For the assessment of specific learning disabilities, the SB-5 provides a linking sample with the Woodcock Johnson-III Tests of Achievement that allows intelligence–achievement comparisons for individuals from school-age children to 19-year-olds. The SB-5 also was co-normed with Bender Visual Motor Gestalt Test, Second Edition.

The SB-5 is essentially untimed; therefore, it may provide a more accurate reflection of other cognitive abilities for individuals with processing speed deficits than other intelligence batteries that significantly weight performance speed. However, the lack of processing speed measures has been cited as one of the key factors contributing to the limited use of the SB-5 by neuropsychologists.

The extension of the SB-5 to 85+ years of age and the addition of memory tasks emphasizing working memory appropriate for use with adults and the elderly make the test newly relevant for neuropsychologists serving these populations. The manual reports special validity case studies with adult populations including individuals diagnosed with Alzheimer's or dementia.

In evaluating the clinical utility of the SB-5, caution should be used in extending research findings related to prior versions due to the significant changes in test content and design reflected in the newest version. Equal caution is warranted in any attempt to interpret score differences between the Fifth Edition and prior versions. Even on subtests that are similarly named, changes in structure and score scales impede meaningful comparisons. For example, the Picture Absurdities task previously has been used in assessment for dementia; however, it is now one component that contributes to the Nonverbal Knowledge score rather than a discretely scored subtest.

In terms of clinical limitations, despite providing a fixed battery across all ages, variation in tasks used to measure a construct at different levels on the SB-5 complicates interpretation. For example, an examinee's Nonverbal Knowledge score may reflect performance on form board tasks, procedural knowledge tasks, picture absurdities, or all the three. The validity of the SB-5's factor structure has been questioned; for example, independent studies of the SB-5 factor structure fail to find support for five separate factors for any age group (Canivez, 2008; DiStefano & Dombrowski, 2006). The high intercorrelations (0.89–0.98) reported among the factors limit the validity of factor-based interpretation. Evidence weakly supports verbal and nonverbal domains until the age of 10 years only. Overall, factor-analytic studies suggest that the SB-5 is best viewed as a measure of a single factor – general intelligence. Therefore, interpretations are most robustly made at the composite, rather than the subtest or factor level.

Research evidence available thus far suggests that the SB-5 is a solid measure of general intelligence and may be particularly useful with special populations including intellectually gifted, very young, and low-functioning individuals. While the SB-5 reflects many changes designed to increase its utility for clinical neuropsychologists, additional data is needed regarding the use of the SB-5 with most of the clinical populations commonly seen by neuropsychologists.

Cross References

► Bender Visual-Motor Gestalt Test II
► Crystallized Intelligence

- ▶ Factor Analysis
- ▶ Intelligence
- ▶ Intelligence Quotient
- ▶ Metal Age
- ▶ Wechsler Adult Intelligence Test (All Versions)
- ▶ Wechsler Intelligence Scale for Children
- ▶ Wechsler Preschool and Primary Scale of Intelligence
- ▶ Woodcock-Johnson Cognitive-Achievement Battery

References and Readings

Canivez, G. L. (2008). Orthogonal higher order factor structure of the Stanford-Binet intelligence scales-fifth edition for children and adolescents. *School Psychology Quarterly, 23*(4), 533–541.

Carroll, J. B. (1993). *Human cognitive abilities: A survey of factor-analytic studies.* New York: Cambridge University Press.

Coolican, J., Bryson, S. E., & Zwaigenbaum, L. (2007). Brief report: Data on the Stanford-Binet intelligence scales (5th ed.) in children with autism spectrum disorder. *Journal of Autism and Developmental Disabilities, 38*(1), 190–197.

DiStefano, C., & Dombrowski, S. C. (2006). Investigating the theoretical structure of the Stanford-Binet-fifth edition. *Journal of Psychoeducational Assessment, 24*(2), 123–136.

Flanagan, D. P., & Harrison, P. L. (Eds.). (2005). *Contemporary Intellectual Assessment.* New York: Guilford Press.

Kamphaus, R. W., & Kroncke, A. P. (2003). "Back to the future" of the Stanford-Binet intelligence scales. In G. Goldstein, & S. R. Beers (Eds.), *Comprehensive handbook of psychological assessment, intellectual and neuropsychological assessment* (Vol. 1., pp. 77–86). Hoboken, New Jersey: Wiley and Sons.

Leffard, S. A., Miller, J. A., Bernstein, J., DeMann, J. J., Mangis, H. A., & McCoy, E. L. B. (2006). Substantive validity of working memory measures in major cognitive test batteries for children. *Applied Neuropsychology, 13*(4), 230–241.

Roid, G. H. (2003). *Stanford-Binet intelligence scales, fifth edition, technical manual.* Rolling Meadows, IL: Riverside Publishing.

Roid, G. H., & Barram, R. A. (2004). *Essentials of Stanford-Binet intelligence scales (SB5) assessment.* Hoboken, New Jersey: Wiley and Sons.

Sattler, J. M. (2008). Stanford-Binet intelligence scales, fifth edition (SB5). In *Assessment of children: Cognitive foundations* (5th ed.). La Mesa, CA: Jerome M. Sattler, Publisher, Inc.

Strauss, E., Sherman, E., & Spreen, O. (2006). General cognitive functioning and assessment of premorbid intelligence. In *A compendium of neuropsychological tests: Administration, norms, and commentary.* New York: Oxford University Press.

Thorndike, R. L., Hagen, E. P., & Sattler, S. M. (1986). The Stanford-Binet Intelligence Scale: Fourth Edition. Chicago: Riverside.

Static Encephalopathy

- ▶ Cerebral Palsy

Statistical Significance

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Definition

Researchers frequently wish to evaluate whether an observed finding is related to chance or to some systematic process. Typically, statistical significance is described in terms of probability. For example, to say that an observed group difference is statistically significant at the $p < 0.01$ level means that a difference that is large is likely to be found by chance one time in one hundred. Therefore, it is likely that the observed group difference was due to some systematic effect related to membership in the group. Statistical significance can also be applied to correlation coefficients. There the difference is said to be different from zero. In each of these cases, the statistical test is a mathematical examination of the hypothesis.

Current Knowledge

In traditional hypothesis testing, the null hypothesis is that which is put to the test. The null hypothesis is that the mean difference between groups is zero or that the value of the correlation coefficient is zero. If the difference is found to be statistically significant, we say that we reject the null hypothesis. In testing the difference between group means, the likelihood of statistical significance is increased when the variability of scores is decreased, when the observed difference in means is increased, and when the sample size is increased.

Cross References

- ▶ ANOVA
- ▶ Correlation Coefficient
- ▶ MANOVA

References and Readings

Frick, R. W. (1996). The appropriate use of null hypothesis testing. *Psychological Methods, 1*, 379–390.

Hagen, R. L. (1997). In praise of the null hypothesis statistical test. *American Psychologist, 52*, 15–24.

Rodgers, J. L. (2010). The epistemology of mathematical and statistical modeling: A quiet methodological revolution. *American Psychologist, 65*, 1–12.

Status Epilepticus

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Synonyms

L'état de mal épileptique

Definition

Status epilepticus was defined by an International League Against Epilepsy (ILAE) task force as "a seizure that shows no clinical signs of arresting after a duration encompassing the great majority of seizures of that type in most patients *or* recurrent seizures without interictal resumption of baseline central nervous system function" (Blume et al., 2001).

Historical Background

Status epilepticus (SE), sometimes referred to as the maximum expression of epilepsy, is a medical emergency requiring prompt pharmacological therapy. It was formally defined by Gastaut at the 1962 Marseilles Colloquium on SE as "epileptic seizures which are so frequently repeated and prolonged as to create a fixed and enduring epileptic condition." In recent decades, the generally accepted temporal criterion or minimum duration of seizures used to define SE has shrunk from 30 to 5 min, so that SE recently was defined as continuous seizure activity or rapidly recurrent seizures without resumption of consciousness for $>$ 5 min. This definition takes into account that seizures in adults that continue for $\geq$ 5 min are likely to continue for $\geq$ 30 min and seizures that have continued for $\geq$ 30 min are more difficult to treat. By way of contrast, the typical single generalized convulsive seizure lasts for approximately 1 min, with a standard deviation approaching 15 s.

However, some seizures defined as SE by the 5-min duration criterion will resolve spontaneously. Therefore, Wasterlain and Treiman (2006) employ the term *impending SE* to refer to "An acute epileptic condition characterized by continuous generalized convulsive seizures for at least 5 minutes, or by continuous nonconvulsive seizures (clinical or electrographic) or focal seizures for at least 15 minutes, or by two seizures without full recovery of consciousness between them." This definition of impending SE makes clear that SE is not a single entity. Moreover, it is a *clinical* or operational definition intended for physicians making diagnostic and treatment decisions and emphasizes urgency in preventing possible morbidity, mortality, and refractory SE. It should be noted that this clinical definition applies to patients $>$ 5 years old. One reason for this age-restricted aspect of the definition is that febrile seizures are common and may last $>$ 5 min in very young children without necessarily evolving into SE. Thus, a traditional definition of SE as seizures lasting $>$ 30 min often is adhered to in the treatment of children $\leq$ 5.

In addition, Wasterlain and Treiman (2006) proposed that a second, more traditional, and ironclad definition of SE is appropriate for the work of epidemiologists and other researchers. In this context, SE is defined as "An acute epileptic condition characterized by continuous seizures (partial or generalized, convulsive or nonconvulsive) for at least 30 minutes, or by 30 minutes of intermittent seizures without full recovery of consciousness between seizures."

Current Knowledge

Incidence

A population-based epidemiological study of SE, conducted in the Richmond, VA metropolitan area of the U.S., adhered to the 30-min duration criterion in establishing the incidence of SE. The absolute incidence of SE in that investigation was 41 per 100,000 population. Other studies, including those of a retrospective nature, have determined an incidence from 8 to 18 per 100,000 population. This difference can be explained in part by the fact that SE was more common in the large African-American population in the Richmond study, with incidences of 19/100,000 versus 57/100,000 in whites and African-Americans, respectively. Thus, the influence of socioeconomic and/or cultural factors on SE remains to be determined. Because of the difficulty in identifying all cases of SE, the overall incidence figure from the Richmond study was thought to be an underestimate. On the basis of the statistically corrected incidence, it has been estimated that 100,000 to 150,000 individuals experience at least one

episode of SE per year in the USA. Across the world, a conservative estimate suggested 3.5 million people experience SE per year, with almost one million associated deaths (Wasterlain & Treiman, 2006).

Age Distribution

The age distribution of SE is U-shaped. Infants less than 1 year old have the highest incidence of SE and it is next most common in those more than 60 years old. Because the US elderly population is increasing, SE will become more common. It is notable that among both children and adults, about 10% of patients seeking medical attention for a first unprovoked seizure present in SE. Children have the highest recurrence rate of SE, but in general recurrence is highest in patients with progressive symptomatic SE.

Etiology

Overall, about 50% of those who develop SE have no previous history of epilepsy and among the elderly about 70% have no such history. Thus, SE occurs in patients with acute systemic and neurological illnesses, as well as those with a history of epilepsy. More specifically, acute and remote CVAs are one common cause of SE in adults, with infections and fever being the most common etiology in children. Low antiepileptic drug levels are the apparent etiology of SE in about one fifth of children and one third of adults with pre-existing epilepsy, but fortunately mortality is relatively low in this group. Finally, it appears that a genetic predisposition may make some patients more susceptible to the development of SE.

Seizure Types

Early discussions of SE focused on generalized tonic-clonic or generalized convulsive SE (GCSE). Other forms of SE were not studied systematically until late in the twentieth century. Convulsive SE can be generalized or focal and the same is true for nonconvulsive SE. Generalized convulsive seizure types include primarily generalized, secondarily generalized, and myoclonic. Focal convulsive SE types include focal motor SE and epilepsia partialis continua. Nonconvulsive SE is "is a term used to denote a range of conditions in which electrographic seizure activity is prolonged and results in nonconvulsive clinical symptoms" (Walker et al., 2005). Absence SE is a form of generalized nonconvulsive SE. The focal nonconvulsive category includes complex partial SE and focal SE with nonmotor features such as aphasic or sensory SE. These divisions apply to patients after early childhood, because SE in neonates and infants is not so easily categorized.

An ictal alteration of consciousness differentiates complex partial seizures from simple partial seizures, as it does complex partial SE from simple partial SE. Partial or focal seizures can evolve into generalized seizures. In one study that included partial, secondarily generalized seizures under the classification of partial SE, approximately two thirds of both children and adults presented with partial seizures as the initial manifestation of SE. Conversely, GCSE was the final form of SE in almost three fourths of children and adults. Thus, SE may often begin with partial seizures that develop into generalized seizures. However, an initial generalized onset is most common in children less than one year old, the group in which SE is most likely to occur.

SE is a dynamic entity. During its course, seizures frequently wax and wane and clinical and EEG manifestations evolve. For example, the motor characteristics of GCSE may become less pronounced with prolonged SE and after this transition the state has been described as "subtle" GCSE. However, subtle GCSE can be the initial form of SE after a severe brain insult or accompany metabolic encephalopathies. The subtle form of GCSE, also called electrographic SE, can go undetected because patients often do not show obvious clinical signs of seizure activity. For example, studies have found a significant minority of comatose patients without overt seizures to be in electrographic SE. The term nonconvulsive SE (NCSE) is sometimes used synonymously with electrographic SE to describe these patients. However, NCSE also is used to refer to absence and nonmotor partial SE. Absence and partial SE may be characterized by frequent or prolonged subclinical epileptiform discharges and serial neuropsychological testing in such cases may uncover cognitive deficits that can remit after appropriate treatment.

Differential Diagnosis

Many medical and psychiatric conditions can be mistaken for SE. Potential physiologic imitators include tremor, dystonia, sleep disorders, hemifacial spasm, tic disorder, locked-in state, catatonia, cataplexy, transient global amnesia, drug side effects, etc.

Pseudostatus epilepticus (PSE) is defined as prolonged or repeated episodes of psychogenic nonepileptic

seizures (PNES). Ten to 30% of patients with PNES will present at some point with PSE. However, it should be noted that the combination of PNES and epilepsy in the same patient is not rare, and so a history of PNES does not rule out the possibility of SE.

Morbidity

More research is needed to establish the frequency of cognitive and neurologic abnormalities after SE that are separate from the effects of a precipitating neurologic insult, including a chronic intractable seizure disorder. The existing limited data suggest that severe cognitive deficits rarely result from SE that is not associated with an acute or progressive neurological insult. Similarly, it is difficult to determine if the development of chronic epilepsy after SE is the direct consequence of SE or of the underlying brain pathology. In children, the risk of developing epilepsy after a first episode of SE does not appear to be much different from the risk of epilepsy after a first seizure. But in adults, there seems to be a strong link between symptomatic SE and the risk of subsequent chronic epilepsy (Shorvon, Trinka, & Walker, 2007).

Mortality

Risk of mortality is lowest in children and highest in the elderly. As a specific example, the mortality rate from SE was 3% in children versus 38% in the elderly in the Richmond study. With effective medical management, death as a direct result of SE is relatively rare. Concurrent illness (acute symptomatic SE) is a strong risk factor for short-term mortality, but outcome varies with the specific etiology. For example, when anoxia after cardiac arrest results in SE the risk of death within 30 days is high. In addition to the short-term risk, SE is associated with an increased long-term mortality. Finally, both the incidence of SE and its short-term mortality risk appear to be higher in males, suggesting an association between SE and hormonal factors.

Treatment

Multiple physiologic and sometimes pathologic changes occur and progress in inadequately treated SE or experimental SE in animals. These include increased systemic, pulmonary, and left atrial pressure and increased heart rate and glucose concentration due to release of catecholamines, cardiac arrhythmias, impaired respiratory function, pulmonary edema, acidosis, hyperpyrexia, and cerebellar damage. As the pattern of clinical and EEG changes associated with GCSE progresses, SE becomes more resistant to treatment and neuronal damage becomes more likely.

Rectal diazepam is now regularly used to rapidly treat repetitive seizures or SE in the home or long-term care facility. When such treatment cannot be initiated, EMS personnel can administer IV lorazepam or diazepam prior to the patient's arrival at a hospital emergency department. One hospital treatment protocol for GCSE calls for a three-level approach that begins with IV lorazepam. If this initial therapy fails, the patient may respond to phenytoin or fosphenytoin. IV valproate can be substituted in patients allergic to phenytoin. If SE is treatment-refractory at that point, standard third-line drugs have a low success rate and so a general anesthetic, such as midazolam, propofol, or pentobarbital, is administered following intubation and ventilation.

EEG monitoring is essential to verify cessation of GCSE if it cannot be confirmed on clinical grounds alone. Treatment of SE should include a search for its etiology, with a review of the patient's medical, neurologic, and medication history and performance of laboratory, imaging, and possibly lumbar puncture studies.

Future Directions

Experimental study of SE did not gain strong momentum until the 1970s. While understanding of the progression and complications of SE is rapidly increasing, the development of new treatment tools has not kept pace. With animal models of SE in place, more research is needed to acquire additional information and determine its applicability to the treatment of human SE. Continuing support from federal agencies and pharmaceutical companies will be necessary in this quest toward improvement of therapeutic options and decision-making (Wasterlain & Treiman, 2006).

Cross References

- ▶ Absence Epilepsy
- ▶ Anticonvulsants
- ▶ Complex Partial Seizures
- ▶ Electroencephalography
- ▶ Epilepsy
- ▶ Grand Mal Seizure
- ▶ Psychomotor Epilepsy
- ▶ Simple Seizure (Partial)
- ▶ Temporal Lobe Epilepsy

Reference and Readings

Blume, W. T., Luders, H. O., Mizrahi, E., Tassinari, C., van Emde Boas, W., & Engel, J., Jr. (2001). Glossary of descriptive terminology for ictal semiology: Report of the ILAE task force on classification and terminology. *Epilepsia, 42*, 1212–1218.

Drislane, F. W. (Ed.). (2006). *Status epilepticus: A clinical perspective.* Totowa, NJ: Humana Press.

Feen, E. S., Bershad, E. M., & Suarez, J. I. (2008). Status epilepticus. *Southern Medical Journal, 101*, 400–406.

Shorvon, S. D. (Ed.). (1994). *Status epilepticus: Its clinical features and treatment in children and adults.* Cambridge: Cambridge University Press.

Shorvon, S. D., Trinka, E., & Walker, M. C. (Eds.). (2007). The proceedings of the first London colloquium on status epilepticus – University College London. *Epilepsia, 48*(Suppl 8), 1–109.

Walker, M., Cross, H., Smith, S., Young, C., Aicardi, J., Appleton, R., Aylett, S., Besag, F., Cock, H., DeLorenzo, R., Drislane, F., Duncan, J., Ferrie, C., Fujikawa, D., Gray, W., Kaplan, P., Koutroumanidis, M., O'Regan, M., Plouin, P., Sander, J., Scott, R., Shorvon, S., Treiman, D., Wasterlain, C., & Wieshmann, U. (2005). Nonconvulsive status epilepticus: Epilepsy Research Foundation workshop reports. *Epileptic Disorders, 7*, 253–296.

Wasterlain, C. G., & Treiman, D. M. (Eds.). (2006). *Status epilepticus: Mechanisms and management.* Cambridge, MA: MIT Press.

Steele–Richardson–Olszewski Syndrome

► Progressive Supranuclear Palsy

Stent

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Definition

A stent is a small wire metal mesh tube inserted inside an artery during an angioplasty procedure to keep the blood vessel open.

Current Knowledge

Stents are used to treat arterial blockage caused by atherosclerosis. A stent starts as a small diameter tube placed over a folded balloon on the tip of a catheter. During an angioplasty, the catheter is inserted into a blood vessel to gain access into the circulation. Under radiographic fluoroscopic visual guidance, the catheter is threaded along the course of the vessels to reach the target vessel, which may be a carotid artery, a renal artery, a peripheral artery in the leg, or most commonly, a coronary artery. At that time, the balloon is expanded to dilate the vessel, and the stent is opened, released, and adhered to the artery wall. The stent, which remains in the artery permanently, forms a "scaffold" to keep the artery open, thereby maintaining adequate blood flow through the previously compromised blood vessel. In time, the inner lining of the artery wall grows over the metal surface of the stent, thereby locking it in place. Following the procedure, most patients take antiplatelet or anticoagulation medications. Some stents contain and emit medication; these are called "drug-eluting stents." Stents are used commonly now; it is estimated that 70% of angioplasty procedures involve the use of stents.

Cross References

► Angioplasty
► Anticoagulation
► Antiplatelet Therapy
► Atherosclerosis
► Cerebrovascular Disease
► Coronary Disease
► Myocardial Infarction
► Peripheral Vascular Disease

References and Readings

American College of Cardiology, American Heart Association, Society for Cardiovascular Angiography and Interventions (2006). ACC/AHA/SCAI 2005 guideline update for percutaneous coronary intervention: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/SCAI Writing Committee to Update the 2001 Guidelines for Percutaneous Coronary Intervention). *Circulation, 113*, e166–e286.

Stereognosis

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Synonyms

Astereognosis; Object agnosia; Tactile object agnosia

Definition

Stereognosis is the ability to recognize and identify common objects through tactile manipulation without the use of visual cues. Conversely, difficulty in recognizing items by touch when primary sensory modalities (e.g., pain, temperature, and vibration) are intact is termed *astereognosis* or *tactile object agnosia*.

Categorization

Astereognosis is a type of tactile agnosia.

Epidemiology

Lesions to the somatosensory cortex, specifically the postcentral gyrus of the parietal lobe are implicated in the development of astereognosis, with damage usually being expressed contralaterally (Campbell, 2005; DeMyer, 2003; Husain, 2002; Roland, 1976; Tomberg & Desmedt, 1999). Because the vast majority of healthy individuals can identify common objects by touch, even one mistake on a test for stereognosis may be pathognomic of cerebral dysfunction (Dean & Davis, 2007).

Evaluation

Testing for stereognosis commonly includes blindfolding the patient, and then having them identify a series of commonly known shapes, items, or objects solely by manipulating them in their hand. Examples of assessment objects include keys, paperclips, coins, and buttons (Dean & Woodcock, 2003; Reitan & Wolfson, 2002). Such tasks are considered effective screeners because they test the entire sensory pathway, beginning with the finger tips all the way through to the parietal lobes and their cortical and subcortical connections (Bauer & Demery, 2003).

Treatment

While diagnosis of astereognosis is not difficult, addressing treatment options can be complicated by the fact that difficulty in identifying objects through manual manipulation can stem from a number of problems not necessarily related to a primary somatosensory deficit. Imaging research using functional MRI techniques has shown multiple areas of activation in the brain during tactile object recognition tasks, implicating not only the parietal somatosensory region but also the visual association cortex and the frontal polar cortex (Deibert, Kraut, Kremen, & Hart, 1999). Such findings may suggest that tactile object representation includes multiple networks of cortical pathways, including those necessary for motor, visual, and lexical processing. Deficits in any of these pathways could result in difficulty with tactile object recognition. Other deficits, including memory disorders, dysnomia, dementia, nerve damage, spinal cord damage, and motor deficits, can all contribute to symptoms that manifest as difficulty in recognizing and identifying shapes through touch (Bauer & Demery, 2003; Campbell, 2005; Husain, 2002; Ropper & Brown, 2005). Because of this, evidence of astereognosis must be carefully reviewed and differentially diagnosed from other disorders that might mimic the effects of astereognosis when considering treatment options.

Cross References

- ▶ Dean Woodcock Neuropsychological Assessment System
- ▶ Halstead–Reitan Neuropsychological Battery
- ▶ Somatosensory Cortex
- ▶ Somatosensory System
- ▶ Tactile Agnosia

References and Readings

Bauer, R. M., & Demery, J. A. (2003). Agnosia. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (4th ed., pp. 236–295). New York: Oxford University Press.

Campbell, W. W. (2005). General outline of the neurologic examination. In A. F. Haerer (Ed.), *DeJong's the neurologic examination* (pp. 37–40). Philadelphia: Lippincott, Williams & Wilkins.

Caselli, R. J. (1991). Rediscovering tactile agnosia. *Mayo Clinic Proceedings, 66*, 129.

Dean, R. S., & Davis, A. S. (2007). The Dean–Woodcock neuropsychological assessment system. In A. M. Horton & D. Wedding (Eds.), *The neuropsychology handbook*(3rd ed., pp. 397–418). New York: Springer.

Dean, R. S., & Woodcock, R. W. (2003). *Dean–Woodcock sensory motor battery.* Itasca, IL: Riverside Publishing.

Deibert, E., Kraut, M., Kremen, S., & Hart, J. (1999). Neural pathways in tactile object recognition. *Neurology, 52*, 1413–1420.

DeMyer, W. (2003). Examination of the general somatosensory system. In *Technique of the neurologic examination: A programmed text* (pp. 439–497). New York: McGraw-Hill.

Gaubert, C., & Mockett, S. P. (2000). Inter-rater reliability of the Nottingham method of stereognosis assessment. *Clinical Rehabilitation, 14*, 153–159.

Husain, M. (2002). Parietal lobe syndrome. In J. E. Harrison & A. M. Owen (Eds.), *Cognitive deficits in brain disorders* (pp. 59–77). London: Taylor & Francis.

Reitan, R. M., & Wolfson, D. (2002). Using the tactile form recognition test to differentiate persons with brain damage from control subjects. *Archives of Clinical Neuropsychology, 17*, 117–121.
Roland, P. E. (1976). Astereognosis. Tactile discrimination after localized hemispheric lesions in man. *Archives of Neurology, 33*(8), 543–550.
Ropper, A. H., & Brown, R. H. (2005). Other somatic sensations. In A. H. Ropper & R. H. Brown (Eds.), *Adams and Victor's principles of neurology* (8th ed., pp. 129–143). New York: McGraw-Hill.
Stewart, C., Evans, W. B., & Fitch, J. L. (1985). Oral form perception skills of stuttering and nonstuttering children measured by stereognosis. *Journal of Fluency Disorders, 10*(4), 311–316.
Tomberg, C., & Desmedt, J. E. (1999). Failure to recognise objects by active touch (astereognoisa) results from lesion of parietal-cortex representation of finger kinaesthesis. *Lancet, 354*, 393–394.

Stereotactic Conformal Radiotherapy

► Stereotactic Radiation Therapy

Stereotactic Radiation Therapy

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Synonyms

Stereotactic conformal radiotherapy

Definition

Stereotactic radiation therapy is a variant of conventional radiation therapy, involving the emission of precise and focused single high-dose radiation via sophisticated three-dimensional computerized imaging. This procedure is primarily indicated for inoperable brain tumors, residual brain tumor tissue, and lesions that are adjacent to essential or critical brain regions (e.g., the brainstem). Stereotactic radiation therapy is distinguished from stereotactic radiosurgery by the treatment duration. For example, stereotactic radiosurgery is often completed in an individual session, whereas stereotactic radiation therapy can be fractionated (i.e., administered over period of days or weeks). Stereotactic radiation therapy has also reportedly been associated with attention and memory impairments, the long-term effects of which are still indeterminate (Steinvorth et al., 2003). However, research has indicated that multiple smaller doses may improve patient outcomes and reduce the treatment's side effects (Saran et al., 2002).

Cross References

► Stereotactic Radiosurgery

References and Readings

Saran, F. H., Baumert, B. G., Khoo, V. S., Adams, E. J., Garré, M. L., Warrington, A. P., et al. (2002). Stereotactically guided conformal radiotherapy for progressive low-grade gliomas of childhood. *International Journal of Radiation Oncology Biology Physics, 53*, 43–51.
Steinvorth, S., Welzel, G., Fuss, M., Debus, J., Wildermuth, S., Wannenmacher, M., et al. (2003). Neuropsychological outcome after fractionated stereotactic radiotherapy (FSRT) for base of skull meningiomas: A prospective 1-year follow-up. *Radiother Oncol, 69*, 177–182.

Stereotaxic Surgery

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Definition

Stereotaxic surgery is a minimally invasive form of surgical intervention which uses three-dimensional coordinates to locate a precise target within the body without having to open a larger surgical field.

Current Knowledge

Introduced in neurosurgery to guide operative management such as resections, biopsies, and implantations, this technique requires the use of a reliable frame of reference such as a frame rigidly fixed to the skull. Ernest Spiegel and Henry Wycis developed the first stereotatic frame using the Cartesian coordinate system. Based upon a stereotactic atlas which outlined targeted anatomical structures, surgeons could position the frame or apparatus using bony landmarks such as the external auditory meatus, inferior orbital ridges, and the bregma which could be identified in the conventional radiographs.

Current technology allows for the identification of the targeted tissue via a preoperative CT or MRI and placement of the surgical frame based on the target coordinates. Stereotactic localization can also guide radiosurgery such as gamma knife and cyberknife treatments. Although stereotactic guidance is based upon the assumption that the anatomic imaging prior to entering the operating room accurately depicts the effects on tissues during a procedure, technology is now allowing for intraoperative MRI or ultrasound to take stereotaxic surgery to another level of detail.

References and Readings

Peters, T. M. (2000). Image-guided surgery: From X-rays to virtual reality. *Computer Methods in Biomechanics Biomedical Engineering, 4*, 27–57.

Peters, T. M. (2006). Image-guidance for surgical procedures. *Physics in Medicine and Biology, 51*, R505–40.

Willems, P. W., van der Sprenkel, J. W., Tulleken, C. A., Viergever, M. A., & Taphoorn, M. J. (2006). Neuronavigation and surgery of intracerebral tumours. *Journal of Neurology, 253*, 1123–1136.

Stereotypy

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Synonyms

Recurrent stereotypic utterance; Repetitive utterance

Definition

Verbal stereotypy is a nonpropositional utterance characterized by repetition of a syllable, word, or phrase (e.g., "ba-ba-ba," "yep," "bloody hell," "wait a minute"), typically used in high frequencies and as emotional exclamations (Alajouanine, 1956). Stereotypies are a feature of advanced frontotemporal dementia (Mendez & Perryman, 2002) and other disorders of left hemisphere language regions,which are distinct from instances of perseveration of a correct word to a subsequent utterance (e.g., saying "husband" for "wife" when the previous topic was "husband"). Present early in acute, severe nonfluent aphasia and often persisting for weeks, stereotypies may be coupled with prosody that mimics linguistic forms, e.g., "*ba ba ba?*" with the intonation of "*How are you?*," but lack the phonetic variation of neologisms typical of fluent aphasia.

Cross References

- ▶ Aphasia
- ▶ Apraxia of Speech
- ▶ Frontal Temporal Dementia
- ▶ Perseveration
- ▶ Prosody

References and Readings

Alajouanine, T. (1956). Verbal realization in aphasia. *Brain, 79*, 1–28.

Blanken, G., & Marini, V. (1997). Where do lexical speech automatisms come from? *Journal of Neurolinguistics, 10*(1), 19–31.

Goodglass, H., & Kaplan, E. (1972). *Assessment of Aphasia and related disorders.* Philadelphia: Lea and Febinger.

Mendez, M. F., & Perryman, K. M. (2002). Neuropsychiatric features of frontotemporal dementia: Evaluation of consensus criteria and review. *Journal of Neuropsychiatry and Clinical Neuroscience*, (14), 424–429.

Steroids

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Synonyms

Sex hormone

Definition

Naturally occurring steroids are estrogen, cortisol, progesterone, and testosterone. Estrogen and progesterone are primarily produced in the ovary and placenta while testosterone is primarily produced in the testes. Pharmacologically, they are used in several ways: alter steroid metabolism via statins to help lower cholesterol, as corticosteroids to help control inflammation, asthma and lupus, and other forms to help hormone deficiency, such as delayed puberty, as well as diseases that result in

loss of lean muscle mass, such as cancer and AIDS. Anabolic steroids, often abused, are used to increase muscle mass and can cause many psychological and physical problems.

Cross References

- ▶ Pharmacodynamics
- ▶ Pharmacokinetics
- ▶ Psychopharmacology

References and Readings

Ahboucha, S., Butterworth, R., Pomier-Layrargues, G., Vincent, C., Hassoun, Z., & Baker, G. (2008). Neuroactive steroids and fatigue severity in patients with primary biliary cirrhosis and hepatitis C. *Neurogastroenterology & Motility, 20*(6), 671–679.

Dean, S., & McCarthy, M. (2008). Steroids, sex and the cerebellar cortex: implications for human disease. *Cerebellum, 7*(1), 38–47.

De Kloet, E., Van Acker, S., Sibug, R., Oitzl, M., Meijer, O., Rahmouni, K., et al. (2000). Brain mineralocorticoid receptors and centrally regulated functions. *Kidney International, 57*(4), 1329–1336.

Stiff-Legged Gait

- ▶ Spastic Gait

Stiffness

- ▶ Rigidity

Stimulants

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Synonyms

Psychostimulants

Definition

Stimulants are substances that cause temporary improvements in mental and/or physical function. Amphetamines increase alertness and physical activity by increasing the heart and respiration rates, blood pressure, pupil dilation, and decreasing appetite. Common side effects are anxiety, blurred vision, sleeplessness, and dizziness. Stimulants can be used as medications (such as epinephrine) to increase heart and respiration rate and brain function. When properly administered, stimulants can be used to treat narcolepsy, a rare sleep disorder, and to help children with minimal brain dysfunction. Other types, and often abused stimulants are caffeine, nicotine, and cocaine.

Cross References

- ▶ Amphetamine
- ▶ Epinephrine
- ▶ Psychopharmacology

References and Readings

Gardner, B., Sheppard, D., & Efron, D. (2008). The impact of stimulants on a clinical measure of attention in children with ADHD. *Child Neuropsychology, 14*(2), 171–186.

Humphries, T., Kinsbourne, M., & Swanson, J. (1978). Stimulant effects on cooperation and social interaction between hyperactive children and their mothers. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 19*(1), 13–22.

Snyder, A., Maruff, P., Pietrzak, R., Cromer, J., & Snyder, P. (2008). Effect of treatment with stimulant medication on nonverbal executive function and visuomotor speed in children with attention deficit/hyperactivity disorder (ADHD). *Child Neuropsychology, 14*(3), 211–226.

Stimulus Control

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Synonyms

Conditioned stimulus

Definition

In classical or operant conditioning, stimulus control is present when the subject is trained so that the stimulus evokes the conditioned response or operant behavior. Stimulus control can increase or decrease the probability that the behavior will occur, depending on the conditioning. Stimulus control is often used to establish discriminative stimuli, so that the desired behavior occurs in the presence of a specific stimulus. Behavioral therapies based on stimulus control have been developed, such as stimulus control therapy for insomnia. These therapies seek to control behavior by conditioning patients to stimuli that act as antecedents to the desired behaviors.

Cross References

► Stimulus-Bound Behavior

Stimulus Fading

► Errorless Learning

Stimulus Generalization

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Synonyms

Generalization

Definition

Stimulus generalization is the tendency of a new stimulus to evoke responses or behaviors similar to those elicited by another stimulus. For example, Ivan Pavlov conditioned dogs to salivate using the sound of a bell and food powder. The unconditioned stimulus (food powder) was paired with a conditioned stimulus (sound of a bell) until the conditioned stimulus produced the response (salivation) in the absence of the unconditioned stimulus (food powder). The dogs were then noted to salivate in response to other noises similar to the bell used in conditioning, even though these noises had never been used during conditioning.

Cross References

► Stimulus Control
► Stimulus-Bound Behavior

Stimulus Strength

► Cue Dominance

Stimulus-Bound Behavior

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Synonyms

Automatic behavior; Stimulus-driven behavior

Definition

Stimulus-bound behavior is commonly found in frontal lobe syndrome and other executive functioning disorders and is a response to stimuli in one's environment – an externally oriented cognitive approach. For example, the behavior displayed seems to depend primarily upon available objects and subject predisposition, rather than the activation of a specific drive such as hunger, anger, sex-drive, etc. A person exhibiting stimulus-bound behavior may feel the need to use certain items present, regardless of a need to do so. The behaviors are often perseverative in nature and focus on partial information. Immediate stimulus-bound behavior often does not take into account future consequences or long-term outcomes and causes difficulty with planning, organizing, and behavioral initiative.

Cross References

- ▶ Executive Functioning
- ▶ Frontal Lobe Syndrome
- ▶ Frontal Lobes

References and Readings

Archibald, S., Mateer, C., & Kerns, K. (2001). Utilization behavior: Clinical manifestations and neurological mechanisms. *Neuropsychology Review, 11*(3), 117–130.

Feinstein, A., Levine, B., & Protzner, A. (2000). Confabulation and multiple sclerosis: A rare association. *Multiple Sclerosis (13524585), 6*(3), 186–191.

Jackson, A., & Haverkamp, B. (1991). Family response to traumatic brain injury. *Counselling Psychology Quarterly, 4*(4), 355.

Suedfeld, P. (1988). Are simple decisions always worse? *Society, 25*(5), 25–27.

Wachtel, E. (1976). The multiple meaning of symptoms: A case illustration. *Psychotherapy: Theory, research & practice, 13*(3), 219–224.

Stimulus-Driven Behavior

- ▶ Stimulus-Bound Behavior

STM Transfer Rate

- ▶ Span of Apprehension

Strabismus

- ▶ Diplopia
- ▶ Dysconjugate Gaze

Strategy Substitution

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Definition

Strategy substitution is an approach to cognitive rehabilitation that involves circumventing impaired cognitive functions by employing alternative or modified strategies in order to facilitate functional living.

Current Knowledge

Cognitive rehabilitation interventions typically follow either a restorative model (involving restoration of lost functions through practice and retraining) or a compensatory model (involving substitution of learned strategies for impaired functions); strategy substitution is synonymous with the latter model.

Strategy substitution requires an individualized assessment of a person's cognitive strengths and weaknesses in order to identify both impaired functions and intact abilities. Based on this information, a person's approach to a task is modified so that the same functional outcome is achieved in a new way. Specifically, a strategy is created that relies on the intact and circumvents the impaired cognitive functions. Brouwer, Van Zomeren, Berg, Bouma, and de Haan (2002) distinguish internal strategies, which rely on a person's residual cognitive strengths and ability to learn strategies, and external strategies, which involve the use of prosthetic tools (see Table 1).

Strategy Substitution. Table 1 Compensatory applications of internal and external strategy substitution

	Task	"Old" strategy	Impaired ability	Substitute strategy
Internal strategy	Reading the newspaper	Read the words seen on newspaper page	Left visual inattention causes person to miss words on the left side of the page	Self-talk to enhance visual scanning: repeat, "Look Left!" at the start of each line
External strategy	Grocery shopping	Browse aisles and purchase needed items	Memory deficit causes person to forget what items they need	Make a list and systematically cross out each item added to cart

In order for strategy substitution to be successful, a person must have adequate awareness of his or her deficits and the ability to anticipate the functional impact of his or her deficits (Gross & Schutz, 1986), since the person must preemptively initiate the use of a strategy to avoid errors and maximize functioning. The effectiveness of strategy substitution is further enhanced by involving significant others to provide structure, cueing, and to promote awareness of a person's strengths and weaknesses (Wilson, 2003).

Unlike restorative approaches to cognitive rehabilitation, strategy substitution is not intended to "fix" impaired abilities. Therefore, while the implementation of compensatory strategies may become more habitual and even automatic over time and with continued use, compensation for impaired functions through the use of strategy substitution requires continuous implementation if functional outcomes are to be achieved.

Cross References

► Cognitive Rehabilitation
► Compensatory Strategies

References and Readings

Brouwer, E., Van Zomeren, E., Berg, I., Bouma, A., & de Haan, E. (2002). Cognitive rehabilitation: A clinical neuropsychological approach. Amsterdam: Uitgeverij Boom.

Gross, Y., & Schutz, L. E. (1986). Intervention models in neuropsychology. In B. Uzzell, & Y. Gross (Eds.), *Clinical neuropsychology of intervention* (pp. 179–204). Springer-Verlag New York, LLC.

Prigatano, G. P. (1999). Principles of neuropsychological rehabilitation. New York: Oxford University Press.

Wilson, B. (2003). Theoretical approaches to cognitive rehabilitation. In L. H. Goldstein & J. E. McNeil (Eds.), *Clinical neuropsychology: A practical guide to assessment and management for clinicians* (pp. 345–366). Wiley, John & Sons, Incorporated.

Strength of Grip

► Hand Dynamometer

Strength-Based Education

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Definition

Strength-based education focuses on an individual's educational performance, considering what the individual can accomplish. In daily practice, it relates to how an individual engages in the teaching and learning process. It also emphasizes the positive aspects of the individual's effort and achievement.

Historical Background

Clabaugh (2005) claims that strength-based education was designed by Pestalozzi in the late 1700s and redesigned by Froebel in the 1830s. Lopez (2006) argued that strength-based education also related to Alfred Binet's seminal work (Binet & Simon, 1916), which he viewed as related to assessment, devoted to enhancing student skills, and addressing deficits, not just connected to remediating problems.

Rationale or Underlying Theory

Traditionally, each learner's weaknesses and performance deficits were identified, and intervention programs that focused on the remediation of problem areas were developed (Gaddess & Edgell, 1994). But the weaknesses focus was found to be inefficient and ineffective. Many interventions only emphasized children's and adults' weaknesses, and even with the best instruction, children and adults could not easily overcome their weaknesses (D'Amato, Rothlisberg, & Leu, 1999). Thus, children and adults alike, lost their patience and enthusiasm to achieve and often failed in the enterprise of learning. It became clear that a singular focus on deficits could not help many individuals learn.

Goals and Objectives

Accordingly, a strength-based approach becomes critical, especially when working with problem learners or

children who were emotionally or mentally disabled. To learn, teachers needed to focus on what students were good at, helping them to solve problems and difficulties using cognitive plans and flexible thinking. Strength-based approaches also encouraged children to be more willing to overcome their difficulties and achieve tasks which did not focus on their weakness. They needed to learn to work around their problem areas. D'Amato et al. (1999) have advocated that three educational approaches should be used: (1) remediation, (2) compensatory (strength-based), and (3) a combination of both.

Qualifications of Treatment Providers

Most authors (e.g., Witsken, Stoeckel, & D'Amato, 2008) have argued that all individuals involved in teaching others (including parents, educators, and even other children) should use a strength-based approach if they are to be effective in helping others learn.

Cross References

- ▶ Academic Competency
- ▶ Academic Skills
- ▶ Compensatory Education Approach
- ▶ Remedial Education Approach

References and Readings

Binet, A., & Simon, T. (1916). *The development of intelligence in children.* (E. S. Kit, Trans.). Baltimore: Williams & Wilkins.

Clabaugh, G. K. (2005). Strengths-based education: Probing its limits. *Educational Horizons, 83*, 166–170.

D'Amato, R. C., Rothlisberg, B. A., & Leu Work, P. H. (1999). Neuropsychological assessment for intervention. In T. B. Gutkin & C. R. Reynolds (Eds.), *Handbook of school psychology* (3rd ed., pp. 452–475). New York: Wiley.

Gaddes, W. H., & Edgell, D. (1994). *Learning disabilities and brain function: A neuropsychological approach* (3rd ed.). New York: Springer-Verlag.

Lopez, S. J. (2006). Strengths-based education research and resources: Strengths development research and resources. *Educational Horizons, 84*, 157–160.

Witsken, D., Stoeckel, A., & D'Amato, R. C. (2008). Leading educational change: Using a neuropsychological response-to-intervention approach: Linking our past, present, and future. *Psychology in the Schools, 45*(9), 781–798.

Stress

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Definition

Stress can be defined as the process and response subsequent to the demands of a hostile environment. It generally refers to a negative emotional experience accompanied by physiological, cognitive, and behavior adaptation.

Historical Background

Several prevailing theories in stress research were developed by Selye (1936) and Lazarus (1966). Selye proposed the general adaptation syndrome (GAS) which consists of three stages. Stage 1, alarm, is characterized by shock and countershock. Shock manifests as the alteration of homeostatic processes such as blood pressure and glucose production, etc. Countershock describes the body's release of stress hormones to counter the effects of the shock-related responses. Stage 2, the resistance phase, involves adaptation to the negative effects of the stressor, during which the individual is more susceptible to the effects of competing homeostatic challenges. Stage 3, the exhaustion phase, marks the depletion of adaptation energy stores and can lead to the physical ailments described above, or even death. A major criticism of Seyyle's and related theories is the lack of emphasis on the cognitive response to stress.

Lazarus (1966), in the Transactional Model of stress and coping, described stress a stimulus-response interaction. According to the Transactional Model, a life event occurs and is subjected to a primary appraisal in which an individual attempts to assess the threat of the event. If the event is perceived as stressful, the stress response ensues. The continuation of this response is a secondary appraisal of whether the individual's coping strategies are sufficient to deal with the perceived threat of the event. After the secondary appraisal, the individual copes with the stressor emotionally, cognitively, behaviorally, and physiologically.

Current Knowledge

Stress can be measured by self-report and physiologically. Objective questionnaires such as the Life Experiences

Survey (Sarason, Johnson, & Siegel, 1978) and the Schedule of Recent Events (Holmes & Rahe, 1967) can be useful tools. Physiologically, stress can be measured by immune status (T-cell count), immune functioning (natural killer cell activity), herpesvirus (e.g., EBV) titers, cortisol, norepinephrine and epinephrine levels, and health symptoms.

A stressor, the stimulus that results in stress, can be physical or psychological. Some stressors can be purely physical and are thought to bypass cognition interpretation. Examples include physical trauma, overcrowding, noise, and physical exertion. Psychological stressors are thought to be deemed stressful by virtue of their cognitive evaluation. Examples include interpersonal conflict, physical/emotional/verbal abuse, witnessing domesticviolence, and major life transitions. However, most stressors, even physical ones, have some psychological evaluation thought to moderate the perceived level of stress.

Stress, especially, when it is chronic, has physiological consequences, as it activates the HPA (hypothalamic-pituitary-adrenal) axis as well as the sympathetic nervous system. The physical sequalae of stress include elevated blood pressure, increased metabolism (e.g., faster heartbeat and respiration), increased production of blood sugar, more stomach acids, decreased ingestion, raised cholesterol and fatty acids for energy. Physical illnesses associated with stress include migraine headaches, ulcers, TMJ (tempromandibular joint) syndrome, upper respiratory infections, and higher rates of the common cold. Among those with coronary disease, stressful events can even precede sudden death (Lecomte, Fornes, & Nicolas, 1996).

Psychologically, stress has important consequences as well. Stress can lead to errors in thinking ("cognitive distortions"). An example of a distortion is hindsight bias: "If they had never put that medical device in my heart that always shocked me and made me so nervous, I never would have lost my job or my girlfriend." For a complete listing of cognitive distortions, see Beck (1975). Stress can also result in behaviors such as substance use ("self medication"), increased health care utilization, and isolating or aggressive behaviors, etc.

Cross References

- ► Coping
- ► Posttraumatic Stress Disorder
- ► Stress Management

References and Readings

Beck, A. T. (1975). *Cognitive therapy and the emotional disorders*. New York: International Universities Press.

Holmes, T., & Rahe, R. (1967). The social readjustment rating scale. *Journal of Psychosomatic Research, 11*, 213–218.

Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw-Hill.

Lecomte, D., Fornes, P., & Nicolas, G. (1996). Stressful events as a trigger of sudden death: A study of 43 medico-legal autopsy cases. *Forensic Science International, 79*, 1–10.

Sarason, I. G., Johnson, J. H., & Siegel, J. M. (1978). Assessing the impact of life changes: Development of the life experiences survey. *Journal of Consulting and Clinical Psychology, 46*, 932–946.

Sarason, I. G., Levin, M. H., Bashman, R. B., & Saranson, B. R. (1983). Assessing social support: The social support questionnaire. *Journal of Personality and Social Psychology, 44*, 127–139.

Selye, H. (1936). A syndrome produced by diverse nocuous agents. *Nature, 138*, 32.

Stress Management

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Definition

Stress management is the process of acquiring and practicing a variety of cognitive and behavioral techniques with the goal of managing and coping with stress. Stress is defined as the perception that demands exceed personal resources, and can result in both physiological (e.g., increased blood pressure, migraine headaches) as well as psychological effects (e.g., anxiety, depression). Potential sources of stress range from major life events such as death of a loved one, divorce, or job loss to "daily hassles" such as traffic and interpersonal annoyances.

Current Knowledge

Stress management is based on the premise that the experience of stress is not a direct response to a stressor per se; rather the experience of stress is mediated by one's coping abilities and resources, both of which can be modified through stress-management techniques. Stress-management techniques include diaphragmatic breathing, relaxation training (progressive muscle relaxation or autogenic relaxation), meditation, increasing physical activity,

improving nutrition, increasing pleasurable activities, improving goal-setting skills, developing conflict-resolution skills, learning time-management skills, identifying and reframing cognitive distortions, limit-setting and assertiveness training, and utilizing (or expanding) the sources of social support. Stress-management techniques can be learned through self-help resources (books, internet), or can be delivered in individual or group format through professional intervention. However acquired, stress-management techniques must be practiced regularly over a period of time to achieve optimal stress-reduction benefits.

Stress-management interventions are particularly efficacious (and cost effective) in medical populations for whom stress can have a detrimental affect on quality of life and/or disease process. Empirical studies suggest that patients with chronic pain, cardiovascular disease, autoimmune disorders, cancer, HIV/AIDS, spinal cord injury (SCI), and multiple sclerosis (MS) derive substantial psychological and physical benefits from stress-management intervention. Indeed, a testament to the robust effect of stress management lies in the United States Headache Consortium endorsement of behavioral intervention/stress management as a first-line intervention for the prevention and treatment of migraine and tension-type headache. Other specific benefits of stress-management intervention in medical patients include (a) reduced pain levels, blood pressure, blood pressure reactivity, and all-cause mortality rates in patients with cardiovascular disease; (b) reduced pain, anxiety, distress, and fatigue and improved quality of life and role function in patients with autoimmune disorders; (c) reduced cancer-related anxiety during and after a course of cancer treatment; (d) improvement in immune function in patients with HIV/AIDS (found in some but not all studies); (e) decreased levels of pain and fewer role limitations in patients with SCI and MS. Finally, for the personnel in medical settings where high levels of stress and burnout are common, stress-management intervention is effective in improving role function and quality of life in these essential care providers.

Cross References

► Coping
► Relaxation Training

References and Readings

Crepaz, N., Passin, W. F., Herbst, J. H., Rama, S. M., Malow, R. M., Purcell, D. W., et al. (2008). Meta-analysis of cognitive-behavioral interventions on HIV-positive persons' mental health and immune functioning. *Health Psychology, 27*, 4–14.

Davis, M., Eshelman, E. R., & McKay, M. (2007). *The relaxation & stress reduction workbook* (5th ed.). Oakland, CA: New Harbinger.

Rains, J. C., Penzien, D. B., McCrory, D. C., & Gray, R. N. (2005). Behavioral headache treatment: History, review of the empirical literature, and methodological critique. *Headache, 45*(2), S92–S109.

Sapolsky, R. M. (2004). *Why zebras don't get ulcers: The acclaimed guide to stress, stress-related diseases, and coping* (3rd ed.). New York: Holt Publishing.

Striate Cortex

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Synonyms

Primary visual cortex; V1

Definition

The brain has more surface area dedicated to vision than any other sensory function. The primary visual receptive area is the striate cortex located in the occipital lobe. Also referred to as "V1," striate cortex is located in and around the calcarine fissure in each of the occipital lobes. V1 receives information directly from its ipsilateral lateral geniculate nucleus of the thalamus, via the optic radiations. It then sends information via two primary pathways: the "dorsal stream" and the "ventral stream." The dorsal stream, also known as the "where" or "how" pathway, projects to the parietal lobe and is thought to be involved in the location of objects in space. The ventral stream, also referred to as the "what" pathway, projects to the medial temporal lobe and limbic system, and is thought to be involved in object and form recognition.

Cross References

► Calcarine Cortex
► Eye Fields
► Occipital Lobe
► Visual Cortex

Striatonigral Degeneration

► Multisystem Atrophy (MSA)

Striatum

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Synonyms

Neostriatum

Definition

Striatum is a subcortical part of the telencephalon that represents the principal receptive component of the basal ganglia system.

Current Knowledge

The striatum is spatially subdivided into dorsal and ventral striatum. The dorsal striatum is topographically separated by the internal capsule to the caudate and putamen, while the ventral striatum contains the nucleus accumbens, and the striatal part of the olfactory tubercle. The selective distribution of the axonal terminals that originate from cortical sources differentiates three distinct striatal territories: sensorimotor, associative, and limbic. More information on the anatomical divisions of the striatum can be found in Voorn, Vanderschuren, Groenewegen, Robbins, and Pennartze (2004). Immunochemically, particularly based on acetylcholine esterase staining, the adult striatum is made up of two compartments: "striosomes" (weak staining) and matrix (strong staining).

The striatum is homogeneous in terms of neuronal components. It is built of **four** neuronal types: spiny neurons (96% of the striatum), leptodendritic (Deiter's) neurons (2%), spidery cholinergic interneurons (1%), and GABAergic interneurons (1%). The GABAergic interneurons are subdivided into three groups based on expression of parvalbumin, calretinin, or somatostatin. Both parvalbumin- and somatostatin-expressing interneurons express dopamine receptors.

The afferent connections include corticostriatal (glutamatergic), nigrostriatal (dopaminergic), thalamostriatal pathways (glutamatergic), connections that are received from the raphe nuclei (serotoninergic), the locus ceruleus (noradrenergic), and from other elements of basal ganglia such as subthalamic nucleus (glutamatergic) and the external globus pallidus (GABAergic). The corticostriatal pathway is the most massive, it is presented by cortical pyramidal neurons mainly of lamina V that end on the spines of the spiny neurons. The main efferent target of the striatum is pallidonigral set that comprises pathways projecting directly and indirectly to the globus pallidus and the substantia nigra. The neostriatal projections to the different target areas contain one neurotransmitter, GABA, and various neuropeptides such as substance P, enkephalin, dynorphin, and neurotensin.

The striatum is well known for its role in the planning and modulation of movements; it is also involved in a variety of cognitive processes including executive function. In humans, the striatum is activated by stimuli associated with reward as well as by aversive, novel, unexpected or intense stimuli, and cues associated with such events.

Parkinson disease results in loss of dopaminergic innervations to the striatum and other basal ganglia. The loss of these innervations leads to a decreased motor activity (hypokinesia). An accumulated evidence suggests that stimulation of striatal nicotinic acetylcholine receptors may represent a new treatment approach for Parkinson's disease. The loss of basal ganglia neurons accompanied by a decrease in their inhibitory output leads to an increased motor activity (hyperkinesia) which is involved in mechanisms of choreas, Hungtington's disease, athetosis, ballism, dystonia, and Tourette's syndrome. Considerable evidence exists that addiction involves plasticity at striatal synapses.

Cross References

▶ Basal Ganglia
▶ Parkinson's Disease

References and Readings

Bolam, J. P., Ingham, C. A., & Magill, P. J. (2005). *The basal ganglia VIII* (*advances in behavioral biology*). New York: Springer Science/Business Media.

Delgado, M. R. (2007). Reward-related responses in the human striatum. *Annals of New York Academy of Sciences, 1104*, 70–88.

Miller, R. (2007). *A theory of the basal ganglia and their disorders* (*conceptual advances in brain research*). Boca Raton, Fl: Taylor & Francis/CRC press.

Quik, M., Huang, L. Z., Parameswaran, N., Bordia, T., Campos, C., & Perez, X. A. (2009). Multiple role for nicotine in Parkinson's disease. *Biochem Pharmacol, 78*, 677–685.

Voorn, P., Vanderschuren, L. J. M. J., Groenewegen, H. J., Robbins, T. W., & Pennartz, C. M. A. (2004). Putting a spin on the dorsal-ventral divide of the striatum. *Trends in Neurosciences, 27*, 468–474.

Stroke

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Synonyms

Acute cerebrovascular attack; Brain attack; Cerebrovascular accident (CVA)

Short Description/Definition

A *stroke* is a clinical syndrome of sudden onset and without external cause (e.g., trauma), arising from a disruption in the blood supply in the brain. The Greeks were the first to use the term "apoplexy" as applied to cases of individuals who suddenly fell and were unable to move, as if struck by lightning. Although this term continued to be used into the twentieth century, Johanne Jacob Wepfer attributed the symptoms of stroke to disorders of circulation in the mid-1600s. With the rise of scientific medicine in the eighteenth and nineteenth centuries, and new attention to underlying pathology, the concept of disease began to take hold and it was recognized that clinical events caused by obstruction of blood vessels and by the breakdown of blood vessel walls occurred largely in the arterial system, and the notion of cerebrovascular disease emerged. The term "stroke" began to be used as a term of convenience in the early 1960s.

Categorization of Stroke

Anatomy of Cerebral Circulation

The brain is fed by two main arterial sources: the internal carotid arteries and the vertebral arteries. The left and right internal carotids supply the anterior cerebral circulation with about 80% of the brain's blood supply. The vertebral arteries unite at the border of the pons to form the basilar artery that supplies 20% of the brain's blood volume via the posterior cerebral circulation.

At the medial base of the cerebral hemispheres is a unique arterial ring, the circle of Willis, formed by early segments of the anterior, middle, and posterior cerebral arteries (PCAs) and the anterior and posterior communicating arteries.

The left and right anterior cerebral arteries (ACAs) arise from the anterior portion of the circle of Willis, and are connected by the anterior communicating artery (ACoA). The ACoA, as well as small branches from the ACA, penetrate the brain to supply blood to the fornix, septal regions, anterior perforated substance, optic chiasm, optic tract, optic nerve, and suprachiasmatic area. The ACA starts at the bifurcation of the internal carotid, entering the interhemispheric fissure, and then proceeding anteriorly and upward, and then posteriorly as it continues over the superior surface of the corpus callosum. Branches off the early segments of the ACA supply the head of the caudate, the anterior part of the internal capsule, anterior globus pallidus, olfactory regions, and hypothalamus. The ACA supplies to regions such as superior frontal gyrus, cingulate gyrus, and the premotor, motor and sensory areas of the paracentral lobule.

The left and right middle cerebral arteries (MCAs) represent the largest of the major branches of the internal carotid arteries, and supply most of the convex surface of the brain. Off the stem of the MCA are the lenticulostriate branches, named for the structures comprising the lentiform nucleus and striatum (caudate and putamen), and the internal capsule. As the MCA begins its course over the cortical surface, it then subdivides into several different branch configurations, but the most common pattern is a bifurcation into an upper and lower division. The initial segments in these two divisions supply the insula region, before proceeding over a large expanse of the lateral surfaces of frontal, parietal, and temporal lobes. In the upper or superior division, there is supply to the frontal lobe, including the orbital region, the inferior and middle frontal gyri, the pre- and postcentral gyri, as well as the superior and inferior parietal lobules. The lower or inferior division of the MCA provides circulation to the parietal and temporal opercula, the posterior temporal, posterior parietal, and temporo-occipital regions. The MCA can also exist in a trifurcation pattern.

The vertebral arteries, as they course up the spine into the skull, provide arterial supply to the brain stem and cerebellum, before merging into the basilar artery at the level of the pons. The PCAs are typically formed by

the bifurcation of the basilar artery at the circle of Willis where they are connected by the posterior communicating artery (PCoA). The PCAs continue to course superiorly along the lateral part of the brain stem, with penetrators supplying segments of the thalamus, before turning posterior as they pass over the tentorium and onto the medial and inferior surfaces of the temporal and occipital lobes.

In addition to the three major cerebral arterial territory distributions, there are so-called "central arteries" that provide penetrating branches into deep brain. Among these are the anterior and posterior choroidal arteries. The anterior choroidal artery, usually arising from the internal carotid artery, supplies the optic tract, lateral geniculate body, medial temporal lobe, and the anterior one-third of the hippocampus, the uncus, and part of the amygdala. Some of the perforating branches also feed the posterior limb of the internal capsule, optic radiations, the basal ganglia, and the ventrolateral region of the thalamus. Arising from the PCA, the posterior choroidal artery has one medial and two lateral branches and collectively feed superior and medial parts of the thalamus, the choroid plexus of the lateral ventricle, and the posterior two-thirds of the hippocampus.

Pathophysiology of Stroke

In order to survive, the neurons and supportive tissue in the brain rely on a steady supply of oxygen and glucose via the circulatory system. Autoregulation occurs so that neither too little (hypoperfusion) nor too much (hyperperfusion) supply occurs. To maintain adequate function as long as possible, there are compensatory mechanisms that take place in response to disrupted blood flow, including the dilation of arteries to increase cerebral blood flow (CBF) and increasing the amount of oxygen extracted from the blood. Once the oxygen supply is depleted, ischemia is said to begin and the neuron undergoes a well-described series of pathophysiological steps in metabolic function before infarction takes place. In human stroke, the CBF is so low in the ischemic core that infarction occurs rather quickly, but can be high enough in the surrounding region, known as the ischemic penumbra, so that hemodynamic rescue via breakdown of the clot with drugs (e.g., rTPA) or mechanical removal of clot may be achieved. Because it takes time for the ischemic process to run its course, the symptoms of an ischemic stroke can often present as minor syndromes and then progress over time to more disabling clinical events.

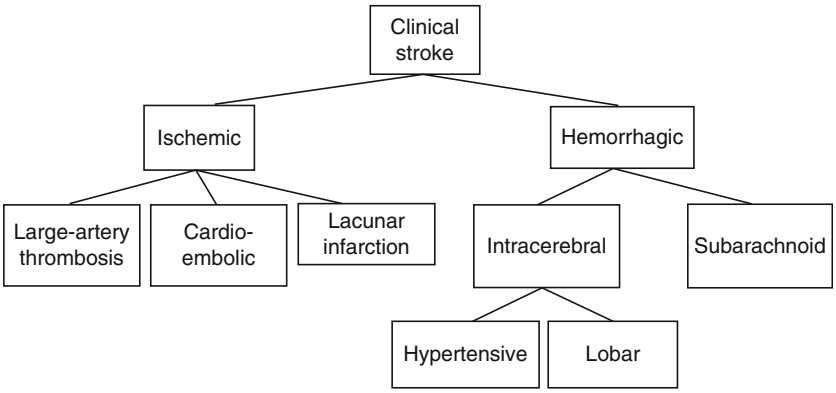
Before the advent of modern imaging (CT and MRI), it was thought that if the signs and symptoms of an ischemic episode resolved in less than 24 h that a permanent injury had not taken place, so that a transient ischemic event (TIA) was felt to have occurred. But over the last decade, advances in MRI sequences (e.g., diffusion-weighted imaging, or DWI) have enabled detection of by-products of infarction with extraordinary sensitivity, even in the setting of a brief clinical event. Thus, the 2009 guidelines of the American Heart Association now state that any neurological event of presumed vascular origin that produces evidence of infarction on imaging is classified as an ischemic stroke.

There are two general classes of stroke as depicted in Fig. 1: ischemic strokes that are caused by the blockage of the cerebral blood supply, resulting in infarction (tissue death), and hemorrhagic strokes in which blood is released from either arterial or, less commonly, venous vessels.

Ischemic Stroke

The three primary mechanisms that cause ischemic stroke are large vessel thrombosis, cardio-embolism, and lacunar infarction, together accounting for 80% of all strokes. Thrombosis is an obstruction of blood flow due to a blood clot that is formed locally within a blood vessel, causing the lumen of the vessel to be occluded, or narrowed. It often occurs at a site of atherosclerosis in which fatty materials form plaques that adhere to the inner wall of the vessel. Platelets then adhere to plaque crevices, forming clumps that facilitate deposit of fibrin, thrombin, and clot. Atherosclerosis occurs most frequently in larger vessels, so that at the point when blood becomes impeded, either partially (stenosis) or completely (occlusion), the downstream region of the brain supplied by that vessel experiences a failure of blood flow perfusion, resulting in ischemia and then infarction. For example, a critical stenosis in the left internal carotid artery creates perfusion failure to the entire distribution territories of the left middle and anterior cerebral arteries, producing a devastating stroke involving most of the left cerebral hemisphere. Sometimes a fragment of a thrombus in a large artery will break off and in a process known as embolism will be carried downstream and plug vessels within the brain, causing cerebral infarction.

A frequent source of embolism is the heart, with 90% of the embolic material going into the carotid arteries and, in turn, into the distribution territories of the MCA. Cardio-embolic stroke therefore represents the



Stroke. Figure 1 The major stroke subtypes

most common cause of focal neuropsychological deficits arising from cerebrovascular disease.

Lacunar infarcts occur in smaller vessels frequently damaged by hypertension where increased arterial pressure leads to deposits of fibrin in the vessel wall. Less commonly, small vessels can be occluded by atherosclerotic plaque or embolism. Most lacunes occur in small vessels within the lenticulostriate system, affecting the basal ganglia and adjacent white matter, as well in the deep penetrators within the white matter of the centrum semiovale within the frontal lobe.

There are several hereditary diseases for which stroke is a known clinical manifestation. Among these include cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy, or CADASIL, which is an inherited small artery disease of the brain caused by mutations of the NOTCH3 gene on chromosome 19. This condition produces a vascular dementia of subcortical type because of its affect on the small penetrating arteries in the white matter. While the dementia in CADASIL does not usually arise until the 40s, cognitive dysfunction can begin in childhood in mitochondrial encephalomyopathy lactic acidosis and stroke-like episodes, or MELAS, which can involve both cortical and subcortical regions. MELAS is caused by mutations in mitochondrial DNA that lead to vascular lesions. There is no one particular clinical pattern in this syndrome, with cognitive effects ranging from mild dysfunction to mental retardation, and behavioral sequelae that can resemble a schizophrenic-like picture.

Hemorrhagic Stroke

Bleeding that occurs within the brain parenchyma as a result of leaking or frank rupture of blood vessel walls is referred to as intraparenchymal, parenchymatous, or intracerebral hemorrhage. The clinical effects of hemorrhage within the substance of the brain arise because of the displacement of brain tissue from the mass effect of the clot, and diagnosis is most frequently made on CT scanning. Often occurring in the penetrating arteries of the basal ganglia and thalamoperforant arteries, one common subtype of bleeding arises often among those with hypertension, referred to as hypertensive intracerebral hemorrhages. Despite the relatively small size of the associated clots, the behavioral and physical syndromes often occur suddenly and with devastating consequences. Intracerebral hemorrhage from larger vessels, also known as lobar hemorrhage, are most commonly caused by cerebral amyloid angiopathy, cerebral arteriovenous malformations (AVMs), and cerebral aneurysms.

The most fatal of all strokes are the subarachnoid hemorrhages (SAH) that are most commonly caused by the rupture of a cerebral aneurysm, when blood is deposited into the subarachnoid space between the arachnoid membrane and pia matter that surround the brain. The neurologic and neuropsychological effects can come about not only because of the diffuse effects of raised intracranial pressure from swelling but also because of infarction from vasospasm of cerebral vessels. The Hunt and Hess, and the Fischer Scales are commonly used to grade the severity of an SAH.

The most commonly used clinical instruments to measure the effects of stroke are the NIH Stroke Scale, assessing impairment, and the modified Rankin Scale and the Barthel Scale which evaluate more functional outcomes. Quality of life can be studied either with the Stroke Specific Quality of Life Scale (SSQoL) or the Stroke Impact Scale (SIC). These scales can also be used for intracerebral hemorrhages.

Epidemiology of Stroke

Each year about 795,000 people experience a new or recurrent stroke, according to the 2009 update of Heart Disease and Stroke of the American Heart Association (American-Heart-Association, 2009). About 600,000 of these are first-time attacks, and 185,000 are recurrent attacks. Each year, about 55,000 more women than men have a stroke. Men's stroke incidence rates are greater than women's at younger ages but not at older ages. The male/female incidence ratio is 1.25 at ages 55–64, 1.50 for ages 65–74, 1.07 at 75–84, and 0.76 at 85 and older. Blacks have almost twice the risk of first-ever stroke compared with whites. The age-adjusted stroke incidence rates at ages 45–84 are 6.6 per 1,000 population in black males, 3.6 in white males, 4.9 in black females, and 2.3 in white females. The Brain Attack Surveillance in Corpus Christi project (BASIC) found an increased incidence of stroke among Mexican Americans compared with non-Hispanic whites. The crude cumulative incidence was 168/10,000 in Mexican Americans and 136/10,000 in non-Hispanic whites. Specifically, Mexican Americans have an increased incidence of intracerebral hemorrhage and subarachnoid hemorrhage compared with non-Hispanic whites, as well as an increased incidence of ischemic stroke and TIA at younger ages. Of all strokes, 87% are ischemic, 10% are intracerebral hemorrhage, and 3% are subarachnoid hemorrhage.

Natural History, Prognostic Factors, and Outcomes of Stroke

Stroke is the third leading cause of mortality in the USA (143,400 in 2005), but the leading cause of disability (Feigin, Lawes, Bennett, & Anderson, 2003), with more than 1.1 million American adults reporting functional limitations in 1999 as a result of stroke (Rosamond et al., 2008). The Framingham Heart Study showed that among stroke survivors at 6 months, 50% had some hemiparesis, 26% were dependent in their ADLs, and 19% had aphasia (Kelly-Hayes et al., 2003). About one out of every three patients has some form of stroke rehabilitation (Centers for Disease Control and Prevention (CDC 2007), "Outpatient Rehabilitation Among Stroke Survivors – 21 States and the District of Columbia, 2005").

Most patients have at least some degree of recovery after ischemic stroke, especially first-time stroke, with less-favorable outcomes following a recurrent event. Most recovery occurs within the initial 90 days after the stroke onset, but it is also known that recovery can still occur years after injury. Historically, the most highly predictive factors have been age, initial syndrome severity, and lesion volume, assuming no recurrence or new cardiovascular event, such as myocardial infarction. Patients who are sedentary do less well after stroke. Although there is some controversy about the effectiveness of different forms of rehabilitation, the general rule is that patients will have better functional recoveries if behavioral intervention is started sooner rather than later after the stroke onset. Major depression is also a common occurrence after stroke, and studies have shown that depression is associated with excess stroke disability, cognitive impairment, extended hospitalization, diminished social reintegration, poorer quality of life, and mortality and morbidity.

The greatest likelihood of recurrent stroke is soon after a prior one, with large, community-based studies indicating rates up to 4% in the first 30 days. Although there are unmodifiable risk factors, such as age, gender, ethnicity, and heredity, changes in daily behavior are known to affect risk of both initial and recurrent stroke. Targets for reduction of stroke occurrence and disability include hypertension, cardiovascular disease (e.g., atrial fibrillation), diabetes, hyperlipidemia, carotid stenosis, cigarette smoking, and alcohol abuse. Of special interest lately is the tetrad of insulin resistance, hypertension, obesity, and elevated lipids, now called metabolic syndrome, which poses significant risk for both stroke and cardiovascular disease.

Psychology and Neuropsychology of Stroke

Although interest in cognitive and behavioral function also date back to the Egyptians and Greeks, many historians point to the mid-nineteenth century work of Paul Broca as seminal in putting forth the notion that there is laterality to brain function, and specifically the importance of the left hemisphere for language function. For more than 100 years, cerebral localization of cognitive and behavioral deficits was only possible on postmortem examination, and so there was little understanding of how the brain changed over time in any single patient as function evolved. But with the advent of CT in the mid-1970s and MRI in the early 1980s, new insights emerged with regard to brain-behavior relationships, first with regard to greater neuroanatomic understanding and

more recently to functional principles and to the presence of networks.

There is a staggering literature that has sought to describe the vast array of neuropsychological deficits after stroke, mainly cerebral infarction. This is because ischemic stroke is such a prevalent neurological condition, images are so readily available, abnormalities of cognitive and behavioral function are so frequently part of the clinical presentation, and deficits most commonly occur in well-described territories of the three major cerebral arteries. Furthermore, stroke provides a unique model of cognitive syndromes produced by a definable lesion (on imaging) that is relatively circumscribed soon after onset; few causes of neuropsychological deficits produce such a relatively static lesion at such an early point.

Table 1 below summarizes the most commonly described deficits, based on the associated location in the cerebral circulation where the events most often occur. The specific nature and severity of deficits for a given ischemic stroke event depend on the extent of cortical and/or subcortical involvement. In contrast, intracerebral hemorrhage can produce clot that impinges on brain tissue in more than one arterial territory, so that the characterization of impairment is less predictable.

Finally, the clinical features associated with subarachnoid hemorrhage are the least well understood. The spectrum of disorders measured after hemorrhage has included verbal memory deficits, visual disorders, and defects in information processing. Frontal-lobe dysexecutive syndromes are also commonly reported. One of the complicating factors in determining the cognitive consequences of SAH is that the majority of patients have also received either surgical (clipping the neck of the aneurysm) or endovascular (placing coils in the aneurysm via a catheter) treatment, the effects of which on cognitive function remains in debate. It had long been assumed that one determining factor has been the location of the bleed. As the most common site of SAH, rupture of the ACoA has been proposed by some investigators to be associated with the triad of an amnestic disorder, confabulation and alteration of personality, collectively labeled the "ACoA syndrome." Comparisons of neurocognitive outcomes of patients with ruptured aneurysms in this and other regions, however, have not yielded consistent behavioral differences.

In addition to cognitive impairments, stroke can produce different forms of emotional distress. As noted above, depression is frequently observed after

Stroke. Table 1 Common neuropsychological deficits after ischemic stroke

Left middle cerebral artery (LMCA)	Right middle cerebral artery (RMCA)
Broca's aphasia (upper division)	Left visual neglect
Wernicke's aphasia (lower division)	Constructional apraxia
Conduction aphasia (upper division)	Dressing apraxia
Global aphasia (upper and lower divisions)	Motor impersistence
Alexia with agraphia	Amusia
Gerstmann's syndrome	Anosognosia
Apraxia of speech	Aprosodia
Ideomotor apraxia	Tactile agnosia
Acalculia	Poststroke delirium
Dysarthria (upper-motor neuron type)	Reduplicative paramnesia
Color anomia	Dysarthria (upper-motor neuron type)
Tactile agnosia	Topographical disorientation
Vertebrobasilar/posterior cerebral artery (PCA)	
Alexia without agraphia	
Verbal Amnesia (posterior choroidal artery)	
Achromatopsia	
Anarthria (cerebellum)	
Color agnosia	
Simultanagnosia	
Dysarthria (coordination type)	
Anterior cerebral artery (ACA)	
Alien hand syndrome	
Akinetic mutism	
Bilateral syndromes	
Abulia	
Labile affect	
Visual agnosia	
Cortical blindness	
Prosopagnosia	
Optic aphasia	
Balint's syndrome	

stroke. Reported incidence ranges widely (likely as a result of methodologic differences among the various studies), but pooled estimates suggest that approximately one-third of stroke survivors exhibit symptoms of depression at some point after stroke (Hackett, Yapa, Parag, & Anderson, 2005). While early studies found depression to be more common after left than right hemisphere lesions, more recent findings have not supported such laterality. Poststroke anxiety is less common than depression but disabling nonetheless, since many survivors fear recurrence or shy away from resumption of prior social roles in their disabled state. Pharmacotherapy is frequently an effective treatment for both depression and anxiety, but there is surprisingly little available information about the efficacy of psychological treatments.

The neuropsychologist must also recognize that stroke is a family affair, with potential for particular consequences – physical, social, emotional, and financial – for primary caregivers. These issues often do not arise during acute stroke hospitalization when matters of survival and medical management have priority, and even during inpatient rehabilitation during which there is a highly structured environment. After patients are discharged home, however, caregivers often have new and often unexpected challenges. A readjustment of marital roles may be necessary, and there may be changes in the survivor's capacity for sexual function, and limitations on driving and vocational or avocational activities to confront. Proactive education and supportive counseling may be useful, and long-term follow-up is advisable, when feasible.

Treatment of Stroke

Once a stroke has run its course, resulting in infarction of tissue, there is no current treatment for the restoration of neuronal functional. When stroke symptoms first arise, however, when still-surviving tissue may lie in the ischemic penumbra that surround a region of infarction, there are two FDA-approved treatments that can sometimes provide restoration of flow to rescue dysfunctional neurons and local supportive tissue. If patients come to the emergency department within 4.5 h of symptom onset, they may undergo intravenous injection of the agent rTPA that can break up, or lyse the clot. In cases when it is felt that patients are likely to have devastating syndromes if left untreated, then a device can be placed with a catheter just before the blockage and a corkscrew-like wire can be inserted into the clot which is then extracted from the vessel. Intervention with rTPA and the clot-extracting device carry high risk, and so there are strict eligibility criteria for their use.

Once the completed stroke occurs, there is a major effort to reduce the likelihood of a recurrent stroke. Patients are usually given some form of antithrombotic drug therapy that can be some combination of anticoagulation medication (e.g., warfarin) and/or an agent to reduce platelet aggregation, such as aspirin. There is also patient education designed to reduce modifiable risk factors, such as obesity, lipids, hypertension, diabetes, smoking, and alcohol intake.

For stroke survivors in the middle range of impairment, rehabilitation therapies are typically offered; those with severe strokes may benefit less well from such interventions, while those who are only mildly affected might not require them. Neuropsychologists working in rehabilitation settings play multiple roles in the treatment of stroke survivors (Caplan, 2010). In addition to conducting neuropsychological evaluations, they provide individual and family counseling, consult with other team members about the management of behavioral problems such as poor motivation and impaired awareness of deficits (anosognosia), provide cognitive rehabilitation, and advise about post-discharge planning.

Cross References

- ▶ Abulia
- ▶ Acalculia
- ▶ Achromatopsia
- ▶ Akinetic Mutism
- ▶ Alexia
- ▶ Alien Hand Syndrome
- ▶ Amnestic Disorder
- ▶ Amusia
- ▶ Anarthria
- ▶ Anosognosia
- ▶ Anterior Cerebral Artery
- ▶ Apraxia of Speech
- ▶ Aprosodia
- ▶ Balint's Syndrome
- ▶ Broca's Aphasia
- ▶ Carotid Angiography

- ▶ Color Agnosia
- ▶ Color Anomia
- ▶ Conduction Aphasia
- ▶ Constructional Apraxia
- ▶ Cortical Blindness
- ▶ Dysarthria
- ▶ Gerstmann's Syndrome
- ▶ Global Aphasia
- ▶ Hemispatial Neglect
- ▶ Lability
- ▶ Motor Impersistence
- ▶ Posterior Cerebral Artery
- ▶ Prosopagnosia
- ▶ Simultanagnosia
- ▶ Spastic Gait
- ▶ Tactile Agnosia
- ▶ Topographical Disorientation
- ▶ Visual Agnosia
- ▶ Wernicke's Aphasia

References and Readings

American-Heart-Association. (2009). *Heart disease and stroke statistics — 2009 update.* Dallas, Texas: ©2009, American Heart Association.

Caplan, B. (2010). Rehabilitation psychology and neuropsychology with stroke survivors. In R. Frank, M. Rosenthal, & B. Caplan (Eds.), *Handbook of rehabilitation psychology* (2nd ed.). Washington, DC: American Psychological Association.

Centers for Disease Control and Prevention (CDC). (2007). Outpatient rehabilitation among stroke survivors – 21 states and the district of Columbia, 2005. *MMWR Morbidity Mortality Weekly Report, 56*(20), 504–507.

Feigin, V. L., Lawes, C. M., Bennett, D. A., & Anderson, C. S. (2003). Stroke epidemiology: a review of population-based studies of incidence, prevalence, and case-fatality in the late 20th century. *Lancet Neurol, 2*(1), 43–53.

Festa, J. R., & Lazar, R. M. (Eds.). (2009). *Neurovascular neuropsychology.* New York: Springer.

Hackett, L. M., Yapa, C., Parag, V., & Andersom, C. S. (2005). Frequency of depression afte stroke. *Stroke, 36*, 1330.

Kelly-Hayes, M., Beiser, A., Kase, C. S., Scaramucci, A., D'Agostino, R. B., & Wolf, P. A. (2003). The influence of gender and age on disability following ischemic stroke: the Framingham study. *J Stroke Cerebrovascular Diseases, 12*(3), 119–126.

Mendoza, J., & Foundas, A. L. (2008). *Clinical neuroanatomy: a neurobehavioral approach.* New York: Springer.

Mohr, J. P., Choi, D. W., Grotta, J. C., Weir, B., & Wolf, P. A. (Eds.). (2004). *Stroke: pathophysiology, diagnosis, and management.* Philadelphia: Churchill-Livingstone.

Rosamond, W., Flegal, K., Furie, K., Go, A., Greenlund, K., Haase, N., et al. (2008). Heart disease and stroke statistics – 2008 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation, 117*(4), e25–e146.

Stroke Activity Scale

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Synonyms

SAS

Definition

The Stroke Activity Scale (SAS) (Holbrook & Skilbeck, 1983) assesses activities of daily living beyond the limited self-care assessments previously available. The author's-goal was to develop a brief scale of lifestyle to determine rehabilitation goals. The SAS is a five-item scale that was developed by physiotherapists as a measure of motor function at the level of disability in stroke patients for use in the clinical setting. It consists of five items (getting out of bed, sitting balance, sitting to standing, stepping and walking, and bringing a glass to the mouth) that take fewer than 10 min to administer. The score levels of the SAS make the distinction between "normal" and "adaptive" movement. Scoring of the SAS items uses a 4-point ordinal scale ranging from 0 = unable to 1 = attempts with adaptive movement to 3 = achieves with adaptive movement to 4 = achieves normal or nearly normal movement. The SAS is scored simply by summing the five-item scores, allowing for a possible range of scores between 0 ± 16. The two subscales defined by a factor analysis are gender-sensitive.

Current Knowledge

Horgan, Finn, O"Regan, and Cunningham (2003) investigated the SAS's internal consistency, inter-rater and intra-rater reliability. The authors found the reliability for total scores was excellent, the reliability for individual item scores was good (kappa ≥ 0.7), and an internal consistency reliability using Cronbach's alpha was also good when testing 12 hospital-based patients. The SAS is a reliable instrument for hospitalized stroke patients. It can be administered in less than 10 minutes and requires minimal equipment and training.

Cross References

- ▶ Fugl-Meyer Assessment of Sensorimotor Impairment
- ▶ Motor Assessment Scale
- ▶ Rivermead Motor Assessment Scale
- ▶ Stroke Impact Scale
- ▶ Wolf Motor Function Test

References and Readings

Holbrook, M., & Skilbeck, C. E. (1983). An activities index for use with stroke patients. *Age and Ageing,* May, *12*(2), 166–170.

Horgan, N. F., Finn, A. M., O'Regan, M., & Cunningham, C. J. (2003). A new stroke activity scale-results of a reliability study. *Disability and Rehabilitation,* Mar 18, *25*(6), 277–285.

Stroke Impact Scale

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Synonyms

SIS; SIS 2.0; SIS 3.0; SIS-16

Description

The Stroke Impact Scale (SIS) is a quality of life measure designed specifically for patients with stroke. The instrument is a self-report questionnaire assessing eight domains: strength, hand function, activities of daily living (ADL)/instrumental ADL (IADL), mobility, communication, emotion, memory and thinking, and social participation. Each item is rated on a scale from 1 to 5 (indicating either high or low quality of life, depending on the specific question) and an additional item assesses the patient's global perception of percent recovery.

Historical Background

Duncan et al. developed the SIS in 1999 using the input from patients and caregivers to assess the health-related quality of life, many aspects of which were not included in previous stroke-specific outcome measures. The first published version of the SIS (version 2.0) contained 64 items. After Rasch analysis determined that three SIS items did not assess their proposed constructs, these items were removed to form the SIS version 3.0 in 2003. A shorter version focusing on physical function that includes only 16 of the original 64 questions (SIS-16) has also been validated as have several SIS language translations and the use of the SIS by caregiver proxy.

Psychometric Data

The SIS has demonstrated good internal consistency (Cronbach α: approximately 0.9 for each domain) and test-retest reliability (ICC: 0.70–0.92 for all domains except emotion: 0.57). Convergent validity has been confirmed by moderate to strong correlations (0.44–0.84) with established outcome measures designed to assess the same construct as individual SIS domains. SIS domain scores have also been shown to discriminate across four Rankin levels, thus confirming discriminant validity. In addition, the SIS has demonstrated sensitivity to change over time.

Rasch analysis has revealed that all SIS domains are unidimensional and have good reliability. However, the communication, memory, and emotion domains exhibit a substantial ceiling effect, while the remaining domains, which comprise a composite physical domain, have an excellent range of item difficulty.

The SIS version 2.0, SIS version 3.0, SIS-16, SIS by caregiver proxy, and SIS foreign language translations all appear to have comparable psychometric properties.

Clinical Uses

Although originally designed for persons with mild to moderate stroke, the SIS is now used (and has demonstrated good psychometric properties) as an outcome measure for persons with a wide range of disability due to stroke, both in clinical and research settings. Each domain is scored individually and a total score is also derived. The scoring algorithm is the same as that used for the SF-36 (cross-reference SF-36 entry) and all scores range from 0 to 100. Minimum clinically important difference, the smallest change that is meaningful for an individual patient, has been estimated at 10–15 points for SIS domains and total score.

Cross References

- ▶ Ceiling Effect
- ▶ Quality of Life

- ▶ Sensitivity
- ▶ Stroke

References and Readings

Carod-Artal, F. J., Coral, L. F., Trizotto, D. S., & Moreira, C. M. (2008). The stroke impact scale 3.0: Evaluation of acceptability, reliability, and validity of the Brazilian version. *Stroke; A Journal of Cerebral Circulation, 39*(9), 2477–2484.

Carod-Artal, F. J., Ferreira Coral, L., Stieven Trizotto, D., & Menezes Moreira, C. (2009). Self- and proxy-report agreement on the stroke impact scale. *Stroke; A Journal of Cerebral Circulation, 40*(10), 3308–3314.

Duncan, P. W., Bode, R. K., Min Lai, S., Perera, S., & Glycine Antagonist in Neuroprotection Americans Investigators. (2003). Rasch analysis of a new stroke-specific outcome scale: The stroke impact scale. *Archives of Physical Medicine and Rehabilitation, 84*(7), 950–963.

Duncan, P. W., Lai, S. M., Bode, R. K., Perera, S., & DeRosa, J. (2003). Stroke impact scale-16: A brief assessment of physical function. *Neurology, 60*(2), 291–296.

Duncan, P. W., Wallace, D., Lai, S. M., Johnson, D., Embretson, S., & Laster, L. J. (1999). The stroke impact scale version 2.0. evaluation of reliability, validity, and sensitivity to change. *Stroke; A Journal of Cerebral Circulation, 30*(10), 2131–2140.

Edwards, B., & O'Connell, B. (2003). Internal consistency and validity of the stroke impact scale 2.0 (SIS 2.0) and SIS-16 in an Australian sample. *Quality of Life Research: An International Journal of Quality of Life Aspects of Treatment, Care and Rehabilitation, 12*(8), 1127–1135.

Stroke-Adapted Sickness Impact Profile

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Synonyms

SA-SIP; SA-SIP30

Definition

An adaptation of the full version of the 136-item Sickness Impact Profile (SIP). The Stroke-Adapted Sickness Impact Profile (SA-SIP30) contains 30 items within 8 subscales: body care and movement, social interaction, mobility, communication, emotional behavior, household management, alertness behavior, and ambulation. Scoring for the SA-SIP30 is conducted in the same manner as for the full version SIP and yields the same subscales, dimensions, and total score. Total scores range from 0% to 100% with higher scores corresponding to worse health.

Current Knowledge

In a sample of 319 individuals at 6 months poststroke, the homogeneity of the SA-SIP30 was very good (Cronbach's alpha = 0.85). The SA-SIP30 explained 89–91% of the variance in the scores of the original SIP (136 items). The SA-SIP30 showed good discriminant validity by differentiating between patients with lacunar infarctions and patients with cortical or subcortical lesions (Van Straten et al., 1997).

In a sample of 418 individuals 6 months poststroke, the physical dimension and total scores of the SA-SIP30 and SIP were associated with the Barthel Index and Rankin Scale, but not the EuroQol (a health-related quality of life measure). Most individuals with SA-SIP30 total scores greater than 33 or SIP total scores greater than 22 had poor health profiles (Van Straten, deHaan, Limburg, & van der Bos, 2000).

Cross References

- ▶ Barthel Index
- ▶ EuroQuol
- ▶ Rankin Scale
- ▶ Sickness Impact Profile

References and Readings

Van Straten, A., deHaan, R. J., Limburg, M., Schuling, J., Bossuyt, P. M., & van den Bos, G. A. M. (1997). A stroke-adapted 30-item version of the Sickness Impact Profile to assess quality of life (SA-SIP30). *Stroke, 28*, 2155–2161.

Van Straten, A., deHaan, R. J., Limburg, M., van den Bos, G. A. M. (2000). Clinical meaning of the Stroke-adapted Sickness Impact Profile-30 and the Sickness Impact Profile-136. *Stroke, 31*, 2610–2615.

Stroop Color and Word Test, Children's Version

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Description

The Stroop Color and Word Test, Children's Version (2003), is designed to measure the ability to inhibit a prepotent reading response in order to engage a naming response. According to the manual, when used with children, the test can also provide information regarding the development and dominance of the reading system. This version of the Stroop paradigm uses three cards with 100 items each. On the first card, the child is asked to read a list of color words (e.g., red and green) printed in black ink. The second card contains columns of nonword stimuli (XXXX) printed in different colors and the child is asked to name the color of each stimulus. On the final card, color words are printed in colors different from the word (e.g., *blue* printed in green ink) and the child is required to name the color rather than read the word. In each part, the child is given 45 s to read or name as many items as possible.

The manual suggests the test can be administered in group format, during which children perform the task silently. However, use of the group format should be restricted to high-functioning individuals (e.g., college students, older children, or adults in a research project); therefore this method is unlikely to be used in the context of neuropsychological evaluation. The entire test takes no longer than 10 min to administer. Four scores are derived: word reading, color naming, color-word naming, and interference (in children, this can be calculated by subtracting the raw score for color-word naming from color naming). T scores are provided based on normative data from 182 children aged 5–14. The demographic characteristics of the normative sample are described only cursorily.

Historical Background

The Stroop Color and Word Test has its roots in Wilhelm Wundt's lab, where James Cattell (1886) discovered that it takes longer to name colors or objects than it does to read words or letters. Half a century later, John Riley Stroop thought to explore how interference might influence this effect. In his dissertation, Stroop gave participants a list of color words printed in a different colored ink (e.g., *red* printed in green ink) and measured the time it took for them to name the color of ink. Over time, the original task has been modified along several different dimensions, including the number of colors used, the presentation of colors, number of items, and scoring criteria. The version presented by Golden in 1978 is widely used and is identical to the Children's Version described here. The Children's Version has been updated to provide normative data for children ages 5–14. Other versions of the Stroop paradigm have been modified for use with children, including subtests of the D-KEFS and the Cognitive Assessment System.

Psychometric Data

The authors of the test manual present little psychometric data regarding the use of the test in samples of children. The manual indicates that the test–retest reliability of the Stroop paradigm is fairly robust across test versions (nearly all $rs > 0.8$). Using a Dutch sample, Neyens and Aldenkamp (1996) examined the test–retest reliability for children ages 4–12 and reported similar results ($r = 0.81$ for word reading, $r = 0.87$ for color naming, $r = 0.90$ for color-word reading, and $r = 0.81$ for the interference score). MacLeod (1991) suggests that, across all ages and versions, the Stroop paradigm displays good validity. However, the Stroop Color and Word Test may not demonstrate good construct validity in younger children. Younger children, as well as those individuals with reading difficulties, have not developed reading proficiency and therefore will not exhibit the same scoring patterns as older children and adults (e.g., they may produce a low interference score because reading is not a prepotent response for them).

Clinical Uses

Traditionally, the Stroop paradigm is used to assess executive functioning, particularly working memory, attention, and response inhibition. In addition, the word reading and color naming scores of the Stroop task tend to correlate with measures of speed of information processing (e.g., ▶ Trail Making Test – A, ▶ verbal fluency). Therefore, the Stroop task may prove useful in assessing populations with frontal lobe dysfunction or slowed processing speed.

The Stroop Color and Word Test, Children's Version (2003) is fairly new and few studies have cited the new manual. Research using the original 1978 Golden version has been performed in a number of clinical populations, including children with: attention-deficit/hyperactivity disorder (ADHD), learning disabilities, traumatic brain injury, epilepsy, cerebral palsy, autism, mental retardation, bipolar disorder, and phenylketonuria (PKU) (e.g., Golden & Golden, 2002; Homack & Riccio, 2004).

The manual warns that the interpretation of scores from pediatric clients must take into account their developmental stage, as well as possible confounding factors (e.g., emotional problems and learning disabilities). Descriptions of common scoring patterns as well as their likely clinical interpretation are provided in the manual, but users are reminded that "no single etiological conclusion can be reached from Stroop scores alone, no matter the pattern."

Cross References

- ▶ Attention
- ▶ Cognitive Assessment System
- ▶ Delis-Kaplan Executive Function System (D-KEFS)
- ▶ Executive Functioning
- ▶ Interference
- ▶ Stroop Color Word Test (Adult)

References and Readings

Cattell, J. M. (1886). The time it takes to see and name objects. *Mind, 11*, 63–65.

Golden, C. J. (1978). *Stroop Color and Word Test: A manual for clinical and experimental uses.* Wood Dale, IL: Stoelting Co.

Golden, Z., & Golden, C. J. (2002). Patterns of performance on the Stroop Color and Word Test in children with learning, attentional, and psychiatric disabilities. *Psychology in the Schools, 39*(5), 489–495.

Golden, C. J., Freshwater, S. M., & Golden, Z. (2003). *Stroop Color and Word Test, Children's version for Ages 5–14: A manual for clinical and experimental uses.* Wood Dale, IL: Stoelting Co.

Homack, S., & Riccio, C. A. (2004). A meta-analysis of the sensitivity and specificity of the Stroop Color and Word Test with children. *Archives of Clinical Neuropsychology, 19*, 725–743.

Macleod, C. M. (1991). Half a century of research on the Stroop effect – an integrative review. *Psychological Bulletin, 109*(2), 163–203.

Neyens, L. G. J., & Aldenkamp, A. P. (1996). Stability of cognitive measures in children of average ability. *Child Neuropsychology, 2*, 161–170.

Stroop Color Word Test (adult)

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Synonyms

SCWT

Description

The purpose of the SCWT is to assess the ability of the individual to inhibit a habitual response for one that is less readily available. First introduced in 1935 by J. R. Stroop, multiple versions of the Stroop Test have been developed, all of which consist of either a 10 × 10 grid (read across the rows) or 5 × 20 grid (read down the columns). The 5 × 20 grid version was chosen for standardization (Golden and Freshwater, 2002) because impaired individuals had difficulty reading across the rows, tending to lose their place. The Word component consists of color words (i.e., red, green, & blue) in black type. The Color component consists of colored bars (e.g., xxxx) that are colored red, green, or blue. The Color-Word component consists of color words, but each one is printed in a color that differs from the written word (e.g., the word "red" will be printed in green or blue, but not in red). Examinees are given 45 seconds to read the items going down the columns. Four scores are derived, three of which are based on the number of correct responses to each set of stimuli. A fourth score, the Interference Score, is derived via a formula based on the difference between the Color-Word score and the Word and Color scores.

Normative data was initially collected by Golden (1978) and updated using an additional 300 normal individuals for the most recent update (Golden and Freshwater, 2002). The age range of the SCWT normative sample is 15–90 (mean = 38.23, SD = 15.42), with education ranging from 2–20 years (mean = 12.04, SD = 3.84). The current scoring system involves prediction of a normal individual's performance based on age and years of education. The difference between the obtained and predicted scores is then converted to T-scores.

Current Knowledge

Reliability of the Stroop test has been examined in multiple studies. Test-retest reliability has been examined for

periods ranging from 1 minute to 10 days and has generally been satisfactory for all versions of the test (Lezak, Howieson, and Loring, 2004). Practice effects have varied across studies, with some showing little improvement and others showing considerable gains (McCaffrey, Duff, and Westervelt, 2000; Strauss, Spreen, and Sherman, 2006).

Gender effects have been noted for the SCWT. Women have consistently performed better on Color naming (Baroun and Alansari, 2006; Jensen, 1965), but no gender differences were noted for the Word naming. Gender differences on the Color-Word component are more equivocal, with some researchers finding that women performed better than men (Peretti, 1971) while others found no statistically significant differences (Alansari and Baroun, 2004).

Age-related differences have been demonstrated, with older adults performing more slowly than younger adults and demonstrating greater relative interference effects (Cohn, Dustman, and Bradford, 1984; Panek, Rush, and Slade, 1984).

Cultural differences have also been explored. Rosselli, Ardila et al. (2002) found that Spanish-English bilinguals were 5% to 10% slower than monolingual individuals (i.e., English or Spanish only) on the Color-Word Component, although one-way ANOVAs were not significant. There was a significant effect of language proficiency, but no difference in performance in balanced bilinguals. Moering, Schinka et al. (2002) examined the performance of 236 community-dwelling older African-American adults (60–84 years) and found significant age, education, and gender effects. They developed normative tables to adjust for the influence of these factors.

Neuropsychological performance on the SCWT has been thoroughly researched, and comprehensive reviews are available (Golden and Freshwater, 2002; Lezak et al., 2004; Strauss et al., 2006). Briefly, regional cerebral blood flow (rCBF) and positron emission tomography (PET) studies indicated preferential activation of the frontal lobes, including the left anterior cingulated cortex, supplementary motor area, left premotor cortex, and orbitofrontal cortex (Bench et al., 1993; Pardo et al., 1990). Consistent with the neuroanatomical findings, individuals with frontal lobe versus posterior lesions and left versus right hemisphere lesions performed more poorly on Word, Color fluency, and Interference components (Lezak et al., 2004). With persons with dementia of mild to moderate severity, performance on the interference trial was significantly slower (Bondi, Serody, Chan et al., 2002), but the interference effect diminished in later stages of dementia as response slowing became more generalized.

Cross References

- ▶ Frontal Lobes
- ▶ Stroop Neuropsychological Screening Test (adult)

References and Readings

Alansari, B., & Baroun, K. (2004). Gender differences and cultural performance differences on the Stroop Color and Word Test: A comparative study. [Electronic version] *Social Behavior and Personality.* Retrieved May 15, 2008 from http://findarticles.com/p/articles/mi_qa3852/is_200401/ai_n9379428.

Baroun, K., & Alansari, B. (2006). Gender differences in performance on the Stroop test. [Electronic version] *Social Behavior and Personality.* Retrieved May 15, 2008 from http://findarticles.com/p/articles/mi_qa3852/is_200601/ai_n17187418/pg_1.

Bench, C. J., Frith, C. D., Grasby, P. M., Friston, K. J., Paulesu, E., Frackowiak, R. S. J., & Dolan, R. J. (1993). Investigations of the functional anatomy of attention using the Stroop test. *Neuropsychologia, 31*, 907–922.

Bondi, M. W., Serody, A. B., Chan, A. S., et al. (2002). Cognitive and neuropathologic correlates of Stroop Color-Word Test performance in Alzheimer's disease. *Neuropsychology, 16*, 335–343.

Cohn, N. B., Dustman, R. E., & Bradford, D. C. (1984). Age-related decrements in Stroop Color Test performance. *Journal of Clinical Psychology, 40*, 1244–1250.

Golden, C. J. (1978). *A manual for the stroop color and word test.* Chicago, IL: Stoetling Company.

Golden, C. J., & Freshwater, S. M. (2002). *Stroop color and word test: A manual for clinical and experimental uses.* Wood Dale, IL: Stoetling Company.

Jensen, A. R. (1965). Scoring the stroop test. *Acta Psychologia, 24*, 398–408.

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

McCaffrey, R. J., Duff, K., & Westervelt, H. J. (2000). *Practitioner's guide to evaluating change with intellectual assessment instruments.* NY: Kluwer Academic/Plenum Press.

Moering, R. G., Schinka, J. A., Mortimer, J. A., & Graves, A. B. (2004). Normative data for elderly African Americans for the Stroop Color and Word Test. *Archives of Clinical Neuropsychology, 19*, 61–71.

Panek, P. E., Rush, M. C., & Slade, L. A. (1984). Locus of the age-Stroop interference relationship. *Journal of Genetic Psychology, 145*, 209–216.

Pardo, J. V., Pardo, P. J., Janer, K. W., & Raichle, M. E. (1990). The anterior cingulated cortex mediates processing selection in the Stroop attentional conflict paradigm. *Proceedings of the National Academy of Science, 87*, 256–259.

Peretti, P. (1971). Cross sex and cross educational level performance in a color word interference task. *Psychonomic Science, 16*, 321–323.

Rosselli, M., Ardila, A., Santisi, M. N., Del Rosario Arecco, M., Salvatierra, J., Conde, A., & Lenis, B. (2002). Stroop effect in Spanish-English bilinguals. *Journal of the International Neuropsychological Society, 8*(6), 819–827.

Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). NY: Oxford University Press.

Stroop Effect

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Synonyms

Cognitive flexibility; Interference

Description

Various forms of the Stroop test have been developed. These differ in terms of (a) number of colors used, (b) type of stimuli used to present the color patches on Page 2, (c) presentation of items across columns or down rows, and (d) method of scoring. In general, simpler versions of the test work as well as more complex versions. Scoring may reflect the number of items completed in a given period of time or the time to complete a set number of items. These approaches appear to generate very similar results.

Most versions of the Stroop test consist of three pages. For example, in the first generalized standardized version introduced by Golden (1975), Page 1 consisted of the words "RED," "GREEN," and "BLUE" arranged randomly and printed in black ink on a white 8.5″ × 11″ sheet of paper. No word was allowed to follow itself within a column. Page 2 consisted of 100 items, all written as XXXX, printed in either red, green, or blue ink. No color was allowed to follow itself in a column nor to match the corresponding item on Page 1. Page 3 consisted of the words on Page 1 printed in the colors on Page 2. The two pages were blended item for item: Item I on Page 1 is printed in the color in Item I on Page 2 to produce Item I on Page 3. In no case does the word and the color it is printed in match one another.

While the basic scores for the three pages are similar across versions of the test, there is much more variability for the interference score which seeks to measure the degree of the actual Stroop effect. This is a derived score that compares the performance on the color–word page to the performance on the basic color-naming and word-reading pages. Many of these scores were formed in an attempt to get a pure interference score that was not dependent on the subjects reading or color-naming speed. These scores range from simply subtracting the color score from the color–word score to formulas that subtract a predicted color–word score (based on color naming and word reading scores) from the actual color–word score. Raw scores are then generally converted to a standardized score based on normative data for a specific version of the test. Stroop scores are generally reliable across even short test–retest periods, although repeated practice will generate marginally better scores.

Historical Background

The Stroop color and word test developed from the observation by early experimental psychologists that the naming of color hues is always slower than the reading of color names in literate adults. The earliest published report of this phenomenon was offered by Cattell (1886) (cited in Golden & Freshwater, 2002). Cattell found that words could be read and identified in 1/4 of a second, while it took twice as long to recognize and identify a simple color hue. Brown (1915) found that even with intensive practice color naming was never as fast as word reading.

Stroop (1935) suggested that the difference in color naming and word reading was due to colors being associated with a variety of behavioral responses while words were associated with only one behavioral response: reading. In order to further study the relationship between color naming and word reading, Stroop devised the test which has come to be called the Stroop color and word test. The Stroop test and the Stroop effect have been used to assess for changes reflecting variables such as drive levels, stress, neuroticism, personality, brain damage, creativity, automaticity, cognitive control, and depression.

The earliest version of the Stroop consisted of the words red, green, brown, blue, and purple printed on a page consisting of ten rows and ten columns. Each word was printed in colored ink, but never in the color hue represented by the word (e.g., RED could be printed in blue ink, but never in red ink). Another page of the test consisted of colored ink printed as small rectangles (which were replaced in later versions by swastikas). The final page consisted of color words listed above printed in black ink.

Since Stroop's original studies, several hundred studies have been published on the Stroop test. The Stroop has attracted such attention because of its high reliability in identifying individual differences (and because of its somewhat paradoxical nature. The research has examined the use of the Stroop in cognitive and personality research, in experimental psychopathology, and in the

diagnosis and understanding of organic brain dysfunction. Because of the interest in the Stroop interference effects, numerous attempts have been made to develop other tests measuring the same dimension but none has been as effective or successful as the original version.

Neuropsychological studies have suggested that Stroop interference occurs not at the response stage or in the confusion of the subject, but as a result of interference in verbal processing (Wheeler, 1977). The Stroop stimuli appear to activate an automatic verbal processing response which interferes with the consciously instructed color naming. The subject completes the task either by completing both responses sequentially (reading the word followed by naming the color) or by suppressing the automatic, word reading response through volitional control (Golden, 1976).

Stuss, Floden, Alexander, Levine, and Katz (2001) concluded that Stroop interference was most related to the superior medial frontal area of the brain. Demakis (2004) concluded that the Stroop was a good measure of frontal versus posterior functions, while Alvarez and Emory (2006) concluded that interpretation of the Stroop as a test of frontal functioning was not consistently supported by neuroimaging tests, possibly because of the sensitivity of the Stroop to only certain types of frontal injuries. It is likely that the inconsistencies in the literature are related to the impact of other brain areas and functions and the complexity of interpretation when either color naming or word reading is impaired, as discussed in the clinical use section below.

Psychometric Data

There is extensive psychometric data on the Stroop, but the data varies depending on the form of the test employed. For example, Golden and Freshwater (2002) includes the psychometric properties for Golden's three-color, 45 second version of the test. Although versions differ in a variety of ways as described above, the Stroop effect seems robust across each of the versions which rely on the conflict between color naming and word reading.

Clinical Uses

Overall, the Stroop interference page tests the ability of the individual to separate the word and color naming stimuli. If this can be done, the individual can suppress the reading response and proceed with color naming. In other people, the suppression of the word naming fails to occur and the individual must process both the word and color before responding. In a final group, the word and color response are inextricably confounded, given rise to high levels of interference. This analysis applies only when the word naming response is faster than the color reading response and is automatic. In children and others with weaker and less automatic reading skills, the test appears to measure the relative dominance of the verbal/reading system. Thus, in the adult with normal reading skills, the Stroop stimuli involve, at a basic level, the ability of an individual to sort information from his or her environment and to selectively react to this information. The tapping of this basic skill enables the Stroop test to be useful in investigating a wide range of basic psychological processes in normal and dysfunctional people.

While the concept of the Stroop interference effect is relatively simple, the detection and interpretation of the effect is more difficult. The primary problem is that the effect itself is dependent on having two strongly automatized skills (word reading and color naming) where the word reading skill is significantly more automatized than the color naming skill. There are many situations in which this condition is not met. If a subject has weak reading skills, then this condition will not be met. This occurs frequently in younger children, individuals with poor or no education, individuals with certain learning disabilities, individuals with poor near vision, and individuals taking the test in their secondary rather than primary language. In such cases, reading is not as highly automatized and the automatized superiority over color naming is not present. Similarly, individuals unable to recognize colors easily and quickly due to language, color blindness, or visual problems may have insufficiently automatized color naming skills. Individuals with brain dysfunction may also have lessened skills in both these areas (especially with posterior brain damage or dysfunction). Motivational and psychiatric issues may also interfere with the test effect. Individuals with anxiety or depression may not read or name colors at their highest rate, thus lessening the automaticity of the tasks. Poorly motivated people may perform slowly, as may overcautious individuals. Stroop interpretation and research can be compromised if the individual or population studied includes individuals whose interference effects are compromised by any of these variables.

Markedly lower scores on the third color–word page when compared to performances on the color and word pages are indicative of interference. Such scores indicate the possible presence of either prefrontal pathology (in

which case the dominant word naming cannot be inhibited) or emotional turmoil (in clients who are hysterical or acutely agitated). Normal interference scores in the presence of good word and color scores suggest cognitive flexibility and the ability to respond to task demands. While the Stroop interference effect has been used as a brain injury screening test, normal Stroop scores do not rule out a brain injury, they only reflect the lower probability of such an injury.

Clinically, it is also important to observe the way in which the patient performs the Stroop as well as the scores themselves. For example, if the patient has trouble keeping to the correct column despite attempts to do so, this can suggest a visual-spatial disorder. Perseveration or slurring of speech can indicate a left hemisphere injury. Low scores due to an inability to withstand frustration, as demonstrated by the refusal to go on with the task or temper tantrums, is usually indicative of a poor frustration tolerance.

Cross References

► Executive Functioning
► Frontal Lobes

References and Readings

Alvarez, J., & Emory, E. (2006). Executive function and the frontal lobes: A meta-analytic review. *Brain and Cognition, 45*, 21–43.

Brown, W. (1915) Practice in associating color-names with colors. *Psychological Review, 22*, 45–55.

Cattell, J. M. (1886). The time it takes to see and name objects. *Mind, 11*, 63–65.

Demakis, G. J. (2004). Frontal lobe damage and tests of executive processing: a meta-analysis of the category test, stroop test, and trail-making test. *Journal of Clinical and Experimental Neuropsychology, 26*(3), 441–450.

Golden, C. J. (1975). A group version of the Stroop Color and Word Test. *Journal of Personality Assessment, 39*(4), 386–388.

Golden, C. J. (1976). The diagnosis of brain damage by the Stroop test. *Journal of Clinical Psychology, 32*, 654–658.

Golden, C., & Freshwater, S. (2002). *A manual for the adult Stroop Color and Word Test.* Chicago, IL: Stoelting.

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*, 643–662.

Stuss, D. T., Floden, D., Alexander, M. P., Levine, B., & Katz, D. (2001). The role of pre-frontal regions in the Stroop task. *Neuropsychologia, 33*, 341–352.

Wheeler, D. D. (1977). Locus of interference on the Stroop test. *Perceptual and Motor Skills, 45*, 263–266.

Stroop Neuropsychological Screening Test (adult)

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Synonyms

SNST

Definition

The SNST (Trenerry, Crosson, DeBoe, & Leber, 1989) requires the individual to inhibit a response that is more readily available (e.g., reading the word) for one that is less readily accessible (e.g., stating the color of the ink that the word is printed in). The SNST was developed to provide a standardized method of administration and scoring. As with other versions of the Stroop procedure (Golden, 1978), the SNST is designed to assess the individual's ability to selectively process only one visual feature at a time while inhibiting the processing of other features, which makes it a test of "concentration effectiveness" (Lezak, 2004).

Current Knowledge

As with the Golden version of the Stroop, the SNST requires the individual to read the stimuli down columns rather than across the rows, reducing the likelihood that the person will lose his/her place. It also uses colors that contain only one syllable (e.g., red) as multisyllabic words (e.g., orange) require more time to read.

The SNST consists of two stimulus sheets: Form C and Form C-W. The Form C stimulus sheet consists of 112 color names (i.e., red, green, blue, & tan) arranged in four columns of 28 names. The color names are printed in different color ink, but not in the matching color (e.g., tan may be printed in red, but not tan). Form C-W is the same as Form C except for the order of the color names.

The test has been validated for individuals 18 and older. Although only Form C-W is scored, both

stimulus forms are administered as the normative data is based on administration of both forms. On Form C, the individual is instructed to read the words aloud as quickly and accurately as he or she can. On the color-word task, the individual is instructed to name the color of the ink in which the word is printed. 120 s are allowed for each stimulus sheet for a maximum test time of 4 min.

Two scores are obtained: (1) Color Score = the number of correct responses completed minus incorrect responses on the Color Task; and (2) Color-Word Score = the number of correct responses completed minus incorrect responses on the Color-Word Task. Percentile and probability values are presented in the administration manual for two age ranges: 18–49 and 50 +. The normative sample consists of 156 adults ranging in age from 18 to 79 who were screened to rule out histories of neurological or psychiatric illness, or physical handicaps that might affect performance. Discriminant analyses were used to develop cut-off scores. A score of 99 was determined to have the highest hit rate for discriminating brain-damaged individuals from the normative sample for the 18–49 age group. For the 50+ group a score of 62 produced the highest hit rate.

Reliability and validity data are available in the administration manual (Trenerry et al., 1989). Research using the SNST (Young, Braham, Tyson, & Morris, 2006) has added to the validity of the measure, showing that adults with ADHD performed at a slower rate and were less accurate on the Color, Color-Word, and Interference conditions.

Cross References

- ▶ Attention-Deficit/Hyperactivity Disorder (ADHD)
- ▶ Frontal Lobes
- ▶ Stroop Color and Word Test

References and Readings

Golden, C. J. (1978). A manual for the stroop color and word test. Chicago, IL: Stoetling Company.

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

Trenerry, M. R., Crosson, B., DeBoe, J., & Leber, W. R. (1989). *Stroop neuropsychological screening test manual.* Lutz, FL: Psychological Assessment Resources.

Young, S., Bramham, J., Tyson, C., & Morris, R. (2006). Inhibitory dysfunction on the stroop in adults diagnosed with attention deficit hyperactivity disorder. *Personality & Individual Differences, 41*, 1377–1384.

Structural Equation Modeling

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Definition

Structural equation modeling (SEM) is similar to multiple regression in that it allows the researcher to investigate relations among groups of observations with influences from multiple sources. However, SEM is a more general and powerful technique that allows for and evaluates nonlinearity, latent dependent measures, correlated error terms, and interactions among independent variables.

Current Knowledge

SEM also allows evaluations of models rather than just evaluation of individual coefficients. SEM has been suggested as an alternative to multiple regression, factor analysis, path analysis, time series analysis, and ANCOVA.

Cross References

- ▶ Factor Analysis
- ▶ Path Analysis

References and Readings

Ullman, J. B., & Bentler, P. M. (2004). *Handbook of data analysis.* Newbury Park: Sage.

Structured Clinical Interview

- ▶ Structured Clinical Interview For DSM-IV (SCID-I/SCID-II)

Structured Clinical Interview For DSM-IV (SCID-I/SCID-II)

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Synonyms

Interview schedules; Psychodiagnostic interview; Semi-structured clinical interview; Structured clinical interview

Description

The Structured Clinical Interview for DSM-IV (SCID-I/SCID-II; First, Gibbon, Spitzer, Williams, & Benjamin, 1997) is a semi-structured clinical interview administered by trained clinicians and designed to yield psychiatric diagnoses consistent with DSM-IV/DSM-IV-TR (American Psychiatric Association, 2000) diagnostic criteria. The duration of administration ranges between 15 min and 2 h. The SCID is designed to begin with open-ended questions that introduce each content area (e.g., "Have you ever had. . .?"), followed by a series of scripted questions to be asked verbatim. At the close of each module, the SCID directs interviewers to append as many additional questions as needed in order to be confident about the validity of their ratings. Interviewers are also encouraged to corroborate their assumptions with collateral data whenever possible. The score summary sheets document any SCID Axis I and/or Axis II diagnoses; additional interviewer diagnoses; an indication of psychosocial and environmental problems (Axis IV); and a rating for the Global Assessment of Functioning (GAF) Scale, Axis V.

Though originally intended to be delivered face to face, research has suggested that computer-assisted and telephone administration of the SCID generate comparable diagnoses (Cacciola, Alterman, Rutherford, McKay, & May, 1999). Contrary to its name, the format is considered "semi-structured" in that collateral data review and clinical judgment, in addition to the answers to the scripted questions, are required to determine whether diagnostic criteria have been met. The structure of the interview itself ensures full coverage of the diagnostic possibilities and reduces the opportunity for error in clinical judgment or the introduction of social/cultural biases (Torres, Zayas, Cabassa, & Pérez, 2007). Table 1 indicates the symptoms, episodes, and disorders that are assessed by the SCID modules.

Historical Background

Work on the original SCID began just after the 1980 publication of the DSM-III. Until that time, there were several diagnostic paradigms available to mental health professionals, each with attendant liabilities. Many models were inconsistent with the prevailing medical diagnostic system, the International Statistical Classification of Diseases and Related Health Problems (ICD; World Health Organization), and most lacked universal endorsement and psychometric standardization (Mayes & Horowitz, 2005). To address these shortcomings, Robert Spitzer chaired the development of a new categorical model of mental illness, which was based on the assumption that specific patterns of symptoms reflect a specific disorder or category. Each category represents a prototype; persons whose symptoms approximate that prototype are diagnosed as having that disorder. The publication of the DSM-III, revised from the 1968 DSM-II to reflect this new categorical classification system, heralded the widespread adoption of this new paradigm.

Shortly after the DSM-III publication, work began to standardize an assessment protocol of this new model of psychopathology. For that purpose, Spitzer and colleagues (Spitzer, Williams, Gibbon, & First, 1992) designed the original Structured Clinical Interview for the DSM-III/DSM-III-R (SCID) for use in research and clinical settings. The SCID was revised in 1996 to reflect changes in the DSM-IV, and revised again in 2001 to parallel the DSM-IV-TR. Presently, the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) generates major DSM-IV Axis I diagnoses, while the SCID-II is used for making DSM-IV Axis II, or personality disorder, diagnoses.

Among the current versions, the Research Version for Axis I Disorders (SCID-I-RV; First, Spitzer, Gibbon, & Williams, 2002) is designed to generate exhaustive and diagnostically useful information in research settings, and a Clinician Version (SCID-CV; First, Spitzer, Gibbon, & Williams, 2002) is abbreviated to reflect only those diagnoses commonly seen in clinical settings. There are two editions of the Research Version: the SCID Patient Edition (SCID-I/P) to be used with psychiatric patients and the SCID Non-Patient Edition (SCID-I/NP) designed and validated for use with community and family samples (Shear et al., 2000). Unlike the abbreviated SCID-CV, the research version is adaptable to clinical hypothesis testing, and users are directed to customize the assessment as needed.

There are several other contemporary versions of the SCID that were designed to reflect growing diversity in practical settings and populations. The SCID-CV and SCID-II can be administered to a client by a clinician via computer using the "Computer-Assisted SCID-CV" (CAS-CV) and (CAS-II) (Biometrics Research Department, 2007). There is an official Spanish language translation (Biometrics Research Department, 2007), in addition to available versions in Danish, Dutch, French, German, Greek, Hebrew, Italian, Mandarin, Portuguese, Romanian, Swedish, Turkish, and Zulu. There is also a SCID for use with pediatric populations (KID-SCID; Matzner, Silva, Silvan, Chowdhury, & Nastasi, 1997) that specifically assesses attention-deficit/hyperactivity disorder (ADHD), conduct disorder, and oppositional defiant disorder. Finally, there is some research on an additional SCID module assessing impulse control disorders (i.e., intermittent explosive disorder, pyromania, kleptomania, pathological gambling, trichotillomania, Internet addiction, skin picking, pathological shopping, and compulsive sexual behavior) (Biometrics Research Department, 2007).

Psychometric Data

Reliability

Hundreds of studies have documented the utility of the SCID in clinical practice, and dozens more have studied its reliability. The reliability of categorical ratings, such as

Structured Clinical Interview For DSM-IV (SCID-I/SCID-II). Table 1 Diagnostic coverage of SCID modules

Module A: *Mood episodes*	Major depressive episode (current/past)
	Manic episode (current/past)
	Hypomanic episode (current/past)
	Dysthymic disorder (current only)
	Mood disorder due to a general medical condition
	Substance-induced mood disorder
Module B: *Psychotic symptoms*	Delusions
	Hallucinations
	Disorganized speech and behavior
	Catatonic behavior
	Negative symptoms
Module C: *Psychotic disorders*	Schizophrenia
	Paranoid type
	Catatonic type
	Disorganized type
	Undifferentiated type
	Residual type
	Schizophreniform disorder
	Schizoaffective disorder
	Delusional disorder
	Brief psychotic disorder
	Psychotic disorder due to a general medical condition
	Substance-induced psychotic disorder
	Psychotic disorder not otherwise specified
Module D: *Mood disorders*	Bipolar I disorder
	Bipolar II disorder
	Other bipolar disorders (cyclothymic disorder, bipolar disorder NOS)
	Major depressive disorder
	Depressive disorder not otherwise specified

Structured Clinical Interview For DSM-IV (SCID-I/SCID-II). Table 1 (Continued)

Module E: *Substance use disorders*	Alcohol dependence
	Alcohol abuse
	Amphetamine dependence
	Amphetamine abuse
	Cannabis dependence
	Cannabis abuse
	Cocaine dependence
	Cocaine abuse
	Hallucinogen dependence
	Hallucinogen abuse
	Opioid dependence
	Opioid abuse
	Phencyclidine dependence
	Phencyclidine abuse
	Sedative/hypnotic/anxiolytic dependence
	Sedative/hypnotic/anxiolytic abuse
	Polysubstance dependence
	Other or unknown substance dependence
	Other or unknown substance abuse
Module F: *Anxiety disorders*	Panic disorder with agoraphobia
	Panic disorder without agoraphobia
	Agoraphobia without history of panic disorder
	Social phobia
	Specific phobia
	Obsessive–compulsive disorder
	Post-traumatic stress disorder
	Generalized anxiety disorder (current only)
	Anxiety disorder due to a general medical condition
	Substance-induced anxiety disorder
	Anxiety disorder not otherwise specified
Module G: *Somatoform disorders*	Somatization disorder (current only)
	Undifferentiated somatoform disorder (current only)
	Pain disorder (current only)
	Hypochondriasis (current only)
	Body dysmorphic disorder (current only)
Module H: *Eating disorders*	Anorexia nervosa
	Bulimia nervosa
	Binge eating disorder (appendix category)
Module I: *Adjustment disorder*	Adjustment disorder (current only)
Module J: *Optional module*	Acute stress disorder
	Minor depressive disorder (appendix category)
	Mixed anxiety depressive disorder (appendix category)
	Symptomatic details of past major depressive/manic episodes

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those generated by the SCID (e.g., whether a diagnosis is present or not) is often reported as kappa values, or the degree to which the tool is used consistently across settings and clinicians. Kappa values above 0.70 reflect good agreement between raters, values from 0.50 to 0.70 represent fair agreement, and values below 0.50 represent poor agreement overall. Tables 2 and 3 below highlight just a few of the studies of inter-rater agreement on the SCID-I and SCID-II.

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Research suggests that the reliability of the SCID is dependent on the rigorous interviewer-training requirement for users (Ventura, Liberman, Green, Shaner, & Mintz, 1998). When this requirement has been met, discrepancies between raters (reflected in lower kappa values) can be attributed to a number of factors that are unique to psychological variables and independent of the SCID itself. The first is the consistency of the patient's narrative report. In many studies, the same patient is evaluated by multiple examiners in succession over time, leaving open the possibility that the conversation about symptoms was qualitatively different at each meeting. Also, mental illness is present in only a small percentage of the population which, by virtue of the low base rate, can preclude an accurate appraisal of the tools used for its study. Finally, the disagreement between raters may reflect differences of opinion about severity and not diagnosis. Given DSM-IV diagnostic requirement that patients endorse several criteria (e.g., "five of seven") in order for a diagnosis to be applied, there is some likelihood that raters may have differed only on the criteria and not on the category. Substantiating this possibility is a study (Maffei et al., 1997) that assessed both categorical and dimensional inter-rater agreement in the administration of the SCID-II to a group of 231 psychiatric patients. The data were reviewed blindly and the raters paired randomly for analysis. Internal consistency coefficients ranged from 0.71 to 0.94. Inter-rater reliability coefficients ranged from 0.48 to .98 for categorical diagnosis. Notably,

Structured Clinical Interview For DSM-IV (SCID-I/SCID-II). Table 2 Selected SCID-I reliability studies (kappa values)

Reference	Skre et al. (1991)	Zanarini et al. (2000)	Zanarini et al. (2000)	Segal et al. (1995)	Wiliams et al. (1992)	Zanarini et al. (2001)	Zanarini et al. (2001)
Population studied	*N* = 54	*N* = 27	*N* = 52	*N* = 40	*N* = 592; Inpt, Outpt, Non-Pt.	*N* = 45	*N* = 30
Version of SCID	DSM-IIIR	DSM-IV	DSM-IV	DSM-IIIR	DSM-IIIR	DSM-IIIR	DSM-IIIR
Design of reliability study	Joint; audiotape	Joint; 84 pairs from four sites	7–10 day interval, test–retest	Joint; audiotape	1–3 week interval, test–retest	Joint; observed live	7–10 day interval, test–retest
Major depressive disorder	0.93	0.80	0.61	0.90	0.64	0.90	0.73
Dysthymic disorder	0.88	0.76	0.35	0.53	0.40	0.91	0.60
Bipolar disorder	0.79				0.84		
Schizophrenia	0.94				0.65		
Alcohol dependence/abuse	0.96	1.0	0.77		0.75	1.0	
Other substance dependence/abuse	0.85	1.0	0.76		0.84	0.95	0.77
Panic disorder	0.88	0.65	0.65	0.80	0.58	0.88	0.82
Social phobia	0.72	0.63	0.59		0.47	0.86	0.53
OCD	0.40	0.57	0.60		0.59	0.70	0.42
GAD	0.95	0.63	0.44		0.56	0.73	0.63
PTSD	0.77	0.88	0.78			1.0	1.0
Any somatoform disorder	-0.03			0.84			
Any eating disorder		0.77	0.64				

Structured Clinical Interview For DSM-IV (SCID-I/SCID-II). Table 3 Selected SCID-II reliability studies

Study	First et al. (1995)	Weiss et al. (1995)	Arntz et al. (1992)	Fogelson (1991)	Dreessen et al. (1998)	Maffei et al. (1997)
n	284	31	70	15	43	231
Method	1–3 week interval, test–retest	12-month interval, test–retest	Joint with live observer	Joint with audiotape	1–4 week interval, test–retest	Joint with live observer
Statistic	Kappa	Kappa	Kappa	ICC	ICC	Kappa
Version	DSM-III-R	DSM-III-R	DSM-III-R	DSM-III-R	DSM-III-R	DSM-IV
Avoidant	0.54	-0.15	0.82	0.84	0.80	0.97
Dependent	0.50	0.43	1	[c]	0.49	0.86
Obsessive–Compulsive	0.24	0.26	0.72	[c]	0.75	0.83
Passive–aggressive	0.47	0.71	0.66	[c]	0.62	0.91
Self-Defeating	0.33	[c]	1	[c]	0.53	[b]
Depressive	[a]	[a]	[a]	[a]	[a]	0.65
Paranoid	0.57	0.47	0.77	0.70	0.66	0.93
Schizotypal	0.54	0.78	0.65	0.73	0.59	0.91
Schizoid	[c]	[c]	[c]	0.60	[c]	0.91
Histrionic	0.62	0.59	0.85	[c]	0.24	0.92
Narcissistic	0.42	0.59	1	[c]	[c]	0.98
Borderline	0.48	0.02	0.79	0.82	0.72	0.91
Antisocial	0.76	0.41	[c]	[c]	0.75	0.95

[a]Not included in SCID-II for DSM-III-R
[b]Not included in SCID-II for DSM-IV
[c]Not reported because too few cases

though, inter-rater reliability on dimensional judgments ranged from 0.90 to 0.98.

Validity

In an effort to establish the validity of the SCID, Spitzer proposed a method he termed the "LEAD" standard. "This standard involves conducting a longitudinal assessment (L) (i.e., relying in data collected over time), done by expert diagnosticians (E), using all data (AD) that are available about the subjects, such as family informants, review of medical records, and observations of clinical staff" (from http://scid4.org/psychometric/scidI_ validity.html). To date, several studies (Basco et al., 2000; Fenning et al., 1994; Fenning et al., 1996; Kranzler et al., 1995; Kranzler et al., 1996) have adopted the LEAD standard and have demonstrated the SCID-I to be superior to standard clinical intake interviews. Furthermore, an assessment of the SCID-II using a LEAD standard found the diagnostic rigor (the ratio of true test results to total number of tests administered) to vary from 0.45 for narcissistic personality disorder to 0.95 for antisocial personality disorder. Overall, the diagnostic power of the SCID-II was 0.85 or greater for five personality disorders.

Several studies have assessed individual components of the SCID and their relations to other, similar measures in order to establish concurrent and convergent validity. Studies have demonstrated strong associations between diagnoses made using the SCID and those generated by the Millon Clinical Multiaxial Inventory (MCMI; Renneberg, Chambless, Dowdall, Fauerbach, & Gracely, 1992), the Personality Disorder Examination (PDE; O'Boyle & Self, 1990; Oldham, Skodol, Kellman, Hyler, & Rosnick, 1992), clinical interviews (Steiner et al. 1995), and self-report measures over time (Modestin, Erni, & Oberson, 1998; Skodol, Rosnick, Kellman, Oldham, & Hyler, 1988). One project investigated the relationship between the single item query about trauma on the SCID to the Stressful Life Events Screening Questionnaire (Elhai, Franklin, & Gray, 2008). Among a population of medical patients with a trauma history, the

SCID query correctly identified 76% of them, and in a population of college students with trauma histories, it correctly identified 66% of them (sensitivity). The SCID correctly identified 67% of non-traumatized medical patients and 87% of non-traumatized college students (specificity). These results suggest that the SCID trauma query is an effective screening tool for trauma. As evidence of the proven utility of the SCID, in a validation study for the substance abuse disorder questions of the National Survey on Drug Use and Health (NSDUH; Jordan, Karg, Batts, Epstein, & Wiesen, 2008), the SCID was used as the gold standard assessment tool.

Clinical Uses

There are three conventional uses for the SCID. In research settings, the SCID is often used as the "gold standard" for the assessment of DSM-IV Axis I (clinical disorders) and Axis II (personality disorders) diagnoses. There, it can be used to justify the inclusion of research subjects in a particular design or to rule out or exclude those subjects with an unwanted diagnosis. The SCID may also assist in defining the makeup of a study sample (past and present psychiatric diagnoses). In a clinical setting, the SCID can generate categorical diagnostic information (e.g., whether a disorder is present or not) or dimensional information (e.g., how severe the symptoms are). It can also be used for clinical hypothesis testing or to confirm a suspected diagnosis. Notably, the SCID can be used by student clinicians to hone interview techniques and increase familiarity with the DSM-IV.

The SCID is used to identify DSM-IV disorders among "medical patients, family members, community samples, college students, the homeless, the elderly, and in short, any English-speaking adult who is able to participate in the interview" (Biometrics Research Department, 2007). In research paradigms, as the SCID has served as the "gold standard" by which group membership is assigned and comparisons between diagnostic groups made possible. To date, that body of research includes the study of obsessive–compulsive disorder (Mataix-Cols, 1999), major depression (Hill, Keshavan, Thase, & Sweeney, 2004), toxic environmental exposures (Morrow, Stein, Bagovich, Condray, & Scott, 2001; White et al., 2001), ADHD (Seidman et al., 1997), kleptomania (Grant, Odlaug, & Wozniak, 2007), antisocial personality disorder (Dolan & Park, 2002), and post-traumatic stress disorder (Samuelson et al., 2008).

The SCID also has the utility as a diagnostic tool in research with specific neuropsychological populations. The SCID has been used to investigate the psychopathological consequences of injury and has been especially useful in the study of traumatic brain injury (TBI) (e.g., Asmundson et al., 1999; Kennedy et al., 2005; Rapoport et al., 2005; Whelan-Goodinson et al., 2009). In a study of the emotional sequelae of TBI, the SCID was used to diagnose personality change (Rao, Spiro, Schretlen, & Cascella, 2007); it has also been used to identify the psychological correlates of aggressive behavior post-TBI (Rao et al., 2009). Used in a study on the nature and frequency of psychiatric disorders pre- and post-TBI (Whelan-Goodinson et al., 2009), a high incidence of post-TBI psychopathology was documented, including major depression, generalized anxiety disorder, post-traumatic stress disorder, panic disorder, and phobias. As this study demonstrates, SCID data can offer researchers important information about the comorbidity of psychiatric conditions and neurological disorders, in addition to offering providers insight into the subjective experience of affected individuals.

Cross References

- ▶ Anxiety
- ▶ Clinical Interview
- ▶ Depressive Disorder
- ▶ Differential Diagnosis
- ▶ Millon Clinical Multiaxial Inventory (MCMI)
- ▶ Multiaxial Assessment
- ▶ Schizotypal Personality Disorder
- ▶ Substance Abuse Disorders
- ▶ Test Reliability and Validity

References and Readings

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders-IV-TR*. Washington DC: American Psychiatric Association.

Arntz, A., van Beijsterveldt, B., Hoekstra, R., Hofman, A., Eussen, M., & Sallaerts, S. (1992). The interrater reliability of a Dutch version of the structured clinical interview for DSM-III-R personality disorders. *Acta Psychiatrica Scandinavica, 85*(5), 394–400.

Asmundson, G. J., Cox, B. J., Larsen, D. K., Frombach, I. K., & Norton, G. R. (1999). Psychometric properties of the Accident Fear Questionnaire: An analysis based on motor vehicle accident survivors in a rehabilitation setting. *Rehabilitation Psychology, 44*(4), 373–387.

Basco, M. R., Bostic, J. Q., Davies, D., et al. (2000). Methods to improve diagnostic accuracy in a community mental health setting. *American Journal of Psychiatry, 157*, 1599–1605.

Biometrics Research Department, New York State Psychiatric Institute (2010). Structured Clinical Interview for DSM Disorders

Official Website. Available at www.SCID4.org. Accessed on 27 January 2010.

Cacciola, J. S., Alterman, A. I., Rutherford, M. J., McKay, J. R., & May, D. J. (1999). Comparability of telephone and in-person Structured Clinical Interview for DSM-III-R (SCID) diagnoses. *Assessment, 6*(3), 235–242.

Dolan, M., & Park, I. (2002). The neuropsychology of antisocial personality disorder. *Psychological Medicine, 32*, 417–427.

Dreessen, L., & Arntz, A. (1998). Short-interval test-retest interrater reliability of the Structured Clinical Interview for DSM-III-R personality disorders (SCID-II) in outpatients. *Journal of Personality Disorders, 12*(2), 138–148.

Elhai, J. D., Franklin, C. L., & Gray, M. J. (2008). The SCID PTSD module's trauma screen: Validity with two samples in detecting trauma history. *Depression and Anxiety, 25*, 737–741.

Fennig, S., Craig, T., Lavelle, J., Kovasznay, B., & Bromet, E. J. (1994). Best-estimate versus structured interview-based diagnosis in first-admission psychosis. *Comprehensive Psychiatry, 35*(5), 341–348.

Fennig, S., Naisberg-Fennig, S., Craig, T. J., et al. (1996). Comparison of clinical and research diagnoses of substance use disorders in a first-admission psychotic sample. *American Journal of Addiction, 5*(1), 40–48.

First, M. B., & Gibbon, M. (2004). The structured clinical interview for DSM-IV axis I disorders (SCID-I) and the structured clinical interview for DSM-IV axis II disorders (SCID-II). In M. Hilsenroth, & D. L. Segalaniel (Eds.), *Comprehensive handbook of psychological assessment: Vol. 2. Personality assessment* (pp. 134–143). Hoboken, NJ: Wiley.

First, M. B., Gibbon, M., Spitzer, R. L. Williams, J. B. W., & Benjamin, L. S. (1997). *Structured clinical interview for DSM-IV axis II personality disorders, (SCID-II).* Washington, DC: American Psychiatric Association.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured clinical interview for DSM-IV-TR axis I disorders, research version, patient edition.* (SCID-I/P) New York: Biometrics Research, New York State Psychiatric Institute.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured clinical interview for DSM-IV-TR axis I disorders, research version, non-patient edition.* (SCID-I/NP) New York: Biometrics Research, New York State Psychiatric Institute.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured clinical interview for DSM-IV axis I disorders, clinician version (SCID-CV).* New York: Biometrics Research, New York State Psychiatric Institute.

First, M. B., Spitzer, R. L., Gibbon, M., Williams, J. B. W., Davies, M., Borus, J., et al. (1995). The structured clinical interview for DSM-III-R personality disorders (SCID-II). Part II: Multi-site test-retest reliability study. *Journal of Personality Disorders, 9*(2), 92–104.

Fogelson, D. L., Neuchterlein, K. H., Asarnow, R. F., Subotnik, K. L., & Talovic, S. A. (1991). Interrater reliability of the Structured Clinical Interview for DSM-III-R, Axis II: Schizophrenia spectrum and affective spectrum disorders. *Psychiatry Research, 39*, 55–63.

Grant, J. E., Odlaug, B. L., & Wozniak, J. R. (2007). Neuropsychological functioning in kleptomania. *Behaviour Research and Therapy, 45*(7), 1663–1670.

Hill, S. K., Keshavan, M. S., Thase, M. E., & Sweeney, J. A. (2004). Neuropsychological dysfunction in antipsychotic-naive first-episode unipolar psychotic depression. *American Journal of Psychiatry, 161*, 996–1003.

Jordan, K. B., Karg, R. S., Batts, K. R., Epstein, J. F., & Wiesen, C. (2008). A clinical validation of the National Survey on Drug Use and Health Assessment of Substance Abuse Disorders. *Addictive Behaviours, 33*(6), 782–798.

Kennedy, R. E., Livingston, L., Riddick, A., Marwitz, J. H., Kreutzer, J. S., Zasler, N. D., et al. (2005). Evaluation of the Neurobehavioral Functioning Inventory as a depression screening tool after traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 20*(6), 512–526.

Kranzler, H. R., Kadden, R., Burleson, J., et al. (1995). Validity of psychiatric diagnoses in patients with substance use disorders – is the interview more important than the interviewer. *Comprehensive Psychiatry, 36*(4), 278–288.

Kranzler, H. R., Kadden, R. M., Babor, T. F., et al. (1996). Validity of the SCID in substance abuse patients. *Addiction, 91*(6), 859–868.

Maffei, C., et al. (1997). Interrater reliability and internal consistency of the structured clinical interview for DSM-IV axis II personality disorders (SCID-II), version 2.0. *Journal of Personality Disorders, 11*(3), 279–284.

Mataix-Cols, D. (1999). Neuropsychological functioning in a subclinical obsessive-compulsive sample. *Biological Psychiatry, 45*(7), 898–904.

Matzner, F., Silva, R., Silvan, M., Chowdhury, M., Nastasi, L. (1997). Preliminary Test-retest Reliability of the KID-SCID. *Scientific Proceedings, American Psychiatric Association Meeting.* Available at http://cpmcnet.columbia.edu/dept/scid/info/kidscid.html. Accessed 7 May, 2010.

Mayes, R., & Horwitz, A. V. (2005). DSM-III and the revolution in the classification of mental illness. *Journal of the History of the Behavioural Sciences, 41*(3), 249–267.

Modestin, J., & Oberson, B. (1998). A comparison of self-report and interview diagnoses of DSM-III-R personality disorders. *European Journal of Personality, 12*(6), 445–455.

Morrow, L. A., Stein, L., Bagovich, G. R., Condray, R., & Scott, A. (2001). Neuropsychological assessment, depression, and past exposure to organic solvents. *Applied Neuropsychology, 8*(2), 65–73.

O'Boyle, M., & Self, D. (1990). A comparison of two interviews for DSM-III-R personality disorders. *Psychiatry Research, 32*, 85–92.

Oldham, J. M., Skodol, A. E., Kellman, H. D., Hyler, S. E., & Rosnick, L. (1992). Diagnosis of DSM-III-R personality disorders by two structured interviews: Patterns of comorbidity. *American Journal of Psychiatry, 149*(2), 213–220.

Rao, V., Spiro, J. R., Schretlen, D. J., & Cascella, N. G. (2007). Apathy syndrome after traumatic brain injury compared with deficits in schizophrenia. *Psychosomatics, 48*, 217–222.

Rao, V., Rosenberg, P., Bertrand, M., Salehinia, S., Spiro, J., Vaishnavi, S., et al. (2009). Aggression after traumatic brain injury: Prevalence and correlates. *Journal of Neuropsychiatry and Clinical Neuroscience, 21*, 420–429.

Rapoport, M. J., McCullagh, S., Shammi, P., Feinstein, A. (2005). Cognitive impairment associated with Major Depression following mild and moderate traumatic brain injury. *The Journal of Neuropsychiatry and Clinical Neurosciences, 17*(1), 61–65.

Renneberg, B., Chambless, D. L., Dowdall, D. J., Fauerbach, J. A., & Gracely, E. J. (1992). A Structured Interview for DSM-III-R, Axis II, and the Millon Clinical Multiaxial Inventory: A concurrent validity study of personality disorders among anxious outpatients. *Journal of Personality Disorders, 6*(2), 117–124.

Samuelson, K. W., Metzler, T. J., Rothlind, T., Choucroun, G., Neylan, T. C., Lenoci, M., et al. (2008). Neuropsychological functioning in Posttraumatic Stress Disorder and alcohol abuse. *Neuropsychology, 20*(6), 716–726.

Segal, D. L., Kabacoff, R. I., Hersen, M., Van Hasselt, V. B., & Ryan, C. F. (1995). Update on the reliability of diagnosis in older psychiatric

outpatients using the Structured Clinical Interview for DSM-III-R. *Journal of Clinical Geropsychology, 1*, 313–321.
Segal, D. L., Corcoran, J., & Coughlin, A. (2002). Diagnosis, differential diagnosis, and the SCID. In M. Hersen & L. K. Porzelius (Eds.), *Diagnosis, conceptualization, and treatment planning for adults: A step-by-step guide* (pp. 13–34). Mahwah, NJ: Erlbaum.
Seidman, L. J., Biederman, J., Faraone, S. V., Weber, W., Mennin, D., Jones, J. (1997). A pilot study of neuropsychological function in girls with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*(3), 366–373.
Shear, M. K., Greeno, C., Kang, J., et al. (2000). Diagnosis of nonpsychotic patients in community clinics. *American Journal of Psychiatry, 157*, 581–587.
Skodol, A. E., Rosnick, L., Kellman, D., Oldham, J. M., & Hyler, S. E. (1988). Validating structured DSM-III-R personality disorder assessments with longitudinal data. *American Journal of Psychiatry, 145*, 1297–1299.
Skre, I., Onstad, S., Torgersen, S., & Kringlen, E. (1991). High interrater reliability for the Structured Clinical Interview for DSM-III-R Axis I (SCID-I). *Acta Psychiatrica Scandinavica, 84*(2), 167–173.
Spitzer, R. L., Williams, J. B., Gibbon, M., & First, M. B. (1992). The Structured Clinical Interview for DSM-III-R (SCID). I: History, rationale, and description. *Archives of General Psychiatry, 49*(8), 624–629.
Steiner, J. L., Tebes, J. K., Sledge, W. H., et al. (1995). A comparison of the structured clinical interview for DSM-III-R and clinical diagnoses. *Journal of Nervous and Mental Disease, 183*(6), 365–369.
Torres, L. R., Zayas, L. H., Cabassa, L. J., & Pérez, M. C. (2007). Diagnosing co-occurring substance-related disorders: Agreement between SCID, Hispanic clinicians and non-Hispanic clinicians. *Journal of Clinical Psychiatry, 68*(11), 1655–1662.
Ventura, J., Liberman, R. P., Green, M. F., Shaner, A., & Mintz, J. (1998). Training and quality assurance with Structured Clinical Interview for DSM-IV (SCID-I/P). *Psychiatry Research, 79*(2), 163–173.
Weiss, R. D., Najavits, L. M., Muenz, L. R., & Hufford, C. (1995). Twelve-month test-retest reliability of the Structured Clinical Interview for DSM-III-R Personality Disorders in cocaine-dependent patients. *Comprehensive Psychiatry, 36*(5), 384–389.
Whelan-Goodinson, R., Ponsford, J., Johnston, L., & Grant, F. (2009). Psychiatric disorders following traumatic brain injury: Their nature and frequency. *Journal of Head Trauma Rehabilitation, 24*(5), 324–332.
Whelan-Goodinson, R., Ponsford, J., Schonberger, M. (2009). Validity of the Hospital Anxiety and Depression Scale to assess depression and anxiety following traumatic brain injury as compared with the Structured Clinical Interview for DSM-IV. *Journal of Affective Disorders, 114*(1–3), 94–102.
White, R. F., Proctor, S. P., Heeren, T., Wolfe, J., Krengel, M., Vasterling, J., et al. (2001). Neuropsychological function in Gulf War veterans: relationships to self-reported toxicant exposures. *American Journal of Industrial Medicine, 40*(1), 42–54.
Williams, J. B. W., Gibbon, M., First, M. B., Spitzer, R. L., Davis, M., Borus, J., et al. (1992). The Structured Clinical Interview for DSM-III-R (SCID) II. Multi-site test-retest reliability. *Archives of General Psychiatry, 49*, 630–636.
Zanarini, M. C., & Frankenburg, F. R. (2001). Attainment and maintenance of reliability of axis I and axis II disorders over the course of a longitudinal study. *Comprehensive Psychiatry, 42*(5), 369–374.
Zanarini, M. C., Skodol, A. E., Bender, D., Dolan, R., Sanislow, C., Schaefer, E., et al. (2000). The Collaborative Longitudinal Personality Disorders Study: Reliability of axis I and II diagnoses. *Journal of Personality Disorders, 14*(4), 291–299.

Structured Interview of Reported Symptoms (SIRS)

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Description

The Structured Interview of Reported Symptoms (SIRS) is a structured interview developed by Rogers and his colleagues (Rogers, 1992; Rogers, Bagby, & Dickens, 1992), designed to detect malingering and other forms of feigning of mental disorders and to provide additional data about defensiveness, self-appraisal of honesty, and inconsistent responding. It was first developed in 1985 and has gone through several revisions since then. It includes 172 structured interview items including: (1) Detailed Inquiries, which address specific symptomatology and its severity; (2) Repeated Inquiries, which parallel Detailed Inquiries and test for response consistency; and (3) General Inquiries, which probe specific symptoms, general psychological problems, and symptom patterns. These items are organized into eight primary scales for the evaluation of feigning. They include: Rare Symptoms (8 items), Symptom Combinations (10 items), Improbable and Absurd Symptoms (7 items), Blatant Symptoms (15 items), Subtle Symptoms (17 items), Selectivity of Symptoms (32 items), Severity of Symptoms (32 items), and Reported versus Observed Symptoms (12 items). The SIRS strengths and weaknesses have been previously identified (Rogers, 2001). It has well-established reliability and validity and is regarded by many as the strongest structured interview measure of feigned mental disorders. It has been used in a multitude of clinical and forensic settings.

Cross References

- ▶ Effort
- ▶ Malingering
- ▶ Response Bias

References and Readings

American Academy of Clinical Neuropsychology (2009). American Academy of Clinical Neuropsychology Consensus Conference Statement on the Neuropsychological Assessment of Effort, Response Bias, and Malingering. *The Clinical Neuropsychologist, 23*, 1093–1129.

Hom, J., & Denney, R. L. (2003). *Detection of response bias in forensic neuropsychology.* Philadelphia: Haworth Press.
Rogers, R. (1992). *Structured interview of reported symptoms.* Odessa, FL: Psychological Assessment Resources.
Rogers, R. (2001). *Handbook of diagnostic and structured interviewing.* New York: Guilford Press.
Rogers, R. (2008). *Clinical assessment of malingering and deception* (3rd ed.). New York: Guilford Press.
Rogers, R., Bagby, R. M., & Dickens, S. E. (1992). *Structured interview of reported symptoms (SIRS) and professional manual.* Odessa, FL: Psychological Assessment Resources.

Stuck-In-Set

► Perseveration

Stupor

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Synonyms

Obtundation; Torpidity; Torpor

Definition

Stupor is a point along a continuum of consciousness in which the patient demonstrates profound somnolence and is arousable only with consistent and vigorous stimulation (shaking). The term is imprecise but describes the patient who is asleep or densely lethargic but can be awoken and is thus not comatose. Unlike patients in delirium, those in stupor may have grossly intact attention and awareness during periods of arousal. Stupor commonly follows states of unconsciousness (i.e., coma and vegetative state) during recovery from traumatic and nontraumatic insults.

Cross References

► Coma
► Minimally Conscious State
► Vegetative State

References and Readings

Posner, J. B., Saper, C. B., Schiff, N. D., & Plum, F. (2007). *Plum and Posner's diagnosis of stupor and coma.* New York: Oxford University Press.

Stuttering, Acquired

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Synonyms

Drug-induced stuttering; Neurogenic stuttering; Pharmacogenic stuttering; Psychogenic stuttering

Definition

Acquired stuttering is a general term referring to a type of fluency impairment that arises secondary to a specific causal event such as stroke, head trauma, neurodegenerative disease, introduction of a pharmacological agent, or significant psychoemotional stress. Behavioral symptoms are similar to those of developmental stuttering; however, individual disfluency profiles vary widely and may include behaviors not typically observed in developmental stuttering, for example, stuttering while singing.

Current Knowledge

General Characteristics

Acquired stuttering is less common than developmental stuttering. Excessive repetition of sounds or syllables (e.g., *[su- su-]summer*) is the most common symptom; however, prolonged (e.g., *ssssummer*) or "blocked" speech sounds also have been noted, as have excessive repetitions of whole words. Repetition of word-final consonants (e.g., boa[t-t-]t) – a behavior uncharacteristic of developmental stuttering – is prominent in some cases of acquired stuttering. Most case reports of acquired stuttering involve adults; however, acquired stuttering has been documented in children and adolescents also.

Neurogenic Stuttering

Neurogenic stuttering is the most commonly reported and most extensively researched type of acquired stuttering. Onset is usually sudden and coincides with nervous system injury or dysfunction. Common etiologies include stroke, traumatic brain injury, and degenerative central nervous system disease. Concomitant communication disorders, particularly aphasia and dysarthria, are often present. Reliable estimates of prevalence and incidence are difficult to obtain due to the varied and often complex etiology and the tendency for many cases to resolve over time. Available data suggest that neurogenic stuttering is relatively uncommon, but when it does occur, it is most likely to be observed in patients with either ischemic damage or traumatic brain injury. Lesion sites implicated in neurogenic stuttering are wide ranging, and include both cortical and subcortical structures. Stuttering severity may fluctuate across tasks, and stuttering may occur in contexts that are usually executed fluently by individuals with developmental stuttering (e.g., singing, choral speech, and slowed speech). Associated nonspeech behaviors (e.g., rhythmic finger tapping) occur in many cases and are similar to those seen in developmental stuttering. Patients vary widely in their awareness of and reaction to stuttering symptoms. Some individuals may exhibit speech-related anxiety, situational fears, and accompanying participation restrictions that are similar to what is seen in persons with developmental stuttering. Many others, however, exhibit less intense responses (e.g., surprise, bemusement, and annoyance) toward their speech impairment. Treatment strategies are similar to those used with developmental stuttering, and include use of regulated speech articulation (e.g., reduced or monitored articulation rate) and modification of stuttering moments by altering the timing and muscle tension of speech movements. Cognitive behavioral therapy has also been implemented with some cases. Treatment is sometimes augmented with assistive devices, for example, delayed auditory feedback, to facilitate behavioral self-regulation. Treatment research is very limited. Published case reports and clinical survey results suggest that many patients who receive therapeutic intervention realize improved fluency; however, additional research is necessary to clarify issues such as the relative effectiveness of different treatment types.

Drug-Induced Stuttering

A wide variety of drugs reportedly may induce stuttering. For example, clozapine, a dopaminergic agent, has been linked to the onset of stuttered speech in at least four case reports. There also are case reports linking the two serotonin selective reuptake inhibitors, sertraline, and fluoxetine, to stuttered speech. Disfluency symptoms are generally similar to neurogenic stuttering and typically resolve upon withdrawal of the drug.

Psychogenic Stuttering

Psychogenic stuttering refers to cases where acquired stuttering occurs (a) in the absence of discernable nervous system dysfunction or exposure to pharmacological agents known to induce stuttering, and (b) within the context of significant psychoemotional stress. Some cases may realize secondary gain from the speech disability. Disfluency patterns are generally similar to those in neurogenic stuttering, and differentiating psychogenic stuttering from neurogenic stuttering or pure malingering is sometimes challenging. Individuals with psychogenic stuttering often have a history of mental health problems and may show indifference to the stuttering. Some case reports note markedly improved fluency following only one or two speech therapy sessions.

Cross References

- ▶ Aphasia
- ▶ Cognitive Behavioral Therapy
- ▶ Dopamine
- ▶ Neurotransmitters
- ▶ SSRIs
- ▶ Stuttering, Developmental
- ▶ Traumatic Brain Injury

References and Readings

Bloodstein, O., & Ratner, N. B. (2008). *A handbook on stuttering* (6th ed.). Clifton Park: Delmar Learning.

De Nil, L. F., Jokel, R., & Rochon, E. (2007). Etiology, symptomatology, and treatment of neurogenic stuttering. In E. G. Conture & R. F. Curlee (Eds.), *Stuttering and related disorders of fluency* (3rd ed., pp. 326–343). New York: Thieme Medical Publishers.

Saxon, K. G., & Ludlow, C. L. (2007). A critical review of the effect of drugs on stuttering. In E. G. Conture & R. F. Curlee (Eds.), *Stuttering and related disorders of fluency* (3rd ed., pp. 277–293). New York: Thieme Medical Publishers.

Stuttering, Developmental

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Synonyms

Stammering

Definition

Developmental stuttering is an impairment in speech fluency in which the forward flow of speech is disrupted frequently or for an excessively long period of time by one or more of the following types of disfluency: repeated sounds or syllables, repeated words (particularly monosyllabic words), and fixed articulatory postures. Fixed articulatory postures result in either (a) audibly prolonged speech sounds or (b) breaks in speech during which the speaker assumes an articulatory position with little or no sound forthcoming (inaudible prolongations). These symptoms usually emerge between the ages of 2 and 5 years following a period of seemingly normal fluency development, and they may persist throughout the life span.

Historical Background

Definitions of stuttering have evolved substantially during the past century. Early definitions of stuttering often included hypothetical constructs – many of which were psychological in nature. From the 1940s through the 1960s, the prevailing view was that developmental stuttering was essentially a learned behavior, and that parents' reactions to disfluent speech played a critical role in precipitating the onset of stuttering. In contrast, most contemporary models of stuttering explain developmental stuttering from genetic, neurophysiologic, neuroanatomic, and/or psycholinguistic perspectives. Contemporary definitions of stuttering tend to emphasize the salient characteristics of stuttered speech.

Current Knowledge

General Characteristics

The prevalence and incidence rates for developmental stuttering are estimated to be 1% and 5%, respectively, though recent research suggests that these percentages vary somewhat depending upon factors such as the age of the cohort under study. Developmental stuttering affects males more often than females: among children, males are three to four times more likely than females to exhibit the disorder. For many children, developmental stuttering seems to be a short-lived problem. It is estimated that about 75–80% of children who begin to stutter during the preschool years eventually recover, such that their speech fluency resembles that of a typical child. Girls seem more likely to recover from stuttering than boys. Children who recover from stuttering usually do so within 1–2 years of onset, and the odds of recovering from stuttering diminish substantially if stuttering symptoms persist into adolescence. The developmental course of stuttering varies across individuals. For instance, some cases show severe stuttering at onset with gradual improvement or recovery over time, others show mild stuttering at onset with gradual worsening over time, and some others exhibit a pattern of disfluency that remains stable in severity over time. Examples of disfluency types commonly associated with developmental stuttering are presented in Table 1.

Although repetitions of sounds, syllables, and monosyllabic words tend to be a prominent feature of stuttered speech, research shows that people who stutter also produce other types of repetitions (i.e., repetition of polysyllabic words, repetition of phrases) more often than typical speakers. It is common for many of the disfluent breaks to feature multiple types of disfluency (e.g., repeating a part of a word that is subsequently revised; prolonging a sound within a phrase that is repeated). With stuttering, speech disfluencies may be accompanied by signs of excessive physical tension in the speech-related musculature. Although excessive physical tension and signs of struggle are sometimes present at the onset of disorder, more often, they emerge gradually, as the speaker becomes aware of the communication impairment and the ways in which others react to it. Other behaviors, such as forced audible exhalation of air prior to speech initiation may also be symptomatic of stuttering. Speakers also may develop speech-related anxiety and fear, emotional states that most likely reflect the anticipation of negative reactions from a listener.

Individuals who stutter often report that they know precisely which words they want to say, but that they are unable to produce the speech motor movements associated with the words. Many speakers state that they are able to anticipate instances of speech disfluency prior to their actual occurrence. Individuals who stutter may adopt a variety of strategies to conceal their fluency

Stuttering, Developmental. Table 1 Disfluency types commonly associated with developmental stuttering

Disfluency type	Example	Description
Sound repetition	Sometimes he g- g- goes out. . .	Difficulty moving from the [g] to the [o] in "goes." After two unsuccessful attempts, the speaker completes the word.
Syllable repetition	Some-sometimes he goes out. . .	Difficulty moving from the first to the second syllable in "sometimes." After one unsuccessful attempt, the speaker completes the word.
Word repetition	Sometimes he-he- he- he goes out. . .	Difficulty moving from the word "he" to the word "goes." After three unsuccessful attempts, the speaker completes the word.
Audible sound prolongation	Sometimes heeeee goes out. . .	Difficulty moving from the word "he" to the word "goes." The vowel in "he" is prolonged until the speaker is able to proceed to "goes."
Inaudible sound prolongation	Sometimes he (g------)oes out.	Difficulty moving from the [g] to the [o] in "goes." Speaker holds the posture for [g] for an excessive length of time with little or no sound forthcoming and then completes the word.

difficulties from listeners. Examples of such strategies include the following: word avoidance, situation avoidance, word substitution, circumlocution, sentence rephrasing, and insertion of interjections such as "um" prior to words upon which stuttering is expected. Some individuals who stutter find that they can facilitate speech fluency by timing their articulatory movements with rhythmic movements of nonspeech musculature (e.g., finger tapping). This coping strategy and others like it usually are only partially effective, and when used over time, coping behaviors that once had been subtle can become exaggerated, highly noticeable, and detract from communication.

Performance Variability

The disfluencies that characterize stuttered speech are, to some extent, linguistically constrained. That is, stuttering-related behaviors usually occur in conjunction with sounds that lead off syllables rather than sounds that end syllables, and in conjunction with word-initial syllables rather than syllables that occur elsewhere within a word. Syllables that carry linguistic stress are more likely to be stuttered than unstressed syllables. Syntactic effects are observed as well, particularly with children. That is, stuttering behaviors often coincide with the initiation of major grammatical units (i.e., phrases, clauses, utterances). Task complexity appears to affect the likelihood of stuttering as well. For instance, stuttering is more likely to occur at the start of a four-syllable word than it is at the start of a one-syllable word, and an utterance-initial word is more likely to feature stuttering if it is positioned at the start of a long utterance than it is if it is positioned at the start of a short utterance. With children, stuttering is more likely to occur within syntactically complex utterances than it is in syntactically simple utterances.

The symptoms of developmental stuttering are often either absent or markedly reduced in the following conditions: singing, speaking chorally with another person, speaking under delayed or frequency-altered feedback, speaking along with a rhythmic stimulus, and speaking while using a slowed rate of articulation. In most individuals who stutter, the severity of stuttering symptoms will vary, sometimes markedly, across daily activities. A variety of factors, including audience size, the amount of real or perceived time pressure, conversational pace, and listener reactions, have been associated with the contextual variations in stuttering severity.

Etiology

The underlying mechanisms that lead to developmental stuttering are not yet fully understood. As noted, developmental stuttering occurs more often among males than females. The concordance rate for stuttering is significantly higher among monozygotic twins than it is among dizygotic twins, and children who stutter are much more likely to have other relatives who stutter than children who do not stutter. Results from recent research in behavioral and molecular genetics suggest that genetic factors play a principal role in determining one's risk for developing the disorder. Recent brain imaging studies have identified anomalous activation patterns in both cortical and subcortical regions associated with speech

production. Some adults who stutter exhibit atypical anatomical relationships between the left and right auditory temporal cortices. Also, anatomical differences have been identified in both children and adults who stutter in left hemisphere white matter tracts associated with sensorimotor integration for speech-related structures. Studies with both children and adults who stutter have identified an assortment of performance deficits in activities involving motor learning and speech reaction time. Deficits in motor performance have been observed during both oral and manual tasks, particularly during tasks involving complex, sequential movements. Subtle performance deficits also have been detected for select syntactic and phonologic skills as well.

Treatment Approaches

A variety of approaches are used to treat developmental stuttering. As is the case with many other communication disorders, none of the existing treatments for developmental stuttering seems to work for every patient, and none of the treatments can be considered a "cure" for the disorder. Most common are behavioral strategies that seek to improve speech fluency and verbal participation through use of regulated speech articulation (e.g., reduced or monitored articulation rate) and modification of stuttering moments by altering the timing and muscle tension of speech movements. Treatment protocols also may incorporate activities that are designed to improve the speaker's self-awareness of stuttering behaviors and alter how the speaker reacts to stuttered speech (i.e., desensitization). Many speakers find it challenging to generalize fluency management techniques from controlled, clinical settings into activities of daily living. Nonetheless, research suggests that such approaches are generally effective at reducing the severity of fluency impairment, particularly when formal therapy is followed by a systematic maintenance or support program. With adolescents and adults who stutter, treatment may also incorporate cognitive behavioral therapy to address thoughts, attitudes, and beliefs that hinder the person's communicative performance.

Treatment for very young children often aims to shape fluent speech through use of operant conditioning techniques (e.g., praising fluent speech, occasionally highlighting and/or prompting correction of moments of stuttered speech). Such approaches also have been found to be generally effective at improving fluency. Use of assistive devices that present speakers with delayed auditory feedback and/or frequency-altered feedback has increased in recent years. To date, such devices have been primarily used with adults who stutter. Initial reports suggest that the devices are effective at reducing stuttering severity for some people who stutter. As with speakers who use behavioral methods, most speakers who use assistive devices typically exhibit residual stuttering, and speak more fluently in some situations than others.

Future Directions

There is much that remains to be understood about developmental stuttering. Research is ongoing to clarify issues related to the nature of stuttering and ways to best treat it. Issues to be resolved include the following: (a) the role of genetic factors in the transmission and expression of developmental stuttering, (b) the neuroanatomical and neurophysiological mechanisms that lead to stuttered speech, (c) the respective roles of the motor and language systems in stuttered speech, (d) identification of factors that determine whether stuttering will resolve or persist into adulthood, and (e) the development of more effective treatments for developmental stuttering.

Cross References

- ▶ Behavioral Therapy
- ▶ Cognitive Behavioral Therapy
- ▶ Gene
- ▶ Stuttering, Acquired
- ▶ Temporal Lobes

References and Readings

Bloodstein, O., & Bernstein Ratner, N. (2008). *A handbook on stuttering* (6th ed.). Clifton Park, NY: Delmar Learning.

Conture, E. G., & Curlee, R. F. (2007). *Stuttering and related disorders of fluency* (3rd ed.). New York: Thieme Medical Publishers.

Manning, W. (2009). *Clinical decision making in fluency disorders* (3rd ed.). Clifton Park, NY: Cengage Learning.

Yairi, E., & Ambrose, N. G. (2005). *Early childhood stuttering*. Austin, TX: PRO-ED.

Subacute Radiotherapy Effects

- ▶ Early-Delayed Effects of Radiation

Subaponeurotic Hematoma

► Subgaleal Hematoma

Subarachnoid Hemorrhage

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Synonyms

Aneurysmal subarachnoid hemorrhage

Definition

A subarachnoid hemorrhage (SAH) is bleeding into the subarachnoid space that exists between the arachnoid and pia membranes that surround the brain.

Current Knowledge

SAH may result from trauma or occur spontaneously from a ruptured cerebral aneurysm or less commonly from an arteriovenous malformation. SAH comprises 1–7% of all strokes. Incidence is estimated to be about 9 per 100,000 per year. Patients with SAH tend to be younger than those with other types of stroke, but the incidence increases with age. SAH is a medical emergency and can lead to death or severe disability. Up to one-half are fatal, often even before the patient reaches the hospital. Symptoms include rapid onset of a severe headache (often called a "thunderclap headache"), nausea, confusion, altered consciousness, seizure, visual disturbances, cardiac arrhythmias, and focal neurological deficits such as hemiparesis, sensory loss, or aphasia. About one-third of patients have no symptoms other than the headache.

About 85% of all spontaneous SAH results from a ruptured cerebral aneurysm associated with arterial wall weakness that is predisposed to leakage. Most of these aneurysms are located in the circle of Willis. The remaining 15% of subarachnoid hemorrhages are caused by arteriovenous malformations, and less commonly, by drug abuse, anticoagulant use, and bleeding disorders.

Diagnosis is made using neuroimaging, and the specific vascular anomaly may be seen visually on cerebral angiography. SAH may be classified based either on severity of symptoms, using the Hunt and Hess scale, or on size of the hemorrhage, using the Fisher scale. Both of these scales determine treatment course. Medications are used to reduce pain and intracranial pressure and to prevent vasospasm, which can complicate the course of recovery by causing secondary ischemic stroke. Neurosurgical procedures, primarily craniotomy with clipping of the aneurysm or evacuation of the arteriovenous malformation, are used to prevent rebleeding. Recent developments in the management include the use of less-invasive endovascular "coiling," which involves the placement of a "coil" into an aneurysm, performed by threading a catheter through the blood vessels. Choice of which procedure to use depends on the location and size of the aneurysm and the condition of the patient. Complications of SAH include hydrocephalus, seizure, residual neurological deficit (in about 80% of survivors), and a high mortality rate (about 40–50%).

Cross References

► Aneurysm
► Circle of Willis
► Hemorrhagic Stroke
► Intracranial Hemorrhage
► Vascular Malformation
► Vasospasm

References and Readings

Bederson, J. B., Connolly, E. S., Batjer, H. H., Dacey, R. G., Dion, J. E., Diringer, M. N., et al. (2009). Guidelines for the management of aneurysmal subarachnoid hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. *Stroke, 40*, 994–1025.

van Gijn, J., Kerr, R. S., & Rinkel, G. J. (2007). Subarachnoid haemorrhage. *Lancet, 369*, 306–318.

Subcallosal

► Cingulate Gyrus

Subclinical Hypothyroidism

► Hypothyroidism

Subcortical Aphasia

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Definition

Subcortical aphasia refers to language impairments that develop in association with damage-affecting regions other than the cerebral hemisphere, usually the left thalamus and basal ganglia.

Current Knowledge

Although most common following left cortical brain damage, language impairments can occur in association with damage affecting left subcortical regions, including the thalamus and basal ganglia (Nadeau & Crosson, 1997). Subcortical structures serve as relay stations for pathways to and from frontal, parietal, and temporal regions that mediate language processing. Thus, disruption of the relay pathways can lead to impairments in language abilities, though the resulting patterns of impairment are typically milder in form and recover better than aphasias that follow left cortical damage. Because cortical connections for the thalamus and basal ganglia differ, patterns of language breakdown that accompany damage to those structures also tend to differ.

The left pulvinar nucleus of the thalamus has major connections to the left temporal–parietal language cortex (Crosson, 1999). Damage affecting the left thalamus tends to result in fluent forms of aphasia wherein the speaker is able to compose sentence-like utterances. The quality of those sentences is undermined, however, primarily by word-finding difficulties. The pattern of language impairment most typically corresponds to anomic aphasia where word-finding difficulties are the primary symptom. Others with thalamic aphasia present with a pattern of transcortical sensory aphasia, where, in addition to word-finding difficulties, some compromise of auditory comprehension also occurs. That is, the language impairment in thalamic aphasia tends to be associated with disruption of lexical–semantic abilities needed for word-finding and auditory comprehension, with phonologic and grammatic abilities largely intact. Word-finding errors in thalamic aphasia often are characterized by pauses or semantic paraphasias, that is, mistaken selection of closely associated words (Raymer, Moberg, Crosson, Nadeau, & Rothi, 1997). In either form of thalamic aphasia, repetition abilities remain largely intact.

Damage affecting the left basal ganglia has been reported to result in various patterns of aphasia (Nadeau & Crosson, 1997). Impairments for auditory comprehension and verbal fluency have commonly been reported. Word-finding impairments, particularly for verbs, also have been described in individuals with neurologic conditions affecting left basal ganglia networks. With word-finding difficulties come paraphasias or word selection errors and perseverative verbal responses. As in thalamic aphasia, repetition abilities are more frequently preserved. Thus, some patients with left basal ganglia lesions may present with transcortical motor aphasia. In addition to aphasia, left basal ganglia lesions may lead to anarthria in some patients and acquired stuttering in others.

Because left basal ganglia damage most typically occurs following infarction of a branch of the left middle cerebral artery, some have argued that the language impairment associated with some left basal ganglia lesions is in fact an effect of left cerebral hypoperfusion, which has been documented in many individuals with left basal ganglia stroke (Nadeau & Crosson, 1997). Left basal ganglia lesions, therefore, can produce fluent or nonfluent forms of aphasia, with the severity and extent of impairments related to the extent of cortical hypoperfusion in left frontal and temporal cortex (Hillis et al., 2004).

Assessment of subcortical aphasias follows typical procedures, using standardized aphasia tests to evaluate fluency of verbal expression, auditory comprehension, repetition, and word retrieval. Treatment is then applied to the language symptoms, just as in cortical aphasias. Less in known about the patterns of recovery and response to treatment in these type of aphasias, however.

Cross References

► Anomic Aphasia
► Transcortical Sensory Aphasia

References and Readings

Crosson, B. (1999). Subcortical mechanisms in language: Lexical–semantic mechanisms and the thalamus. *Brain and Cognition, 40*, 414–438.

Hillis, A. E., Barker, P. B., Wityk, R. J., Aldrich, E. M., Restrepo, L., Breese, E. L., et al. (2004). Variability in subcortical aphasia is due to variable sites of cortical hypoperfusion. *Brain and Language, 89*, 524–530.

Nadeau, S., & Crosson, B. (1997). Subcortical aphasia. *Brain and Language, 58*, 335–402.

Nadeau, S., & Rothi, L. J. G. (2008). Rehabilitation of subcortical aphasia. In R. Chapey (Ed.), *Language intervention strategies in aphasia and related neurogenic communication disorders* (pp. 530–542). Philadelphia: Wolters Kluwer.

Raymer, A. M., Moberg, P., Crosson, B., Nadeau, S., & Rothi, L. J. G. (1997). Lexical–semantic deficits in two patients with dominant thalamic infarction. *Neuropsychologia, 35*, 211–219.

Subcortical Dementia

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Synonyms

Dementia syndrome of depression; Hydrocephalus; Progressive supranuclear palsy (PSP); Vascular dementia

Definition

Subcortical dementia is a neurological condition associated with degenerative dysfunction of subcortical brain regions such as the substantia nigra, striatum (i.e., caudate nucleus, putamen), and globus pallidus. It is typically seen in vascular, infectious, autoimmune, inflammatory, and movement disorders. Common to most subcortical dementia syndromes are symptoms, including motor dysfunction (e.g., tremor, chorea, gait disturbance), memory deficits (i.e., learning, retrieval), bradyphrenia, and executive dysfunction (e.g., apraxia, disinhibition, disorganization). Apathy, anhedonia, depression, suicidal ideation, blunted affect (i.e., hypomimia), and hallucinations may develop in some individuals as well, and these changes may manifest prodromally or following development of motor and cognitive symptoms. Subcortical dementias generally develop in the sixth and seventh decades, although symptoms can present as early as the third and fourth decades.

Historical Background

Accounts of subcortical dementia date back to as early as 400 BC, with Hippocrates having described the first case of hydrocephalus. More recently identified have been subcortical dementia syndromes such as Parkinson's disease (early 1800s), Huntington's disease (1930s), and progressive supranuclear palsy (1960s).

Current Knowledge

Classification: Dementia syndromes can be classified as having cortical, subcortical, or "mixed" (i.e., both cortical and subcortical) features. An acute confusional state (i.e., delirium) also can mimic dementia. Table 1 lists the clinical features of cortical and subcortical dementia, and delirium.

Features of Subcortical Dementia Syndromes: The incidence and prevalence of dementia varies between syndromes, although subcortical dementias overall, affect approximately 2–5% of the Western population.

- Vascular Dementia: Vascular dementia affects 1–2% of older adults in the United States, and it is the most commonly occurring form of subcortical dementia. It evolves secondary to microvascular ischemic disease that typically is associated with medical risk factors, such as smoking, hypertension, diabetes, and heart disease. However, it can also develop secondary to more acute cerebrovascular events, such as subarachnoid hemorrhage and subdural hematoma. The presenting symptoms can vary from person to person depending on the location and extent of vascular changes which can be identified on neuroimaging.
- Parkinson's disease: Of the 1–2% of the US population diagnosed with Parkinson's disease, approximately 20–40% develops dementia approximately 5–10 years into the disease process. The average age of onset is in the 6th decade of life. Parkinson's disease is characterized by a triad of motor symptoms, including tremor, bradykinesia, and rigidity, with gait dysfunction often developing later in the disease process. Cognitive symptoms tend to follow a pattern of frontal-subcortical dysfunction.
- Huntington's disease: Huntington's disease occurs in less than 1% of the Western population, and it affects men more than women. Symptoms generally manifest in the 4th decade, although onset depends on the number of CAG repeats. The greater the number of repeats, the earlier the age of onset and the more severe the physical and cognitive symptoms are expected to be. It can develop from idiopathic, spontaneous genetic mutations, as well as from genetic transmission from parent to child. In these latter cases, there is 50% inheritance and 100% penetrance.
- PSP: PSP is a rare neurological condition typically diagnosed in the sixth decade. Men and women are

Subcortical Dementia. Table 1 Clinical features of cortical and subcortical dementia, and delirium

Feature	Cortical dementia	Subcortical dementia	Delirium
Onset	Insidious	Insidious	Sudden
Duration	Months to years	Months to years	Hours to days
Course	Progressive	Progressive or constant	Fluctuating
Attention	Normal	Normal (slow response time)	Fluctuating arousal and inattention
Speech	Normal	Hypophonic, dysarthric, mute	Slurred, incoherent
Language	Aphasic	Normal or anomic	Anomia, dysgraphia
Memory	Learning deficit (AD)[a]	Retrieval deficit	Encoding deficit
Cognition	Acalculia, concrete (AD)	Slow, dilapidated	Disorganized
Awareness	Impaired	Usually preserved	Impaired
Demeanor	Unconcerned, disinhibited	Apathetic, abulic	Apathetic, agitated
Psychosis	May be present	May be present	Often florid
Motor signs	None	Tremor, chorea, rigidity, dystonia	Tremor, asterixis
EEG	Mild diffuse slowing	Normal or mild slowing (diffuse or focal)	Moderate-to-severe diffuse slowing

[a]*AD* Alzheimer's disease

Reproduced with permission from Feinberg and Farah (2003, Table 43.5, p. 505)

equally affected, and progression of the disease tends to be relatively rapid. Vertical gaze palsy (particularly trouble looking downward spontaneously) is a hallmark feature of this disease process, and it contributes to falling. As such, falling and poor balance, in addition to personality changes, typically often are the initial presenting symptoms in patients with this syndrome.

- Hydrocephalus: Estimates regarding incidence and prevalence of hydrocephalus are limited and variable, as hydrocephalus can occur congenitally, idiopathically in adults (i.e., normal pressure hydrocephalus) or secondary to neurological illnesses such as tumor or head injury (i.e., obstructive hydrocephalus). In the latter cases, hydrocephalus often is classified as sequelae of neurological illness rather than a primary medical condition. With this said, the average prevalence of hydropcephalus in adults and children is estimated to be less than 1%. Independent of etiology, typical symptoms include personality change, incontinence, and gait dysfunction.
- Dementia Syndrome of Depression: Statistics for Dementia syndrome of depression are variable and not well-reported, as the use of this diagnostic label is somewhat controversial. Many patients often are misdiagnosed as having dementia rather than a primary psychiatric disorder, whereas others are classified in the literature as having a major depressive episode with limited mention of cognitive and motor sequelae.

Neuropsychological Examination: Patients with subcortical dementia typically perform well on the Mini Mental State Examination (MMSE), which is sensitive to cortical yet not subcortical dysfunction. Therefore, alternate measures of mental status that are sensitive to both cortical and subcortical dysfunction (e.g., ▶ Mattis Dementia Rating Scale) are preferred. To evaluate attention, mental processing speed, and executive skills, administration of measures such as a digit span test, trail-making test, Luria motor sequencing, and Stroop Test are recommended, as these tasks are sensitive to frontal lobe dysfunction. The Wisconsin Card Sorting Test lacks sensitivity for older individuals and, therefore, is of limited benefit for the assessment of executive dysfunction in this population. Verbal fluency can be assessed with category and letter fluency tasks, such as FAS, CFL, and animal fluency. In subcortical dementias, phonemic fluency typically is more compromised than category fluency. Word-finding ability typically is preserved, and this can be evaluated with a picture naming test such as the Boston Naming test. Deficits in spatial processing also can be observed, although it is important to first rule out whether frank vision problems (e.g., cataracts) and time limitations (i.e., bradyphrenia) contribute to reduced performances in this domain. In areas of learning and memory, learning of rote, list information (e.g., HVLT, RAVLT) tends to be more compromised than learning of semantically organized material, such as stories (e.g., ▶ WMS-III). This learning disparity reflects predominant dysfunction in frontal and

subcortical brain regions. That is, list learning is mediated by subcortical memory structures, whereas story learning is regulated by neocortical memory systems. Related to this, individuals may show diminished learning and retrieval of information, yet they perform very well on recognition measures. Psychologically, hypomimia can be misinterpreted as depression; therefore, evaluation of mood should extend beyond behavioral observation and include objective assessment of apathy and anhedonia, as well as suicidality and hopelessness.

Treatment: Most treatment options for subcortical dementia are palliative and help reduce the degree of disability caused by cognitive, motor, and mood symptoms.

- Pharmacology: Various medications can be used to provide symptom relief. For example, antidepressants and sedatives can help alleviate mood symptoms. Cholinesterase inhibitors help slow the progression of memory dysfunction. Dopamine and glutamate agonists can relieve motor symptoms in movement disorders. However, side effects of dopaminergic agonists include dyskinesias, hallucinations, and sleep disturbances.
- Rehabilitation: Speech and language symptoms, including hypophonia, dysarthria, and dysnomia, can be addressed with speech therapy. Cognitive skills training (a.k.a. cognitive remediation) can help individuals develop strategies to compensate for cognitive deficits, particularly as they pertain to attentional and executive dysfunction. Occupational and physical therapies may help alleviate motor difficulties.
- Psychotherapy: Cognitive-behavioral psychotherapeutic treatment (individual or group) is an efficacious treatment option.
- Surgical Intervention: In subcortical dementias such as Parkinson's disease and Dementia syndrome of depression, deep brain stimulation of the subthalamic nucleus can reduce motor and mood symptoms, respectively.
- Caregiver Training and Support: Caregiver burden increases as subcortical dementia progresses. Therefore, it is helpful for caregivers to become involved in educational and psychotherapeutic support groups. Participation in these groups can help reduce the burden and enhance availability and implementation of treatments for individuals with dementia.
- Monitor Progress: Serial neuropsychological evaluations can help track progression of the disease process, assess efficacy of treatments, and provide information about a patient's level of independence and decision-making abilities.

Future Directions

Research studies currently are exploring the possible causes and cures for subcortical dementia syndromes. At present, there exist medication and neurosurgical trials that aim to help slow the progression of cognitive deterioration. Neuroimaging, genetic, and neuropsychological studies also are attempting to identify predictors of dementia and related motor symptoms.

Cross References

- ▶ Basal Ganglia
- ▶ Binswanger's Disease
- ▶ CADASIL
- ▶ Caudate Nucleus
- ▶ Cognitive Rehabilitation
- ▶ Cortical–Subcortical Loop
- ▶ Corticobasal Ganglionic Degeneration
- ▶ Dementia with Lewy Bodies
- ▶ Globus Pallidus
- ▶ Huntington's Disease
- ▶ Movement Disorders
- ▶ Multi-Infarct Dementia
- ▶ Multiple Sclerosis
- ▶ Parkinson's Disease
- ▶ Parkinson Plus Syndromes
- ▶ Pseudodementia
- ▶ Striatum

References and Readings

Blumenfeld, H. (2002). Basal ganglia. In H. Blumenfeld (Ed.), *Neuroanatomy through clinical cases* (pp. 689–735). Massachusetts: Sinauer Associates, Inc.

Cato, M. A., & Crosson, B. A. (2006). Stable and slowly progressive dementias. In D. K. Attix & K. A. Welsh-Bohmer (Eds.), *Geriatric neuropsychology: Assessment and intervention* (pp. 89–102). New York: The Guilford Press.

Feinberg, T. E., & Farah, M. J. (2003). *Behavioral neurology and neuropsychology* (2nd ed.). New York: The McGraw-Hill Companies.

Lichter, D. G., & Cummings, J. L. (2001). *Frontal-subcortical circuits in psychiatric and neurological disorders*. New York: The Guilford Press.

Morgan, J. E., & Ricker, J. H. (2008). *The textbook of clinical neuropsychology*. New York: Taylor and Francis.

Ropper, A. H., Brown, R. H., & Brown, R. I. (2005). *Adams and Victor's principles of neurology*. New York: McGraw-Hill Professional Publishing.

Welsh-Bohmer, K. A., & Warren, L. H. (2006). Neurodegenerative dementias. In D. K. Attix & K. A. Welsh-Bohmer (Eds.), *Geriatric neuropsychology: Assessment and intervention* (pp. 56–88). New York: The Guilford Press.

Subcortical Leukoencephalopathy

► Binswanger's Disease
► Vascular Dementia

Subcortical Vascular Dementia

► Binswanger's Disease

Subcortical Vascular Leukoencephalopathy

► Small Vessel Ischemic Disease

Subdural Hematoma

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Synonyms

SDH

Definition

A subdural hematoma (SDH) is a bleeding inside the skull but outside of the brain, located on the surface of the brain, usually resulting from severe head trauma.

Current Knowledge

SDH occurs when there is tearing of the tiny bridging veins that connect the dura covering and the surface of the brain. In acute SDH, the collection fills up with blood rapidly, compressing brain tissue, and resulting in brain damage and obvious symptoms. Chronic SDH tends to be more subtle, occurring after minor head trauma, with no or minimal symptoms, going unnoticed for days to weeks. Occasionally, SDH occurs spontaneously without obvious cause. SDH occurs with greater frequency in older adults than in younger persons because of the frequency of falls and the altered brain anatomy that places their bridging veins at greater risk of tearing. Anticoagulant use and alcohol abuse also increase the risk. Symptoms include lethargy or confusion, balance or gait difficulty, nausea or headache, seizure or visual disturbances, and weakness or sensory loss. Diagnosis is made on neuroimaging. Treatment involves immediate craniotomy and evacuation of the hematoma as promptly as possible. Outcome depends on the location and size of the hematoma and how quickly treatment is instituted. Long-term complications include seizures, cognitive dysfunction, hemiparesis, sensory loss, and other neurological deficits.

Cross References

► Intracranial Hemorrhage

References and Readings

Maurice-Williams, R. S. (1999). Chronic subdural hematoma: An everyday problem for the neurosurgeon. *British Journal of Neurosurgery 13*, 547–549.

Weigel, R., Schmiedek, P., & Krauss, J. K. (2003). Outcome of contemporary surgery for chronic subdural haematoma: Evidence based review. *Journal of Neurology, Neurosurgery & Psychiatry, 74*, 937–943.

Subdural Hygroma

► Hygroma

Subfornical Organ

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Synonyms

Circumventricular organ

Definition

Overlying the third ventricle, the subfornical organ is one of the circumventricular organs and has fibers that project

to the hypothalamus. The subfornical organ is very important in maintaining the constancy of plasma osmolarity and is the principal site for control of salt-intake behavior. Its neurons regulate fluid electrolyte balance by controlling thirst, sodium excretion, blood volume regulation, and secretion of vasopressin by the hypothalamus. Because the subfornical organ is not protected by the blood–brain barrier, its neurons can respond to plasma osmolarity as well as to factors present in the systemic circulation such as angiotensin.

Cross References

► Blood–Brain Barrier
► Hypothalamus

Subgaleal Hematoma

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Synonyms

Subaponeurotic hematoma

Definition

Potential space, referred to as subgaleal or subaponeurotic space, extends between the skin and the skull from the orbital ridge to the nape of the neck and laterally to the ears. Trauma in this area may result in bleeding into a portion of this space. Such a collection of blood is referred to as a subgaleal (or less commonly, subaponeurotic) hematoma. A subgaleal hematoma may be associated with closed head injury, but because the injury is external to the skull, it does not provide a direct evidence of intracranial or brain injury. Subgaleal hematoma is also a rare, but potentially fatal, complication to the newborn during childbirth.

Cross References

► Head Injury

References and Readings

Coolings, D., & Vicellio, P. (1991). Massive subgaleal hematoma following minor head trauma. *The Journal of Emergency Medicine, 9*(1), 33–5.

Kilani, R. A., & Wetmore, J. (2005). Neonatal subgaleal hematoma: presentation and outcome – Radiological findings and factors associated with mortality. *American Journal of Perinatology, 23*(1), 41–8.

Subgaleal Hemorrhage

► Subgaleal Hematoma

Subgenual

► Cingulate Gyrus

Subiculum

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Definition

The subiculum is a transitional region of cortex between the six-layered paleocortex of the entorhinal cortex and the trilaminar archicortex of the hippocampus. It is usually considered to be part of the "hippocampal formation" along with the hippocampus proper and dentate gyrus, and its neurons contribute to the origin of the fornix. The "perforant path" of the entorhinal input to the hippocampal formation traverses the subiculum to reach the dentate gyrus.

Cross References

► Dentate Gyrus
► Fornix
► Hippocampus

Sublimation

► Inhibition

Suboptimal Effort

► Response Bias

Subpoena

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Definition

A subpoena is a document that commands the appearance or response of an individual. The subpeona requires one to appear at a certain time and place to give testimony upon a certain matter. There two kinds of subpoenas: subpoena testificandum, which requires the individual to testify and subpoena duces tecum, which commands to provide evidence. There are three options in response to a subpoena. The first is to comply, the second is to request a motion to quash and convince the court why you should not comply, and the last is to refuse to comply. If one refuses to comply, the court may hold the individual in contempt.

References and Readings

http://en.wikipedia.org/wiki/Subpoena

Subpoena Duces Tecum

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Definition

A subpoena which requests items to be brought with the person to deposition or trial is called a "subpoena duces tecum." A subpoena is an order directed to an individual commanding him/her to appear at a deposition or in court on a certain day to testify or produce documents in a pending lawsuit. The power to subpoena a person is granted by officers of the court, such as clerks of courts, attorneys, and judges. A person may be subpoenaed to appear in court or any designated location to provide testimony for trial or deposition or produce documents or other evidence. Failure to comply with a subpoena may subject a person to being held in contempt of court if the absence appears to be intentional or without cause.

Melton, Petrila, Poythress, and Slobogin (2007) wrote that fear of a subpoena may cause some clinicians to keep only minimal information in their file or to create a separate "personal" file. Yet, merely labeling a file "personal" does not exclude the clinician from having to respond to the subpoena. If the judge learns about the hiding of such evidence, the clinician could be held in contempt. They suggest that the best approach is to keep complete files. Any information that is irrelevant can and should be excluded by the judge. Information that is relevant should not embarrass the examiner who has done an honest job.

References and Readings

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.

Substance Abuse

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Synonyms

Alcohol abuse; Drug abuse; Poly-substance abuse

Definition

Substance abuse is the destructive, damaging, or dangerous use of psychoactive substances including alcohol and

prescription or illicit drugs. According to the American Psychiatric Association, substance abuse is defined by the Diagnostic and Statistical Manual of Mental Disorders, fourth edition, text revision (2000), as a maladaptive pattern of substance use manifested by recurrent and significant adverse consequences related to the repeated use of substances. The maladaptive pattern leads to clinically significant impairment or distress as manifested by one (or more) of the following, occurring within a 12-month period:

1. Recurrent substance use resulting in a failure to fulfill major role obligations at work, school, or home (such as repeated absences or poor work performance related to substance use; substance-related absences, suspensions, or expulsions from school; or neglect of children or household)
2. Recurrent substance use in situations in which it is physically hazardous (such as driving an automobile or operating a machine when impaired by substance use)
3. Recurrent substance-related legal problems (such as arrests for substance related disorderly conduct)
4. Continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance (e.g., arguments with spouse about consequences of intoxication and physical fights)

Alternatively, the symptoms have never met the criteria for substance dependence for this class of substance (p. 182).

Substance abuse can manifest itself as physical, emotional, cognitive, or behavioral symptoms. For example, a person with substance abuse issues may present with poor personal hygiene, an unkempt or scruffy appearance, bloodshot eyes, and ashen skin. He/she may appear depressed or anxious. Cognitive and behavioral symptoms may include motor slowness, diminished visual tracking ability, verbal outbursts, or treatment noncompliance.

Substance abuse has been associated with the cause of neurologic disability such as spinal cord injury and brain injury and has also been shown to have a long-term impact on person's post-injury or neurologic insult. Neuropsychologists must consider the role of pre- and post-injury substance use and abuse when testing and treating these individuals. Continued use and abuse of substances could have an impact on physical, cognitive, and emotional functioning such as slowed motor responses, limited processing and executive functioning skills, and increased depression and dysphoria. Additionally, substance abuse could impact the effectiveness of prescribed medications, limiting their intended outcome.

Some popular scales for assessing substance abuse include the CAGE – an acronym of four common substance abuse issues (e.g., cut down, annoyed, guilt, eye-opener), the Substance Abuse Life Circumstance Evaluation (SALCE), the Michigan Alcohol Screening Test (MAST), the Screening Brief Intervention and Referral to Treatment (SBIRT), and the Substance Abuse Subtle Screening Inventory (SASSI).

Cross References

- ▶ Alcoholism
- ▶ Alcohol Dependence
- ▶ Substance Abuse Disorders

References and Readings

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

Ersche, K., & Sahakian, B. (2007). The neuropsychology of amphetamine and opiate dependence: Implications for treatment. *Neuropsychology Review, 17*(3), 317–336.

Kunins, H., Gilbert, L., Whyte-Etere, A., Meissner, P., & Zachary, M. (2007). Substance abuse treatment staff perceptions of intimate partner victimization among female clients. *Journal of Psychoactive Drugs, 39*(3), 251–257.

Meara, E., & Frank, R. (2005). Spending on substance abuse treatment: how much is enough? *Addiction, 100*(9), 1240–1248.

Thoma, P., & Daum, I. (2008). Working memory and multi-tasking in paranoid schizophrenia with and without comorbid substance use disorder. *Addiction, 103*(5), 774–786.

Van der Plas, E., Crone, E., Wildenberg, W., Tranel, D., & Bechara, A. (2008). Executive control deficits in substance-dependent individuals: A comparison of alcohol, cocaine, and methamphetamine and of men and women. *Journal of Clinical & Experimental Neuropsychology, 31*(6), 706–719.

Verdejo-García, A., López-Torrecillas, F., Giménez, C., & Pérez-García, M. (2004). Clinical implications and methodological challenges in the study of the neuropsychological correlates of cannabis, stimulant, and opioid abuse. *Neuropsychology Review, 14*(1), 1–41.

Versland, A., & Rosenberg, H. (2008). Substance abuse administrators' attitudes regarding psychosocial functioning of persons with single diagnoses versus severe dual diagnoses. *Journal of Dual Diagnosis, 4*(2), 126–141.

Substance Abuse Disorders

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Synonyms

Addiction; Substance abuse; Substance dependence

Definition

Substance abuse disorders incorporate both substance abuse and substance dependence and are a classification to describe addictive behaviors. Substance abuse is a maladaptive pattern of substance use that results in significant impairment or distress in the individual's life. Substance dependence manifests with at least three of the following indicators: (1) substance tolerance; (2) withdrawal from the substance; (3) the substance is taken in increasing quantities; (4) there is a persistent desire or unsuccessful attempts to reduce the intake of the substance; (5) a significant amount of time is spent in obtaining the substance, using it, or recovering from its effects; (6) the substance use causes significant interference in the individuals social, occupational, or recreational functioning; and (7) the substance use is continued despite threats to health or safety. Substances recognized within the DSM-IV-TR are alcohol, amphetamines, caffeine, cannabis, cocaine, hallucinogens, inhalants, nicotine, opioids, phencyclidine, and sedatives. Risk factors for substance abuse include depressive and anxiety disorders, trauma history, and traumatic brain injury. Other risk factors such as age, gender, race, and ethnicity vary by the type of substance abuse disorder.

Cross References

- ► Alcohol Abuse
- ► Alcohol Dependence
- ► Tolerance

References and Readings

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

Sadock, B. J., & Sadock, V. A. (2003). *Kaplan and Sadock's synopsis of psychiatry: Behavioral sciences, clinical psychiatry.* Philadelphia, PA: Lippincott Williams & Wilkins.

Substance Dependence

► Substance Abuse Disorders

Substantia Gelatinosa

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Synonyms

Rexed's lamina II

Definition

Substantia gelatinosa is a collection of cells in the gray area (dorsal horns) of the spinal cord. Found at all levels of the cord, it receives direct input from the dorsal (sensory) nerve roots, especially those fibers from pain and thermoreceptors. Rather than directly contributing efferent fibers to the anterolateral system or spinothalamic tracts, the substantia gelatinosa's main connections appear to be to other lamina within the gray matter of the cord, including the contralateral lamina II. The substantia gelatinosa is also believed to receive input from descending fibers. Composed of fine networks of interneurons, it contains high levels of substance P as well a large number of opiate type receptors, both of which are involved in the perception of pain. Thus, the substantia gelatinosa is believed to play an important role in the modulation of and/or mediation of pain perception at the spinal level.

Cross References

- ► Pain Perception

References and Readings

Carpenter, M. B., & Sutin, J. (1983). *Human neuroanatomy.* Baltimore: Williams & Wilkins.

Haines, D. E. (Ed.). (1997). *Fundamental neuroscience.* New York: Churchill Livingstone.
Nolte, J. (2002). *The human brain: An introduction to its functional anatomy.* St. Louis: C.V. Mosby.

Substantia Grisea

► Gray Matter

Substantia Nigra

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Definition

The substantia nigra (SN) is a nucleus in the ventral part of the midbrain tegmentum, which is darkly pigmented (hence its name) because neuromelanin is a by-product of the synthesis of catecholamines, like dopamine. The substantia nigra is the principal source of dopamine in the brain. The SN contains two subdivisions: the pars compacta (SNc), which contains the cell bodies of dopaminergic neurons, and the pars reticulata (SNr), which contains the cell bodies of GABA-ergic neurons. The SNc projects primarily to the striatum (caudate and putamen), whereas the SNr projects primarily to the superior colliculus, motor thalamus (VA/VL), and intralaminar nuclei of the thalamus. In Parkinson's disease, there is a substantial loss of dopaminergic neurons in the SNc, which results in a resting tremor, bradykinesia, freezing, and festinating gait.

Cross References

► Dopamine
► Midbrain
► Parkinson's Disease
► Superior Colliculus
► Thalamus

Subsyndromal Depression

► Minor Depressive Disorder

Subtest Analysis

► Subtest Scatter

Subtest Scatter

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Synonyms

Intersubtest scatter; Subtest analysis

Definition

Subtest scatter refers to the variability of individual subtest scaled scores within a test. Common methods for measuring subtest scatter are to examine the difference between the individual's highest and lowest scaled scores within a test or to examine the difference between subtest scores and the average scores across all subtests.

Current Knowledge

Variability of an individual's scaled scores across subtests on cognitive measures used to assess a broad construct such as intelligence or memory may be clinically important. While the research in this area has been inconsistent, variability across subtests may be diagnostically relevant. Some research has found that subtest scatter may suggest learning disability, neurological dysfunction, or emotional problems. Other research has revealed that subtest scatter is ineffective for distinguishing clinical from nonclinical groups. Regardless of the diagnostic utility, subtest scatter is helpful in understanding cognitive strengths and weaknesses as it relates to the individual's functioning.

Evaluation of subtest scatter can allow for hypothesis generation about the individual's abilities and limitations. Such an approach toward interpretation relies heavily on evaluation of individual subtests rather than utilization of global composite scores for interpretation. It has been argued that the examination of individual scatter encourages a multidimensional view of intellectual functioning.

Cross References

► Intelligence
► Intelligence Quotient

References and Readings

Debring, C., Satz, P., Van Gorp, W., Chervinsky, A., & Uchimaya, C. (1994). WAIS-R intersubtest scatter in patients with dementia of Alzheimer's type. *Journal of Clinical Psychology, 50*, 753–758.

Kramer, J. J., Henning-Stout, M., Ullman, D. O., & Schellenberg, R. P. (1987). The viability of scatter analysis on the WISC-R and the SBIS: Examining a vestige. *Journal of Psychoeducational Assessment, 5*, 37–47.

Matarazzo, J. D., Daniel, M. H., Prifitera, A., & Herman, D. O. (1988). Inter-subtest scatter in the WAIS-R standardization sample. *Journal of Clinical Psychology, 44*, 940–950.

Watkins, M. W. (2003). IQ subtest analysis: Clinical acumen or clinical illusion. *The Scientific Review of Mental Health Practice, 2*, 1–45.

Subthalamus-Subthalamic Nucleus, Zona Incerta

► Diencephalon

Subthreshold Depression

► Minor Depressive Disorder

Successive Processing

► Sequential Processing

Suck Reflex

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Definition

The suck reflex is elicited by stimulating the infant's lips and palate, causing a sucking response. It is one of the *frontal release signs*, primitive reflexes that are normal in infants, disappear with brain maturation allowing inhibition, and reappear (are "released") in disorders that affect the frontal lobes. Like most primitive reflexes, the suck reflex probably has evolutionary/adaptive advantage in infant apes, assisting them in suckling.

References and Readings

Goetz, C. G. (2007). *Textbook of clinical neurology* (3rd ed.). Philadelphia: Saunders.

Suicidal Ideation

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Synonyms

SI; Suicidal intention; Suicidal thoughts; Suicidality

Definition

Suicidal ideation refers to an individual's thoughts, intentions, or plans to commit suicide. This includes both passive ideation where an individual may wish to be dead, and more active ideation where an individual has considered the specifics of suicide (e.g., the means of suicide, the timing, or the location), and may even have begun planning the act. It is most commonly seen among individuals with mental illness, including depressive disorders, schizophrenia, alcohol and

other substance dependence, personality disorders, and some anxiety disorders. Physical illnesses, including cancer and HIV are also significant risk factors. Neurological conditions such as traumatic brain injury show significantly increased risk for hopelessness and suicidality. The suicide rate in the USA is approximately 13 per 100,000. While women are at four times the risk for suicide attempts when compared to men, men have four times the risk of dying from suicide. Men are also at increased risk in old age, with their risk more than doubling after the age of 75 years.

Cross References

► Major Depression

References and Readings

Kessler, R. C., Borges, G., & Walters, E. E. (1999). Prevalence of and risk factors for lifetime suicide attempts in the National Comorbidity Survey. *Archives of General Psychiatry, 56*, 617–626.

Sadock, B. J., & Sadock, V. A. (2003). *Kaplan and Sadock's synopsis of psychiatry: Behavioral sciences, clinical psychiatry.* Philadelphia, PA: Lippincott Williams & Wilkins.

Simpson, G., & Tate, R. (2002). Suicidality after traumatic brain injury: Demographic, injury, and clinical correlates. *Psychological Medicine, 32*, 687–697.

U. S. Public Health Service (2001). *National strategy for suicide prevention: Goals and objectives for action.* Rockville, MD: USDHHS.

Suicidal Intention

► Suicidal Ideation

Suicidal Thoughts

► Suicidal Ideation

Suicidality

► Suicidal Ideation

Superior Colliculus

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Definition

There are two superior colliculi located on the dorsal aspect of the brain stem and sit below the thalamus. They are involved with initiation and control of shifts in gaze as well as eye–head coordination. Input to the colliculi originates in the cerebral cortex, the inferior colliculus, the retina, the basal ganglia, and the spinal cord. Fibers from the superior colliculi project to the paramedian pontine reticular formation and the spinal cord.

Cross References

► Basal Ganglia
► Inferior Colliculi
► Pons
► Reticular Activating System

References and Readings

Oster, S. F., & Sretavan, D. W. (2003). Connecting the eye to the brain: The molecular basis of ganglion cell axon guidance. *British Journal of Ophthalmology, 87*(5), 639–645.

Sten, B. E. (2005). The development of a dialogue between cortex and midbrain to integrate multisensory information. *Experimental Brain Research,* 166, 305–315.

Superior Longitudinal Fasciculus

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Definition

The superior longitudinal fasciculus is a pair of long bidirectional association fiber bundles connecting the

frontal, occipital, parietal, and temporal lobes. These fibers are known to be involved in regulation of motor behavior, spatial attention and visual oculomotor functions, transfer of somatosensory information between parietal and motor cortices, articulation of language, and integration of auditory information.

Cross References

► Associational Fibers

Superior Olivary Complex

► Superior Olivary Nucleus

Superior Olivary Nucleus

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Synonyms

Superior olivary complex

Definition

Brainstem nucleus which lies in the tegmental portion of the pons and is associated with the auditory system. The superior olivary nucleus can be divided into a medial and a lateral group. As both receive bilateral input from the dorsal and ventral cochlear nuclei, they represent the first level at which inputs from both ears converge. Due to this bilateral input, these nuclei are believed to play an important role in horizontal sound localization. The superior olivary nuclei give rise to the lateral lemniscus, a white matter auditory pathway that terminates in the inferior colliculi.

Cross References

► Auditory System

Supervision Rating Scale

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Synonyms

SRS

Description

The Supervision Rating Scale (SRS) measures the level and type of supervision that a patient/subject receives from caregivers. Supervision refers to any form of help that might require a caregiver to be in close proximity to the subject (e.g., supervision for safety, verbal cues, and physical assistance). The SRS rates level of supervision on a single 13-point ordinal scale. The raw score can be collapsed into an optional descriptive category (Independent, Overnight Supervision, Part-Time Supervision, Full-Time Indirect Supervision, and Full-Time Direct Supervision). There have been suggestions to further collapse the score into a dichotomy (Independent versus Supervised) or a trichotomy (Independent versus Moderate Supervision versus Heavy Supervision) (Hart, Millis, Novack, & Englander, 2003). The SRS was designed to be rated through interviews with either the subject or an informed caregiver who is aware of the level of supervision the subject receives. Ratings are based on the level of supervision actually received, not how much supervision is needed or might be appropriate. Ratings are not based entirely on the time spent in supervision, but also on quality (direct versus indirect supervision) and intensity (constant observation or need for assistance). A limitation of the SRS is that there is no explanation of why supervision is received, or if it is even necessary.

The SRS is a nonproprietary measure. The SRS can be completed in person, or by phone. A structured interview is available to facilitate standardized ratings. The time to administer the SRS is typically less than 5 min.

Historical Background

The SRS was developed and published by Corwin Boake at The Institute for Rehabilitation Research (TIRR) in

1996. The SRS was adopted for use in the National Institute of Disability and Rehabilitation Research (NIDRR) Traumatic Brain Injury Model Systems (TBIMS) National Database in 1998 as part of the follow-up assessments given to people at 1, 2, 5, 10, and every 5 years post-injury.

Additional information on the SRS is available at the Center for Outcome Measurement in Brain Injury (www.tbims.org/combi/srs).

Psychometric Data

Validity

SRS Ratings showed consistent relations with type of living arrangement (living independently, living in the community with support, living in a facility) and with independence in self-care and instrumental ADL (Multidimensional Functional Assessment Questionnaire). SRS ratings were also strongly associated with ratings on the DRS (Disability Rating Scale) and GOS (Glasgow Outcome Scale) (Boake, 1996). A Rasch analysis of the five level SRS (based on categories) showed that the SRS fit adequately as an indicator of general functioning (Johnston, Shawaryn, Malec, Kreutzer, & Hammond, 2006).

Reliability

Interrater reliability of the SRS, evaluated on a sample of 19 patients was satisfactory (intraclass correlation = .86, weighted kappa = .64) (Boake, 1996). In a separate sample of 19 individuals, there was 100% agreement for the five-level version of the SRS, but a kappa of .46 for the 13-level version (van Baleen et al., 2006).

Ceiling Effects

The SRS appears to be prone to ceiling effects (69% rated as independent of supervision) in persons interviewed at 1 year post moderate to severe brain injury (Hart et al., 2003). Tate found that in a sample of 67 individuals, 20–26 years post severe TBI, there was a ceiling effect of 73% (independent), and further noted that there were many levels of the SRS that were not represented or used (Tate, 2004). Other authors have noted similar long-term findings of ceiling effects at or near 70% (Hall, Bushnik, Lakisic-Kazazic, Wright, & Cantagallo, 2001; Wood & Rutterford, 2006).

Clinical Uses

A patient's level of supervision is a partial proxy for disability. Problems in the areas of cognition, physical ability, and behavior issues should reflect higher levels of supervision. Supervision implies the continuous or intermittent need for another person to provide assistance. Level of supervision is also important as an outcome measure because of its assumed relation to cost and burden of care. This measure has primarily been used with outpatient or community samples of individuals with TBI.

Cross References

- ▶ Center for Outcome Measurement in Brain Injury
- ▶ Traumatic Brain Injury
- ▶ Traumatic Brain Injury Model System

References and Readings

Boake, C. (1996). Supervision rating scale: A measure of functional outcome from brain injury. *Archives of Physical Medicine and Rehabilitation, 77*, 765–772.

Center for Outcome Measurement in Brain Injury, http://www.tbims.org/combi/srs

Hall, K. M., Bushnik, T., Lakisic-Kazazic, B., Wright, J., & Cantagallo, A. (2001). Assessing traumatic brain injury outcome measures for long term follow-up of community-based individuals. *Archives of Physical Medicine and Rehabilitation, 82*, 367–374.

Hart, T., Millis, S., Novack, T., Englander, J., et al. (2003). The relationship between neuropsychologic function and level of caregiver supervision at 1 year after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 84*, 221–230.

Johnston, M. V., Shawaryn, M. A., Malec, J., Kreutzer, J., & Hammond, F. M. (2006). The structure of functional and community outcomes following traumatic brain injury. *Brain Injury, 20*(4), 391–407.

Tate, R. L. (2004). Assessing support needs for people with traumatic brain injury: The care and needs scale (CANS). *Brain Injury, 18*, 445–460.

van Baalen, B., Odding, E., van Woensel, M. P. C., et al. (2006). Reliability and sensitivity to change of measurement instruments used in traumatic brain injury population. *Clinical Rehabilitation, 20*, 686–700.

Wood, R. L., & Rutterford, N. A. (2006). Psychosocial adjustment 17 years after severe brain injury. *Journal of Neurology, Neurosurgery & Psychiatry, 77*, 71–73.

Supervisory Attentional System

- ▶ Controlled Attention

Supplementary Motor Area (SMA)

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Synonyms

Motor area; Motor cortex

Definition

The supplementary motor area (SMA) is situated medially and is in front of the primary motor cortex and medial to the premotor cortex. The SMA is also known as Brodmann area 6. The SMA can be divided into two areas, pre-SMA (rostral) and SMA proper (caudal). The SMA proper is involved in planning of learned complex movements (thought to be internally driven and not driven by visual cues) and in coordinating movements involving both hands. The pre-SMA is involved in the learning of new motor sequences.

Cross References

► Movement Disorders
► Primary Cortex

References and Readings

Bannur, U., & Rajshekhar, V. (2000). Post operative supplementary motor area syndrome: Clinical features and outcome. *British Journal of Neurosurgery, 14*(3), 204–210.

Engel, A., Burke, M., Fiehler, K., Bien, S., & Rösler, F. (2008). Motor learning affects visual movement perception. *European Journal of Neuroscience, 27*(9), 2294–2302.

Kermadi, I., Liu, Y., Tempini, A., Calciati, E., & Rouiller, E. (1998). Neuronal activity in the primate supplementary motor area and the primary motor cortex in relation to spatio-temporal bimanual coordination. *Somatosensory and Motor Research, 15*(4), 287–308.

Yamane, F., Muragaki, Y., Maruyama, T., Okada, Y., Iseki, H., Ikeda, A., et al. (2004). Preoperative mapping for patients with supplementary motor area epilepsy: Multimodality brain mapping. *Psychiatry and Clinical Neurosciences, 58*(3), S16–S21.

Support

► Advocacy

Supported Employment

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Synonyms

Customized employment

Definition

A model of employment supports, originally developed in the 1970s and 1980s, which facilitates the competitive employment of individuals with significant disabilities within integrated employment settings. Supported employment originated within federal and state-funded demonstration projects with the intent to show that persons with significant mental retardation could work. Supported employment is designed for those whom the competitive employment has not traditionally occurred, and who require ongoing support services due to the nature and severity of their disability in order to perform a job.

Supported Employment philosophy rejected the premise of "readiness" which required individuals to progress through a continuum of service settings gaining skills and inclusion as they progressed. This continuum resulted in the systematic community and economic exclusion of individuals with significant disabilities. Service settings consisted of segregated programs such as developmental day and sheltered employment programs.

Supported Employment became a formal service of the federal/state VR program with the Rehabilitation Act Amendments of 1986. The Federal Definition of Supported Employment as stipulated by Public Law 102–569 includes the following basic components: competitive employment; integrated work setting; significant disabilities with no employment history or intermittent history and long-term support.

The model utilizes evidence-based principles to include:

- Consumer Choice in programs and services.
- Employment supports integrated with mental health services, if needed
- Services are focused on employment as the goal
- A rapid job search approach is used
- Job selection is individualized with attention to consumer preferences
- Supports are ongoing
- Benefits counseling is used to educate consumers on the effects of earnings on benefits

Supported employment provides assistance such as employment specialists; customized employment matching and selection; job training to learn tasks associated with a respective job and to maintain performance standards once the task is learned; assistive technology, transportation, and benefits planning. Supports are highly individualized and are person-centered.

Cross References

► Job Development and Placement
► Sheltered Employment

References and Readings

Wehman, P., Inge, K. J., Revell, W. G., & Brooke, V.A. (Eds.). (2007). *Real work for real pay.* Baltimore: Brookes Publishing

Supported Living

► Transitional Living

Suppression

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Definition

The tendency to consistently ignore or fail to respond to stimuli on one side of the body. While this can occur theoretically in any number of situations, it is most likely to occur during double or bilateral, simultaneous stimulation (DSS) procedures when testing visual, somatosensory, or auditory perception. It may be present in an entire tactile or visual hemifield, or limited to a more restricted portion, such as in an upper extremity (tactile) or upper quadrant of the visual field. Depending on the severity of the deficit, it may be invariably present or present more often than not during DSS. When present in a single modality, suppression normally suggests a lesion in the secondary or unimodal association area of the particular modality involved. However, if multimodal, the lesion could involve tertiary cortical or possibly subcortical pathology and/or reflect a unilateral neglect syndrome.

Cross References

► Double Simultaneous Stimulation
► Inhibition

Supramarginal Gyrus

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Definition

The supramarginal gyrus is part of the inferior parietal lobule and consists of a somatosensory-related, higher-order associational cortex.

Current Knowledge

The supramarginal gyrus is part of the inferior parietal lobule. It is typically a U-shaped gyrus, inferior to the intraparietal sulcus, at the caudal terminus of the lateral sulcus. It consists of a somatosensory-related, higher-order associational cortex. As a member of the broadest definition of Wernicke's posterior speech cortex, lesions of the supramarginal gyrus in the left cerebral hemisphere often result in difficulties with tactile aspects of language comprehension.

Cross References

- ▶ Associational Cortex
- ▶ Parietal Lobe
- ▶ Wernicke's Aphasia

Surface Agraphia

- ▶ Agraphia

Surface Alexia

- ▶ Alexia

Surrogate Partner Therapy

- ▶ Sexual Surrogate Therapy

Sustained Attention

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Synonyms

Persistence; Vigilance

Definition

Processes that enable sustained performance on tasks over extended periods of time. Sustained attention is one of the primary elements or components processes of attention. It enables the maintenance of vigilance, selective and focused attention, response persistence, and continuous effort despite changing conditions.

Historical Background

The temporal nature of attention was discussed in the writing of Williams James, and has been considered to be an important aspect of attending by psychologists throughout the twentieth century. Most people can easily relate to sustained attention as part of their everyday experience. Instructions from a teacher or parent to pay attention are essentially efforts to get the child to sustain focused attention. Formal cognitive investigation was a natural outgrowth of research directed at serial processing and factors that affect the performance of people when cognitive processing of stimuli is required. Experimental work on sustained attention picked up in the 1950s, when psychologists and engineers working on factors that influence signal detection in the military, realized that it was essential to account for performance characteristics over time (Jerison, 1970). For example, it was necessary for radar operators to remain vigilant for long periods of time as they monitored incoming aircraft and missiles, so it was important to better understand factors that affected vigilance (i.e., prolonged attending in anticipation of rare signals and events). These considerations continue to be of particular importance for researcher studying human factors in occupational settings.

Many of the early studies of sustained attention focused on the stimulus and response parameters that affected signal detection during vigilance (Baddeley & Colquhoun, 1969; Broadbent & Gregory, 1963; Davies, 1970). Yet, signal detection over long time periods is only one example of a task that requires sustained attention. Research on factors that contribute to performance variability over time was subsequently conducted for a wide variety of cognitive and behavioral conditions. Sustained attention occurs on tasks requiring intense focus and active cognitive processing as opposed to passive anticipation waiting for a rare event to occur. Clinical focus on sustained attention increased dramatically over the past 30 years, as attention deficit disorder (ADD) was identified as a development disorder causing learning disability in children, with impairments of sustained attention a central feature of the disorder.

Current Knowledge

Sustained attention is considered to be a primary element or component in most current neuropsychological models of attention (Mirsky, Anthony, Duncan, Ahearn, & Kellam, 1991, 2001; Cohen, 1993). There are strong theoretical and empirical bases for this view. While it is

possible to conceptualize selective attention at a given moment in time as occurring without the need for sustained attention, most cognitive tasks and real-world situations involve the sequential processing of information over a period of time. Furthermore, human attentional performance is never perfect. Even on the simplest of tasks, people will make occasional errors. For example, if a person sits by the phone anticipating a call, the probability is close to 100% that the ring will be detected and the phone answered, if there is no distraction. However, there is a possibility that the person might fall asleep and miss the call, particularly if the person has been waiting for a long time and has grown tired. In fact, attention must be viewed in a probabilistic manner, with the time dimension being one of the strongest determinants of accuracy. Attention is temporally distributed, and characterizing variations in attention over time is essential to its measurement.

Sustained attention is influenced by a large number of factors. To begin with, sustained attention depends on the more elementary processes of sensory selective attention, and response intention and executive control occurring in a consistent and fluid manner. It also depends on relatively stable attentional capacity, enabling an adequate intensity of focus over time. Factors that interfere with a person's ability to selectively attend either external or internal signals will likely affect sustained attention as well. Yet, it is possible to sustain attention despite experiencing difficulty with selective attention. In contrast, factors that affect response intention and executive control tend to dramatically affect sustained performance. Sustained attention depends on the ability to inhibit competing impulses and responses, and to form an intention to act, to generate a set of responses in a consistent manner, and to persist, switch, or inhibit responding in accordance with task demands. Disruption of these control processes tends to lead to a failure of sustained attention. For these reasons, patients with frontal lobe damage and disturbances affecting frontal-subcortical pathways usually have problems with sustained and focused attention (Cohen, 1993).

Kahneman (1983) proposed that capacity limitations constrain people's ability to attend. These capacity limitations affect the intensity of attentional focus that is possible from moment to moment. Attention capacity is influenced by both intrinsic processing limitations that exist for each person and also energetic factors that are more transient in nature (Cohen, 1993). Both factors affect sustained attention. Among the intrinsic capacity limitations, two of the most significant factors are processing speed and memory. Processing speed is an important determinant of capacity because it influences the amount of information that can be processed per unit of time. This principle is not unique to humans, but is a fundamental aspect of all information and communication systems. Slowing down the rate of processing directly translates into decreased efficiency, greater effort, and longer time requirements for the completion of tasks. All of these factors directly impact on sustained attention. Patients with subcortical disturbances that affect the rate of signal transmission along white matter pathways typically have significant problems in this regard. Consequently, it is common for patients with multiple sclerosis, HIV, and other diseases that affect subcortical brain systems to have problems with sustained attention (Cohen, 1993).

Memory impairments also affect attention capacity by making automaticity more difficult to achieve (Schneider and Shiffrin, 1977). Information related to the task at hand that is encoded into memory can be accessed to facilitate attention, whereas information that is not encoded must be held in short-term or working memory, making automatic access to this information more challenging. In general, tasks with high effortful demands will tend to also tax sustained attention capacity. While brain disorders that affect memory processes may have little effect on basic sensory selective attention, it is likely to have significant impact on tasks that require the maintenance of attentional focus over time.

Capacity limitations associated with energetic factors create inherent variability in attention. Among the factors that affect energetic capacity are arousal and drive. The influence of arousal on sustained attention tends to occur as an inverted U-shaped function, people's performance varies based on their level of wakefulness, their level of anxiety, and a wide range of other factors that affect arousal (Blakeslee, 1979). Natural variations in sustained attention were described in early studies of sleep and circadian influence on behavior (Loeb, 1994). However, alterations in arousal are also a common manifestation of drugs that cause sedation or stimulation, and are also a hallmark of delirium caused by metabolic disturbance (Rohrbaugh et al., 1988). Accordingly, impairments of sustained attention are essential features of disorders that affect arousal.

The other major determinant of energetic capacity is motivation and drive, and external factors that impact on these states. These factors create considerable complexity for the study and assessment of sustained attention. They vary greatly based on individual differences in experience and learning history across people, and also as a function

of inherent differences in the level of reward derived from successful performance. People's attention to food and cues for food will vary greatly depending on how hungry they are. This analogy can be easily extended to a wide range of potential stimuli. For example, the presence of cigarette smoke in a room is a powerful cue that can enhance attentional performance in a smoker, yet may be experienced as aversive and cause distraction for a nonsmoker. The incentive that exists in a particular situation will influence the likelihood that a person will sustain their attention in that situation, though the extent to which people experience reward is not consistent either. This is dramatically evident in the case of patients who are experiencing major depression. During periods of severe depression, people often experience greatly diminished motivation and drive, coexisting with impairments of concentration and sustained attention (Byrne, 1977; Cohen, Lohr, Paul, & Boland, 2001). With successful treatment, these symptoms typically improve. Sustained attention tends to be one of the cognitive functions most affected by major depression. Sustained attention and vigilance deficits are also common in schizophrenia (Nuechterlein, Garmezy, Devine, Schulz, & Tamminga, 1989). Even in completely healthy people, incentive is a major determinant of sustained attention. A soldier in the battlefield will have little difficulty sustaining their attentional focus when they are facing mortal danger.

In light of the wide range of factors that can influence sustained attention and the fact that it is affected by a large number of different brain disorders, it is readily apparent that sustained attention is not a unitary process, but rather the by-product of multiple interacting processes under the control of systems distributed across the brain. Certain brain regions, most notably the frontal cortex, basal ganglia, thalamus, and limbic system appear to play a major role in sustained attention regardless of the task. The attention systems of the parietal lobe seem to have particular relevance when tasks require selective attention and have a spatial demand. Problems with sustained attention frequently coexist with other attentional or cognitive problems in patients with neurological brain diseases. For example, patients with Alzheimer's disease exhibit impairments of sustained attention and decrements of sustained attention occur as a function of aging (Berardi, Parasuraman, & Haxby, 2005). It is also often affected following closed head injury (Parasuraman, Mutter, & Molloy, 1991).

Impairments of sustained and focused attention may present as the primary cognitive disturbance among children with ADD.

Assessment Considerations

The assessment of sustained attention depends on the careful observation and measurement of attentional performance over time. Historically, this was difficult to accomplish without the aid of computers or other instruments that would enable careful timing and acquisition of responses. Clinicians would make inferences about attentional variability based on inconsistencies in performance from one point in the evaluation to another. Comparison of performance across items of a similar level of difficulty would also point to such variability. Today, it is possible to obtain much more accurate information about sustained performance through the use of Continuous Performance Tests (CPT), or other tasks that provide information about error rates across the test period.

Two types of indices are most useful from these types of tests, measures of: (1) vigilance decrement and (2) inconsistency. These indices can be obtained from many current versions of the CPT. Vigilance decrement can be thought of as the negative slope of the curve that defines performance over an extended period of time. The general principle underlying this decrement is similar to that of a muscle that is fatiguing due to sustained use, though in reality this type of decrement is rare for sustained attention. While dramatic vigilance decrement may occur if a patient falls asleep or loses interest in performing the tasks, more commonly, problems with sustained attention are manifest as increased variability of performance over time.

Future Directions

Despite many advances in methodology, most neuropsychological batteries are not geared toward fully characterizing the temporal dynamics of attending. Information regarding sustained attention is usually derived from behavioral observations or from a single test like the CPT. Until recently, most widely used CPT tests provided only limited information about attention performance over time. This is changing as some recent versions of this paradigm now yield indices of vigilance decrement and inconsistency. Going forward, the assessment of attentional inconsistency is likely to become a more routine part of most neuropsychological tasks, particularly as computerized test administration is extended to a wider range of tasks and cognitive domains. In the future, it is likely that more detailed information about variations in performance over the course of the assessment will become available to clinicians and will aid in the

interpretation of whether cognitive impairments are stable or fluctuating.

There is also a need for continued research on the neural substrates of sustained attention. Functional imaging studies have been conducted over the past few years demonstrating the role of specific brain regions in sustained attention (Hester, D'Esposito, Cole, & Garavan, 2007). The recruitment of larger areas of the brain tends to occur when tasks require focused attention and working memory over sustained time periods (Kubler, Murphy, Kaufman, Stein, & Garavan, 2003). These studies support the conclusion that sustained attention occurs as a function of a complex set of different processes controlled by the interaction of multiple brain systems (Cohen, 1993; Lawrence, Ross, Hoffmann, Garavan, & Stein, 2003). However, the nature of these interactions has not yet been well characterized, and the temporal dynamics of the functional brain response are still not well understood. The relationship between the recruitment of secondary brain regions during sustained task performance and the experiences of effort and fatigue require additional study.

Cross References

- ▶ Attention
- ▶ Automaticity
- ▶ Capacity
- ▶ Continuous Performance Test
- ▶ Effort
- ▶ Fatigue
- ▶ Processing Speed

References and Readings

Baddeley, A. D., & Colquhoun, W. P. (1969). Signal probability and vigilance: A reappraisal of the 'signal-rate' effect. *British Journal of Psychology, 60*, 169–178.

Berardi, A. M., Parasuraman, R., & Haxby, J. V. (2005). Sustained attention in mild Alzheimer's disease. *Developmental Neuropsychology, 28*, 507–537.

Blakeslee, P. (1979). Attention and vigilance: Performance and skin conductance response changes. *Psychophysiology, 16*, 413–419.

Broadbent, D. E., & Gregory, M. (1963). Vigilance considered as a statistical decision. *British Journal of Psychology, 54*, 309–323.

Buchsbaum, M. S., Siegel, B. V., Wu, J. C., Hazlett, E., et al. (1992). Brief report: Attention performance in autism and regional brain metabolic rate assessed by positron emission tomography. *Journal of Autism and Developmental Disorders, 22*, 115–125.

Byrne, D. G. (1977). Affect and vigilance performance in depressive illness. *Journal of Psychiatric Research, 13*, 185–191.

Cohen, R. A. (1993). *Neuropsychology of attention.* New York: Plenum.

Cohen, R., Lohr, I., Paul, R., & Boland, R. (2001). Impairments of attention and effort among patients with major affective disorders. *Journal of Neuropsychiatry and Clinical Neurosciences, 13*, 385–395.

Corcoran, D. W. J., Mullin, J., Rainey, M. T., & Frith, G. (1977). *The effects of raised signal and noise amplitude during the course of vigilance tasks.* New York: Academic.

Davies, D. R., & Parasuraman, R. (1982). *The psychology of vigilance.* London, New York: Academic.

Garavan, H., Ross, T. J., Murphy, K., Roche, R. A., & Stein, E. A. (2002). Dissociable executive functions in the dynamic control of behavior: Inhibition, error detection, and correction. *Neuroimage, 17*, 1820–1829.

Hester, R., D'Esposito, M., Cole, M. W., & Garavan, H. (2007). Neural mechanisms for response selection: Comparing selection of responses and items from working memory. *Neuroimage, 34*, 446–454.

Hitchcock, E. M., Warm, J. S., Matthews, G., Dember, W. N., Shear, P. K., Tripp, L. D., et al. (2003). Automation cueing modulates cerebral blood flow and vigilance in a simulated air traffic control task. *Theoretical Issues in Ergonomics Science, 4*, 89–112.

Jerison, H. J. (1970). Vigilance: A paradigm and some physiological speculations. *Acta Psychologica, Amsterdam, 33*, 367–380.

Kahneman, D. (1973). *Attention and effort.* Englewood Cliffs, NJ: Prentice-Hall.

Kubler, A., Murphy, K., Kaufman, J., Stein, E. A., & Garavan, H. (2003). Co-ordination within and between verbal and visuospatial working memory: Network modulation and anterior frontal recruitment. *Neuroimage, 20*, 1298–1308.

Lawrence, N. S., Ross, T. J., Hoffmann, R., Garavan, H., & Stein, E. A. (2003). Multiple neuronal networks mediate sustained attention. *Journal of Cognitive Neuroscience, 15*, 1028–1038.

Loeb, R. G. (1994). Monitor surveillance and vigilance of anesthesia residents. *Anesthesiology, 80*, 527–533.

Mackworth, J. F. (1969). *Vigilance and habituation: A neuropsychological approach.* Harmondsworth: Penguin.

Mirsky, A. F., Anthony, B. J., Duncan, C. C., Ahearn, M. B., & Kellam, S. G. (1991). Analysis of the elements of attention: A neuropsychological approach. *Neuropsychology Review, 2*, 109–145.

Nuechterlein, K. H., Garmezy, N., Devine, V. T., Schulz, S. C., & Tamminga, C. A. (1989). *Sustained, focused attention under high processing loads: Relevance to vulnerability to schizophrenia. In Schizophrenia: Scientific progress* (pp. 95–102). New York: Oxford University Press.

Parasuraman, R., Mutter, S. A., & Molloy, R. (1991). Sustained attention following mild closed-head injury. *Journal of Clinical and Experimental Neuropsychology, 13*, 789–811.

Rohrbaugh, J. W., Stapleton, J. M., Parasuraman, R., Frowein, H. W., et al. (1988). Alcohol intoxication reduces visual sustained attention. *Psychopharmacology, 96*, 442–446.

Schneider, W., & Shiffrin, R. M. (1977) Controlled and automatic human information processing: I. Detection, search, and attention. *Psychological Review, 84*(1), 1–66.

SWLS

- ▶ Satisfaction with Life Scale

Sylvian Fissure

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Synonyms

Lateral Sulcus

Definition

Also known as the lateral sulcus, the sylvian fissure divides the frontal and parietal lobes from the temporal lobe. It is present in both hemispheres and is the most prominent of the cerebral fissures.

Cross References

► Frontal Lobes

References and Readings

Williams, S. M. (June 2, 2008). *Lateral Fissure.* Retrieved June 2, 2008, from www.sylvius.com

Sylvian Seizures

► Benign Rolandic Epilepsy

Symbol Digit Modalities Test

► Digit Symbol Substitution Test

Symbol Search

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Synonyms

Symbol search subtest

Description

Symbol Search is a subtest of the Wechsler Adult Intelligence Scale (WAIS), the Wechsler Intelligence Scale for Children (WISC), and the Wechsler Preschool and Primary Scale of Intelligence (WPPSI). The Symbol Search subtest is designed to assess information processing speed and visual perception. It is one of three subtests that contribute to the Processing Speed Indices derived from the Wechsler intelligence scales. This subtest is similar to other measures of processing speed (e.g., ► Symbol Digit Modalities Test, Symbol-Digit Coding, Digit-Symbol Coding) in that high scores require rapid and accurate processing of nonverbal visual information (i.e., symbols without *a priori* meaning). During Symbol Search, the examinee is asked to mark either the yes or no checkbox with a pencil in response to as many items as possible within 2 min. Each item is presented sequentially as a row. In each row, two symbols are presented to the left of a set of five symbols. The correct answer is yes if the set of five symbols includes either of the two symbols on the left. Matches occur at a rate of 50%, and total correct positive and negative responses are tallied for the raw score.

Historical Background

Symbol Search first appeared as a supplementary subscale of the WISC-III in 1991. It was later included in the WAIS-III in 1997 and the WPPSI-III in (2002). It remains a subtest in the current versions of these scales. The addition of Symbol Search to these scales was a response to factor analyses that provided empirical support for a third Freedom from Distractibility factor, in addition to the traditional Verbal and Performance components. It was expected that the Symbol Search subtest would bolster the Freedom from Distractibility factor. Ultimately four factors were identified and formally incorporated into the scoring process as Verbal

Comprehension, Perceptual Organization, Working Memory, and Processing Speed Indices. Symbol Search is now one of two core subtests that contribute to the Processing Speed Index in the WISC-IV (2003) and WAIS-IV (2008). Digit Symbol Coding, simply named Coding in the fourth editions, is the other core subtest. A supplementary Cancellation subtest may be used in addition to calculate the Processing Speed Index. The addition of Symbol Search and the Processing Speed Index to the Wechsler intelligence scales was empirically driven by factor analyses and an unexpected four-factor finding, rather than by a particular theoretical approach. Despite its status as a supplementary WAIS subtest over the past decade, Symbol Search was commonly administered in neuropsychological assessments and has demonstrated utility clinical research.

Psychometric Data

The Symbol Search subtest has excellent normative data and demonstrates excellent reliability and construct validity. These are well documented in the Wechsler technical manuals. Strong correlations have been reported between Symbol Search, Coding, and Cancellation, and also other measures of information processing speed, such as the Symbol Digit Modalities Test. Symbol Search has also demonstrated sensitivity to discriminate clinical populations in which diminished cognitive processing speed is expected (e.g., ► traumatic brain injury).

Clinical Uses

As a core subtest of the fourth editions of the Wechsler intelligence scales, Symbol Search contributes to Full-Scale IQ and the Processing Speed Index. This subtest has also been used alone as a measure of processing speed and visual perception in clinical and research settings. Comprehensive normative data and well-documented psychometric properties are major advantages when considering the Symbol Search for use in clinical settings.

Cross References

► Information Processing Speed
► Wechsler Adult Intelligence Scale (All Versions)
► Wechsler Intelligence Scale for Children
► Wechsler Preschool and Primary Scale of Intelligence (WPPSI)

References and Readings

Wechsler, D. (2002). *WPPSI-III technical and interpretive manual.* San Antonio, TX: The Psychological Corporation.
Wechsler, D. (2003). *WISC-IV technical and interpretive manual.* San Antonio, TX: The Psychological Corporation.
Wechsler, D. (2008). *WAIS-IV technical and interpretive manual.* San Antonio, TX: The Psychological Corporation.

Symbol Search Subtest

► Symbol Search

Symmetril (Amantadine)

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Synonyms

Amantadine

Definition

Amantadine is indicated in the treatment of idiopathic Parkinson's disease, postencephalitic parkinsonism, symptomatic parkinsonism, and drug-induced extrapyramidal reactions. Of note, it is less effective than Levodopa in the treatment of Parkinson's disease. Amantadine is also used as an antiviral indicated for the prophylaxis and treatment of Influenza A.

Current Knowledge

Mechanism of Action

Amantadine is an anti-Parkinson and an antiviral medication. The mechanism of action in the treatment of Parkinson's disease and drug-induced extrapyramidal reactions is not known. The drug may affect the dopamine neurons.

Contraindications

Deaths have been reported from overdose of Amantadine. Overdose may also cause arrhythmia, tachycardia, hypertension, and cardiac, respiratory, renal, or central nervous system toxicity.

While the mechanism of action is not clear, suicide attempts and ideation have been reported in individuals taking Amantadine as an antiviral. These individuals did not have a previous psychiatric history.

Patients with a history of seizures should be observed for increased seizure activity.

Patients with a history of CHF or peripheral edema should be observed for CHF. It should not be given to patients with untreated angle-closure glaucoma. Amantadine is not recommended for nursing mothers; its use with pregnant women has not yet been adequately tested and hence should not be used.

Adverse Events

Abrupt discontinuation of the medication in patients with Parkinson's disease may cause a parkinsonian crises; it may also precipitate delirium, agitation, delusions, hallucinations, paranoia, anxiety, slurred speech, and depression.

Some cases of neuroleptic malignant syndrome (NMS) have been observed in association with dose reduction or discontinuation.

Because Amantadine is excreted by the kidneys, the dose should be reduced in the elderly and in those with renal compromise.

Side Effects

The most frequently reported side effects include nausea, dizziness, and insomnia. Less frequently reported effects include depression, anxiety, irritability, hallucinations, confusion, anorexia, constipation, ataxia, peripheral edema, agitation, somnolence, diarrhea, and fatigue.

Drug Interactions

Central nervous system stimulants require careful observation when taken with Amantadine. Coadministration of quinine or quinidine may affect renal clearance of Amantadine.

Pharmocodynamics and Kinetics

Its use in the treatment of Parkinson's disease and drug-induced extrapyramidal reactions is not clear. It may have a direct effect on the Dopamine neurons. It also seems to have anticholinergic-like side effects such as dry mouth, urinary retention, and constipation. Amantadine is well absorbed orally. It is primarily excreted in the urine.

The time to peak effect with oral administration of Amantadine was approximately 3 h. The onset of action is usually within 48 h. The half-life averaged 16 h. The renal clearance of Amantadine is significantly reduced in adults with renal insufficiency and with the elderly.

Dosage

The usual dose of Amantadine for individuals with Parkinson's disease and for drug-induced extrapyramidal reactions is 100 mg twice a day. The initial dose for patients who are receiving other anti-Parkinson's medications is 100 mg daily; the dose may be increased after 1 week to twice daily. For individuals with drug-induced extrapyramidal reactions who do not respond to 200 mg daily, the dose may increase up to 300 mg daily in divided doses.

Future Directions

Clinical studies are currently funded in collaboration with NIH include a randomized controlled study looking at Amantadine for patients with severe disorders of consciousness; several other studies are looking at the treatment of irritability and aggression in individuals with traumatic brain injury and behaviors associated dementia with Amantadine. Studies are also ongoing related to the effectiveness of Amantadine in the treatment of dyskinesias and Parkinson's disease.

Cross References

- ▶ Parkinson's Disease
- ▶ Pharmacodynamics
- ▶ Pharmacokinetics
- ▶ Psychopharmacology

References and Readings

Arciniegas, D., Frey, K., Alan Anderson, C., Brousseau, K., & Harris, S. (2004). Amantadine for neurobehavioural deficits following delayed post-hypoxic encephalopathy. *Brain Injury, 18*(12), 1309–1318.

Escher, T., Call, S., Blaha, C., & Mittleman, G. (2006). Behavioral effects of aminoadamantane class NMDA receptor antagonists on schedule-induced alcohol and self-administration of water in mice. *Psychopharmacology, 187*(4), 424–434.

Hughes, S., Colantonio, A., Santaguida, P., & Paton, T. (2005). Amantadine to enhance readiness for rehabilitation following severe traumatic brain injury. *Brain Injury, 19*(14), 1197–1206.

Schneider, W., Drew-Cates, J., Wong, T., & Dombovy, M. (1999). Cognitive and behavioural efficacy of amantadine in acute traumatic brain injury: An initial double-blind placebo-controlled study. *Brain Injury, 13*(11), 863–872.

Williams, S. (2007). Amantadine treatment following traumatic brain injury in children. *Brain Injury, 21*(9), 885–889.

Sympathomimetic Amines/Drugs/Agents/Compounds

► Catecholamines

Symptom

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Synonyms

Indicator

Definition

Symptoms represent self-reported evidence of a disease, disorder, or syndrome (Webster's New Explorer Medical Dictionary, 2006). Symptoms can reflect abnormal functioning in a variety of physical, behavioral, social, or emotional domains that may be associated with organic brain syndromes or psychiatric disorders. Professionals use the occurrence of a specific grouping of symptoms to eliminate certain diagnoses and to more closely consider others. The grouping of symptoms into a symptom complex is especially important since there may be considerable overlap of individual symptoms across diagnoses. For example, the physical symptom that a patient subjectively describes as a "headache" may be a characteristic of numerous diagnoses. Thus, the context in which this symptom occurs, in terms of the presence or absence of other diagnostic indicators, needs to be considered to formulate a diagnosis.

References and Readings

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

Webster's New Explorer Medical Dictionary (New ed.). (2006). Springfield, MA: Merriam-Webster.

Symptom Checklist-90-Revised

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Synonyms

SCL-90-R

Description

The Symptom Checklist-90-Revised (SCL-90-R) (1994) is a multidimensional self-report measure, assessing the severity of current psychological symptoms and distress. It assesses nine symptom dimensions: somatization, obsessive–compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism. It also includes three global indices of psychological distress: Global Severity Index (number of symptoms endorsed and intensity of distress), Positive Symptom Distress Index (average level of distress for those items that were endorsed; exaggerating or attenuating response style), and Positive Symptoms Total (total symptoms endorsed/breadth of distress). Examinees rate each of 90 items on a five-point scale from 0 (*Not at All*) to 4 (*Extremely*) specifying how much each has bothered them during the past 7 days.

The SCL-90-R can be administered either in a paper-and-pencil or a computerized format. Instructions are

provided in 2–5 min, and the measure can typically be completed in 12–15 min. The SCL-90-R has been normed for four separate groups: adult nonpatients, psychiatric inpatients, psychiatric outpatients, and adolescent nonpatients (ages 13–18). Examinees must be able to read at a sixth-grade level and should not have significant cognitive impairment (e.g., mental retardation, and dementia). Raw scores are converted to T-scores and percentiles, and separate T-score transformations are made for males and females in each normative group. Profiles can be interpreted on three levels: global distress scores, elevation in particular symptom dimensions, and at the level of individual symptoms. As a self-report measure, the SCL-90-R provides a subjective report of current distress, and can be administered repeatedly to track changes in symptoms over time. Because it can be administered and scored by a technician, it also makes efficient use of clinician time.

Historical Background

Shortly after Adolph Meyer of Johns Hopkins University developed the first clinician-observer psychiatric rating scale at the turn of the twentieth century, Robert Woodworth introduced a self-rating scale of psychiatric symptoms. Such measures were of great utility, as they allowed assessment of symptomatology that may not be directly observable by clinicians (e.g., subjective distress) and also allowed more efficient assessment of psychiatric symptoms, particularly when access to trained clinicians may be limited, as was the case when the scales were created during World War I.

Given these strengths, self-rating measures began to develop rapidly over the following decades. The Hopkins Symptom Checklist (HSCL; Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974), the precursor of the Symptom Checklist, emanated from this tradition. The HSCL was developed as a research instrument providing assessment of five psychiatric symptom dimensions (somatization, obsessive–compulsive, interpersonal sensitivity, depression, and anxiety). Its clinical usefulness was limited, however, for multiple reasons: the five symptom domains provided only a limited breadth in the assessment of psychopathology and distress, many individual test items loaded on multiple factors, there was no analogous clinician rating scale, and it was not normed for use in individual respondents.

In the early 1970s, Derogatis and colleagues set out to design a new measure, the Symptom Checklist-90 (SCL-90) to address these limitations (Derogatis, Lipman, & Covi, 1973). The SCL-90 retained the five symptom domains of the HSCL and added 45 additional individual items allowing assessment of four new symptom dimensions (hostility, phobic anxiety, paranoid ideation, and psychoticism) that were theoretically and empirically derived. The rating for each item was expanded to a five-point scale allowing additional scope of symptom sensitivity. Further evaluation of the instrument indicated that items from the anxiety and obsesssive–compulsive dimensions were psychometrically flawed. These items were subsequently replaced, the test instructions were revised for clarity, the three global distress scales were developed, and seven "configural" items were added to assist with classification of cases, leading to the final version of the SCL, the SCL-90-R. In describing the development of these measures, the authors clearly state that the SCL-90 is considered to be a prototype and is not valid for clinical use (Derogatis, 1994).

Psychometric Data

Internal consistency reliability for the nine symptom dimensions using within-form correlations between items ranged from 0.77 (psychoticism) to 0.90 (depression) in one study (Derogatis, Rickels, & Rock, 1976). In another, coefficients ranged from 0.79 (paranoid Ideation) to 0.90 (depression) (Horowitz, Rosenbert, Baer, Ureno, & Villasenor, 1988). Test–retest reliability coefficients ranged from 0.80 to 0.90 over a 1-week period (Derogatis et al., 1976) and 0.68 to 0.83 over a 10-week period (Horowitz et al., 1988). These are considered to be more than satisfactory, as the instrument is designed to measure expected fluctuations in symptoms over time and in response to intervention. Factor analysis has indicated adequate internal consistency on most symptom dimensions, though there was some overlap between anxiety and phobic anxiety dimensions and some inconsistency in items included in the psychoticism dimension. The manual reports a variety of studies demonstrating convergent-discriminant validity with correlations to self-report instruments including the Center for Epidemiological Study-Depression Scale (CES-D) and Hamilton Rating Scale (Weissman, Sholomskas, Pottenger, Prusoff, & Locke, 1977) Social Adjustment Scale-Self-Report (SAS-SR) (Weissman, Prusoff, Thompson, Harding, & Meyers, 1978), and General Health Questionnaire (GHQ-28) (Koeter, 1992; Wiznitzer et al., 1992), and the clinician-administered structured interview the Present State Examination (PSE) (Peveler & Fairburn, 1990).

Clinical Uses

The SCL-90-R is designed as a self-report measure of current psychological distress and symptom severity across a broad continuum from subclinical to severe psychiatric illness. As a sensitive measure of distress, it is useful in evaluating changes in symptomatology over time, such as in characterizing the clinical course of psychiatric distress in a variety of mental health and medical disorders. More notably, given its sensitivity to distress and its amenability to repeated administration, the SCL-90-R has been used as an outcome measure in hundreds of published studies assessing the efficacy of psychotherapeutic and pharmacological interventions for DSM-IV Axis I disorders.

With its multidimensional structure, the SCL-90-R has also been utilized as an actuarial tool to characterize more complex psychiatric disorder (e.g., comorbid substance abuse and mood disorder) (Liskow, Powell, Nickel, & Penick, 1991a), to predict risk for negative outcome like suicidality (e.g., Bulick, Carpenter, Kupfer, & Frank, 1990; Coryell, 1988; Swedo, Rettew, Kuppenheimer, Lum, Dolan, & Goldberger, 1991), and to predict treatment outcome based on symptom profile (Liskow, Powell, Nickel, & Penick, 1991b). In neuropsychological populations, the SCL-90-R has been used for a number of purposes, including to document the efficacy of psychotherapy in individuals with acquired brain injury (Bradbury, Christensen, Lau, Ruttan, Arundine, & Green, 2008), to characterize psychiatric symptoms that may be early markers of Huntington's disease (Duff, Paulsen, Beglinger, Langbehn, Stout, & the Predict-HD Investigators of the Huntington Study Group, 2007), to identify psychiatric symptoms that differentiate individuals with severe brain injury from those sustaining multiple trauma (Frenisy et al., 2006), and to evaluate the relationship between elevations on the obsessive–compulsive scale and cognitive impairment in individuals with malignant brain tumors (Kaplan & Miner, 1998).

Despite its widespread use, several limitations of the SCL-90-R have been documented. Some studies indicate that it may provide only limited incremental validity when data from lengthier measures like the MMPI-2 are available (Simonds, Handel, & Archer, 2008). Evidence also suggests that caution is warranted in considering the SCL-90-R as a diagnostic instrument (Woody, Steketee, & Chambless, 1995). While the manual indicates that the nine symptom dimensions are internally consistent, other authors have challenged this notion. For example, the psychoticism dimension was designed to assess positive symptoms such as hallucinations, but also assesses negative symptoms, including loneliness and guilt, that correlate strongly with symptoms of depression. It has been further demonstrated that a single factor with greatest loading of symptoms of anxiety and depression accounts for most of the variance (Simonds et al., 2008) suggesting reduced utility of each of the symptoms dimensions as primary diagnostic measures. Interpretation of single symptom dimensions in making diagnostic determinations may be particularly problematic. For example, the obsessive–compulsive dimension demonstrated poor divergent validity in differentiating clients with obsessive–compulsive disorder from those with panic disorder with agoraphobia (Woody et al., 1995).

Of even greater importance for neuropsychologists, many obsessive–compulsive items load on what O'Donnell, DeSoto, and Reynolds (1984) called a "cognitive deficit" scale. This is not surprising as only 2 of the 10 items on the scale relate to the classic symptoms of obsessive–compulsive disorder, while many of the rest concern cognitive problems such as difficulty concentrating and impaired memory. Woessner and Caplan (1995, 1996) reported that 14 and 19 SCL-90-R items, respectively, were identified by experts as being "usual consequences" of brain injury or stroke. Similarly, patients with malignant brain tumors had elevations on obsessive–compulsive, somatization, and psychoticism subscales, despite not meeting criteria for any psychiatric diagnosis (Kaplan, Miner, Mervis, Newton, McGregor, & Goodman, 1998). These authors called attention to the need for caution when interpreting endorsement of these items by persons with these conditions, as their diagnostic significance may differ from that for physically healthy people on whom the measure was normed.

Despite these limitations, the SCL-90-R's relatively brief administration time and demonstrated utility with a range of medical and psychiatric populations renders it an appealing option for rapid characterization of patient symptom profiles in both research and clinical settings.

Cross References

- ▶ Beck Anxiety Inventory
- ▶ Beck Depression Inventory
- ▶ Brief Symptom Inventory
- ▶ Center for Epidemiologic Studies-depression (CES-d)
- ▶ Hamilton Rating Scale of Depression
- ▶ Minnesota Multiphasic Personality Inventory
- ▶ Self-Report Measures
- ▶ Zung Self-Rating Depression Scale

References and Readings

Bradbury, C. L., Christensen, B. K., Lau, M. A., Ruttan, L. A., Arundine, A. L., & Green, R. E. (2008). The efficacy of cognitive behavior therapy in the treatment of emotional distress after acquired brain injury. *Archives of Physical Medicine & Rehabilitation, 89*(Suppl.12), S61–S68.

Bulik, C. M., Carpenter, L. L., Kupfer, D. J., & Frank, E. (1990). Features associated with suicide attempts in recurrent major depression. *Journal of Affective Disorders, 18*, 29–37.

Coryell, W. (1988). Mortality of anxiety disorders. In R. Noyes, M. Roth, & G. D. Buttows (Eds.), *Handbook of anxiety. Vol. 2: Classification, biological factors and associated disturbances.* New York: Elsevier.

Derogatis, L. R. (1994). *SCL-90-R: Administration, scoring and procedures manual* (3rd ed.). Minneapolis, MN: NCS Pearson.

Derogatis, L. R., Lipman, R. S., & Covi, L. (1973). SCL-90: An outpatient psychiatric rating scale-preliminary report. *Psychopharmocology Bulletin, 9*(1), 13–28.

Derogatis, L. R., Lipman, R. S., Rickels, K., Uhlenhuth, E. H., & Covi, L. (1974). The Hopkins Symptoms Checklist (HSCL): A self-report symptom inventory. *Behavioral Science, 19*, 1–15.

Derogatis, L. R., Rickels, K., & Rock, A. (1976). The SCL-90 and the MMPI: A step in the validation of a new self-report scale. *British Journal of Psychiatry, 128*, 280–289.

Duff, K., Paulsen, J. S., Beglinger, L. J., Langbehn, D. R., Stout, J. C., & the Predict-HD Investigators of the Huntington Study Group (2007). Psychiatric symptoms in Huntington's disease before diagnosis: The Predict-HD Study. *Biological Psychiatry, 62*, 1341–1346.

Frenisy, M., Benony, H., Chahraoui, K., Minot, D., d'Athis, P., Pinoit, J., et al. (2006). Brain injured patients versus multiple trauma patients: Some neurobehavioral and psychopathological aspects. *The Journal of Trauma, 60*, 1018–1026.

Horowitz, L. M., Rosenbert, S. E., Baer, B. A., Ureno, G., & Villasenor, V. S. (1988). Inventory of interpersonal problems: Psychometric properties and clinical applications. *Journal of Consulting and Clinical Psychology, 56*(6), 885–892.

Kaplan, C. P., & Miner, M. E., (1998). Does the SCL-90-R Obsessive Compulsive dimension identify cognitive impairments? *Journal of Head Trauma Rehabilitation, 13*(3), 94–101.

Kaplan, C. P., Miner, M. E., Mervis, I. J., Newton, H., McGregor, J. M., & Goodman, J. H. (1998). Interpretive risks: The use of the Hopkins Symptom Checklist 90-Revised (SCL 90-R) with brain tumour patients. *Brain Injury, 12*(3), 199–205.

Koeter, M. W. (1992). Validity of the GHQ and SCL anxiety and depression scales: A comparative study. *Journal of Affective Disorders, 24*(4), 271–279.

Liskow, B., Powell, B. J., Nickel, E., & Penick, E. (1991a). Antisocial alcoholics: Are there clinically significant diagnostic subtypes? *Journal of Studies on Alcohol, 52*(1), 62–69.

Liskow, B., Powell, B. J., Nickel, E., & Penick, E. (1991b). Diagnostic subgroups of antisocial alcoholics: Outcome at one year. *Comprehensive Psychiatry, 31*(6), 549–556.

O'Donnelll, W. E., DeSoto, C. B., & Reynolds, D. M. (1984). A cognitive deficit subscale of the SCL-90-R. *Journal of Clinical Psychology, 40*, 241–246.

Peveler, R. C., & Fairburn, C. G. (1990). Measurement of neurotic symptoms by self-report questionnaire: Validity of the SCL-90-R. *Psychological Medicine, 20*(4), 873–879.

Swedo, S. E., Rettew, D. C., Kuppenheimer, M., Lum, D., Dolan, S., & Goldberger, E. (1991). Can adolescent suicide attempters be distinguished from at-risk adolescents? *Pediatrics, 88*(3), 620–629.

Simonds, E. C., Handel, R. W., & Archer, R. P. (2008). Incremental validity of the Minnesota Multiphasic Personality Inventory-2 and Symptom Checklist-90-Revised with mental health inpatients. *Assessment, 78*(1), 78–86.

Weissman, M. M., Prusoff, B. A., Thompson, W. D., Harding, P. S., & Meyers, J. K. (1978). Social adjustment by self-report in a community sample and in psychiatric outpatients. *Journal of Nervous and Mental Disease, 166*(5), 317–326.

Weissman, M. M., Sholomskas, D., Pottenger, M., Prusoff, B. A., & Locke, B. Z. (1977). Assessing depressive symptoms in five psychiatric populations: a validation study. *American Journal of Epidemiology, 106*(3), 203–214.

Wiznitzer, M., Verhulst, F. C., Van den Brink, W., Koeter, M., van der Ende, J., Giel, R., et al. (1992). Detecting psychopathology in young adults: The Young Adult Self-Report, the General Health Questionnaire and the Symptom Checklist as screening instruments. *Acta Psychiatrica Scandinavica, 86*(1), 32–37.

Woessner, R., & Caplan, B. (1995). Affective disorders following mild to moderate traumatic brain injury: Interpretive hazards of the SCL-90-R. *Journal of Head Trauma Rehabilitation, 10*, 79–89.

Woessner, R., & Caplan, B. (1996). Emotional distress following stroke: Does the SCL-90-R diagnose or mislead? *Assessment, 3*, 291–305.

Woody, S. R., Steketee, G., & Chambless, D. L. (1995). The usefulness of the obsessive compulsive scale of the Symptom Checklist-90-Revised. *Behavior Research and Therapy, 33*(5), 607–611.

Symptom Exaggeration

► Fake Bad Scale

Symptom Validity Assessment

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Synonyms

Effort testing

Description

Conceptually, a symptom validity test can be considered an effort test. However, a symptom validity test can also assess the "validity" of a symptom, such as loss of tactile sensation. The symptom validity paradigm was

popularized by Pankratz, Fausti, and Peed (1975) and utilized a forced-choice methodology. The symptom validity paradigm, as it applies to cognition, is based on the notion that, if an individual has absolutely no memory for certain information, the lower limits of correct performance should be at chance levels. That is, if there are two alternatives in a forced-choice procedure, the probability of responding correctly is approximately 50% (within a given confidence interval). Thus, patients' performances can be compared to the probability of obtaining a given score by chance (i.e., random responding), via a binomial distribution. For example, if 50 trials of a forced-choice test are administered, the probable range of random responding is 20–30. Scores in this range are consistent with "chance" performance and scores below this range provide compelling evidence that the person is choosing the wrong answer. This paradigm can be applied to cognition (e.g., memory) or sensory functioning (e.g., tactile sensation, visual loss, or hearing loss).

Symptom validity assessment, a terminology used in the National Academy of Neuropsychology (NAN) position paper (Bush et al., 2005), refers to *all methods and procedures* that the practitioner can draw upon to make inferences regarding poor effort during testing and exaggeration of symptoms or problems during the interview or on psychological tests. As such, symptom validity assessment is much broader than the traditional symptom validity test (which historically was based on forced-choice testing). There are three general approaches to using tests within the context of a neuropsychological evaluation for the purpose of identifying exaggeration or poor effort. The first approach is to use tests that have validity indices built-in, such as self-report inventories like the MMPI-2 and the Personality Assessment Inventory. The second approach is to use existing tests of cognitive ability and to identify unusual cutoff scores or performance patterns on these tests. These are sometimes referred to as embedded markers of poor effort. The third approach is to use specialized tests designed specifically for detecting poor effort, such as the Test of Memory Malingering or the Victoria Symptom Validity Test.

Historical Background

The symptom validity paradigm (Binder & Pankratz, 1987; Pankratz, 1983; Pankratz, Binder, & Wilcox, 1987; Pankratz et al., 1975) developed gradually out of some clever forced-choice procedures used with patients with hysterical blindness (Brady & Lind, 1961; Grosz & Zimmerman, 1965; Theodor & Mandelcorn, 1973). The symptom validity paradigm was applied to memory with the Hiscock and Hiscock (1989) procedure, the Portland Digit Recognition Test, Computerized Assessment of Response Bias, 21 Item Test, Word Memory Test, Victoria Symptom Validity Test, and Test of Memory Malingering.

Psychometric Data

Symptom validity tests, such as forced-choice tests, are typically not examined psychometrically using approaches from classical test theory. For example, it is very uncommon to examine the internal consistency or test–retest reliability of these measures. Instead, these tests are typically examined as state or situational markers of poor effort. As such, the vast majority of psychometric research pertains to the validity of specific cut-off scores for identifying poor effort. In a typical study, cut-off scores are selected and applied to one or more clinical groups that are suspected of poor effort versus groups that are not suspected of poor effort (Arnold et al., 2005; Babikian, Boone, Lu, & Arnold, 2006; Greve & Bianchini, 2006; Greve, Bianchini, & Doane, 2006).

Forced-choice digit recognition procedures (e.g., ▶ Portland Digit Recognition Test, ▶ Computerized Assessment of Response Bias, and ▶ the Victoria Symptom Validity Test) and other simple effort tests (e.g., Word Memory Test, Medical Symptom Validity Test, Validity Indicator Profile, b-Test, and Test of Memory Malingering), have been shown to be relatively insensitive to the effects of brain injury. In general, there are two ways to interpret these tests: (a) comparing scores to chance levels via the binomial distribution, and/or (b) using empirically derived basal cut-off scores. Those clinicians who rely on chance performance levels fail to detect a substantial percentage of individuals who are not trying their best. The sensitivity, specificity, and predictive power of cut-off scores for suspecting poor effort are available in test manuals and in published articles.

Clinical Uses

Symptom validity assessment should be a pro forma part of an assessment plan. Clinicians should not wait for obvious evidence of poor effort or exaggeration before giving specialized tests. It is best to use a combination of approaches, including specialized tests and examination of performance patterns on traditional tests (i.e., embedded markers). Practitioners are encouraged to use

well-validated specialized tests (e.g., ► Word Memory Test, ► Victoria Symptom Validity Test, or ► TOMM). "Simple" specialized tests (e.g., TOMM) are given toward the beginning of the evaluation, not the middle or end. It is best to intersperse validity indicators throughout the evaluation. These can be specific specialized tests or general ability tests for which certain "abnormal performance patterns" or embedded markers may be associated with poor effort.

Symptom validity testing is a standard practice in forensic neuropsychology. This is because: (a) poor effort during testing is common (Larrabee, 2003; Mittenberg, Patton, Canyock, & Condit, 2002), (b) poor effort has a very large adverse effect on neuropsychological test results (Vickery, Berry, Inman, Harris, & Orey, 2001), (c) there are well-validated tests for detecting poor effort that has low false positive rates, and (d) in forensic cases, practitioners pay special attention to issues relating to causation and try to rule out factors that might lead to incorrect inferences or interpretations (Iverson, 2008). Clinicians should be encouraged to conceptualize poor effort, exaggeration, and malingering not in simple dichotomous terms, but in continuous terms, through probabilistic considerations. Practitioners need to identify and explain test scores that simply do not make biological or psychometric sense. If the examinee demonstrates clear evidence of poor effort on any test within the evaluation, the entire set of test results is questionable. In that situation, one cannot assume that even broadly normal test scores represent the person's true ability. Those scores might also be diminished due to variable effort. Accordingly, the more conservative conclusion would be that the obtained scores represent the examinee's minimum *overall* performance at the time of the evaluation. Practitioners should avoid trying to use clinical judgment (i.e., make "educated" guesses) to determine which test performances are valid, questionable, or biased. In general, psychologists should avoid overstating symptom validity test results, in either direction ("excellent" effort or poor effort). Rather, psychologists should use phraseology that is clear, objective, reasoned, and unambiguous.

Cross References

► Malingering
► Portland Digit Recognition Test
► Rey 15 Item Test
► Test of Memory Malingering
► Word Memory Test

References and Readings

Arnold, G., Boone, K. B., Lu, P., Dean, A., Wen, J., Nitch, S., et al. (2005). Sensitivity and specificity of finger tapping test scores for the detection of suspect effort. *The Clinical Neuropsychologist, 19*(1), 105–120.

Babikian, T., Boone, K. B., Lu, P., & Arnold, G. (2006). Sensitivity and specificity of various digit span scores in the detection of suspect effort. *The Clinical Neuropsychologist, 20*(1), 145–159.

Binder, L. M., & Pankratz, L. (1987). Neuropsychological evidence of a factitious memory complaint. *Journal of Clinical and Experimental Neuropsychology, 9*(2), 167–171.

Brady, J. P., & Lind, D. L. (1961). Experimental analysis of hysterical blindness: Operant conditioning techniques. *Archives of General Psychiatry, 4*, 331–339.

Bush, S. S., Ruff, R. M., Troster, A. I., Barth, J. T., Koffler, S. P., Pliskin, N. H., et al. (2005). Symptom validity assessment: Practice issues and medical necessity NAN policy & planning committee. *Archives of Clinical Neuropsychology, 20*(4), 419–426.

Greve, K. W., & Bianchini, K. J. (2006). Classification accuracy of the Portland Digit Recognition Test in traumatic brain injury: Results of a known-groups analysis. *The Clinical Neuropsychologist, 20*(4), 816–830.

Greve, K. W., Bianchini, K. J., & Doane, B. M. (2006). Classification accuracy of the test of memory malingering in traumatic brain injury: Results of a known-groups analysis. *Journal of Clinical and Experimental Neuropsychology, 28*(7), 1176–1190.

Grosz, H. J., & Zimmerman, J. (1965). Experimental analysis of hysterical blindness: A follow-up report and new experimental data. *Archives of General Psychiatry, 13*, 255–260.

Hiscock, M., & Hiscock, C. K. (1989). Refining the forced-choice method for the detection of malingering. *Journal of Clinical and Experimental Neuropsychology, 11*(6), 967–974.

Iverson, G. L. (2008). Assessing for exaggeration, poor effort, and malingering in neuropsychological assessment. In A. M. Horton, Jr. & D. Wedding (Eds.), *Neuropsychology handbook* (3rd ed., pp. 125–182). New York: Springer.

Larrabee, G. J. (2003). Detection of malingering using atypical performance patterns on standard neuropsychological tests. *The Clinical Neuropsychologist, 17*(3), 410–425.

Mittenberg, W., Patton, C., Canyock, E. M., & Condit, D. C. (2002). Base rates of malingering and symptom exaggeration. *Journal of Clinical and Experimental Neuropsychology, 24*(8), 1094–1102.

Pankratz, L. (1983). A new technique for the assessment and modification of feigned memory deficit. *Perceptual and Motor Skills, 57*(2), 367–372.

Pankratz, L., Binder, L. M., & Wilcox, L. M. (1987). Evaluation of an exaggerated somatosensory deficit with symptom validity testing. *Archives of Neurology, 44*(8), 798.

Pankratz, L., Fausti, A., & Peed, S. (1975). A forced-choice technique to evaluate deafness in the hysterical or malingering patient. *Journal of Consulting and Clinical Psychology, 43*(3), 421–422.

Theodor, L. H., & Mandelcorn, M. S. (1973). Hysterical blindness: A case report and study using a modern psychophysical technique. *Journal of Abnormal Psychology, 82*(3), 552–553.

Vickery, C. D., Berry, D. T., Inman, T. H., Harris, M. J., & Orey, S. A. (2001). Detection of inadequate effort on neuropsychological testing: A meta-analytic review of selected procedures. *Archives of Clinical Neuropsychology, 16*(1), 45–73.

Syncope

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Synonyms

Fainting

Definition

Syncope is a temporary "black-out" episode (loss of consciousness with interruption of awareness of oneself and one's surroundings) that is caused by a sudden lack of blood supply to the brain. There can be many causes of syncope (both cardiac and noncardiac), but the two most common causes include vasovagal syncope (often triggered by pain or emotional stress) and orthostatic hypotension (triggered by a change from seated to standing position or when a person has been standing for an extended period of time). Seizure-like movements can occasionally accompany syncope, and it is important for physicians to rule out syncope when considering a diagnosis of epilepsy.

Cross References

► Epilepsy
► Seizure

References and Readings

Bergfeldt, L. (2003). Differential diagnosis of cardiogenic syncope and seizure disorders. *Heart, 89*, 353–358.

Grubb, B. P. (2007) *The fainting phenomenon: Understanding why people faint and what to do about it* (2nd ed.). New York: Blackwell Publishing.

Grubb, B. P., Gerard, G., Roush, K., Temesy-Armos, P., Elliott, L., Hahn, H., et al. (1991). Differentiation of convulsive syncope and epilepsy with head-up tilt testing. *Annals of Internal Medicine, 115* (11), 871–876.

Sheldon, R., Rose, S., Ritchie, D., Connolly, S. J., & Koshman, M. L. (2002). Historical criteria that distinguish syncope from seizures. *Journal of the American College of Cardiology, 40*, 142–148.

Syndrome

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Definition

Syndrome is a categorization of signs (objectively identified manifestation of a disorder) and symptoms (subjectively identified manifestation of a disorder), based on their repeated coexistence, which indicates that there may be common causal factors (American Psychiatric Association, 2000). The awareness of the presence of one or more of these signs by a medical or mental health professional would most likely result in assessment for other signs that are believed to coexist in a given condition.

References and Readings

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

Synesthesia

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Definition

Synesthesia is a form of dysesthesia in which a sensory perception is perceived in a different sensory modality. One of the most common forms is "grapheme-color" synesthesia, in which the individual perceives colors when reading letters or numbers. Other examples include patients who "taste" colors or "hear" shapes. Synesthesia appears to be familial, but does not appear to be gender-specific. Estimates of its prevalence vary and may depend on the specific type (e.g., grapheme-color synesthesia has been estimated to occur in 1 in 200 people). Synesthesia is also often reported by individuals under the influence of

psychedelic drugs; however, these individuals tend to experience very complex intersensory experiences, whereas those of the "congenital synesthete" tend to be simpler (letters elicit colors).

With regard to the neural systems involved, hypotheses include cross-activation between adjacent cortical-cortical sensory systems (i.e., visual word form and color-processing areas) and disinhibited feedback from association areas (i.e., temporal–parietal–occipital junction). An additional hypothesis ("reentrant processing") suggests that synesthesia is the result of bidirectional flow of sensory information, although this is not completely distinguished from the disinhibition feedback hypothesis noted above.

Cross References

► Sensorimotor Assessment

References and Readings

Baron-Cohen, S., Burt, L., Smith-Laittan, F., Harrison, J., & Bolton, P. (1996). Synaesthesia: Prevalence and familiarity. *Perception, 25*, 1073–1079.

Cytowic, R. E. (2003). *The man who tasted shapes.* Cambridge, MA: MIT Press.

Nunn, J. A., Gregory, L. J., Brammer, M., Williams, S. C. R., Parslow, D. M., Morgan, M. J., et al. (2002). Functional magnetic resonance imaging of synesthesia of V4/V8 by spoken words. *Nature Neuroscience, 5*(4), 371–375.

Ramachandran, V. S., & Hubbard, E. M. (2001). Synaesthesia: A window into perception, thought and language. *Journal of Consciousness Studies, 8*, 3–34.

Shanon, B. (2002). Ayahuasca visualizations: A structural typology. *Journal of Consciousness Studies, 9*, 3–30.

Syntax

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Definition

The set of rules governing the formation of phrases or sentences in a language.

Cross References

► Agrammatism
► Grammar
► Morpheme
► Paragrammatism
► Phonology

Systemic Lupus Eythematosus

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Synonyms

Lupus; SLE

Definition

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that is characterized by multisystem involvement and diverse manifestations. SLE is typified by the production of autoantibodies, and a hallmark of the disease is the presence of serum antibodies directed to nuclear constituents (i.e., antinuclear antibodies). SLE can affect almost any organ in the body; the primary areas affected by SLE include the heart, lungs, skin, joints, blood-forming organs, kidneys, and the central nervous system (CNS). For more information on definitions, symptoms, diagnosis, treatment, and research in SLE, log onto www.niams.nih.gov/health_Info/Lupus sponsored by the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS) of the National Institutes of Health (NIH).

Categorization

Over 50% of patients with SLE demonstrate major psychiatric and neurologic disorders indicating CNS involvement (Bluestein, 1992; West, 2007). Neuropsychiatric

(NP) syndromes in SLE are diverse and may include major manifestations (i.e., stroke syndromes, seizures, psychotic episodes, etc.), or less severe abnormalities including headaches, minor mood disorders, and cognitive difficulties (West, 2007). In SLE patients with overt NP involvement, pathogenic mechanisms have been separated based on the type of NP presentation. For example, in patients with diffuse manifestations (e.g., psychosis and depression), autoantibodies directed to CNS antigens have been proposed as important pathogenic factors. In contrast to diffuse disease, focal CNS presentations in SLE such as stroke syndromes, focal seizures, movement disorders, and myelopathy have been postulated to be due to ischemia (West, 2007). Patients with neuropsychiatric SLE are typically referred to as NPSLE or CNS–SLE.

In the revised criteria for neuropsychiatric lupus, 19 neuropsychiatric syndromes were defined including 11 CNS disorders and eight peripheral nervous system disorders (The American College of Rheumatology nomenclature and case definitions for neuropsychiatric lupus syndromes, 1999). In this revised nomenclature, cognitive dysfunction was identified as one of the major neuropsychiatric syndromes and was defined as "significant deficits in any or all of the following cognitive functions: complex attention, executive skills (e.g., planning, organizing, sequencing), memory (e.g., learning, recall), visual-spatial processing, language (e.g., verbal fluency), and psychomotor speed."

Epidemiology

As reported in a recent review of the data (Rus, Maury, & Hochberg, 2007), the overall prevalence of SLE in the USA has been reported to be between 14.6 and 122 cases per 100,000. The annual incidence in the USA has been reported between 1.8 and 7.6 cases per 100,000. International studies report similar incidence rates. Prevalence and incidence are higher in females compared to males (female predominance approximately 90%), and higher in Afro-Americans, Afro-Caribbeans, and Asians than in Caucasian populations. The median age of diagnosis is 37–50 in women. The prevalence of NP disease varies between 37% and 95%, with most common syndromes including cognitive dysfunction, headache, mood disorders, cerebrovascular disease, and seizure (Hanly, Kuznetsova, & Fisk, 2007).

Natural History, Prognostic Factors, Outcomes

The revised criteria for the diagnosis of SLE was proposed in 1997 and consisted of four or more of 11 manifestations that are present either serially or simultaneously (Hochberg, 1997). Patients tend to develop SLE in a series of steps, starting with a predisposition of autoantibody activity, followed by the development of the complete clinical SLE syndrome and then periods of intermittent disease flares and improvements. Genetic predisposition as well as a gender influence on disease susceptibility and environmental factors is also important to the development of SLE (Hahn, 2007). The prognosis for NP syndromes in SLE, including cognitive dysfunction, is unclear; some patients demonstrate residual damage, whereas others demonstrate recovery. Those patients with recurrent NPSLE episodes and with antiphospholipid syndrome appear to do worse cognitively than those without these syndromes (West, 2007).

Neuropsychology and Psychology of SLE

Studies using standard neuropsychological tests have reported cognitive impairment in varied percentages of SLE patients, with estimates ranging from 14% to 79%. Inconsistencies across studies are largely methodological, including differences in the characteristics of the lupus sample and the selection of neuropsychological tests used to classify impairment. Across SLE studies, deficits in attention, learning and recall, verbal and nonverbal fluency, complex psychomotor functions, visuospatial skills, and motor dexterity have been reported (Denburg, Laroque, & Denburg, 2004; Kozora, 2008). Attention, learning, and memory problems appear to be the most consistently impaired areas in SLE patients when compared to controls (Kozora, 2008). As expected, SLE patients with overt NP syndromes such as strokes and seizures tend to have more severe and extensive cognitive deficits compared to SLE patients without major NP events. Although the course is variable, the majority of patients have a fluctuating pattern of cognitive dysfunction with only a minority showing progressive decline (Hanly et al., 2007). Given the multiple etiological mechanisms of NP syndromes in SLE, it is not surprising that dominant patterns of cognitive deficits have not emerged, and it is clear that cognitive dysfunction in SLE is not a single syndrome.

Studies to date suggest that cognitive impairment in SLE is in part mediated by autoantibody activity and mechanisms associated with ischemia (Denburg et al., 2004; Hanly et al., 2007). Additional mediators of cognitive function in SLE patients include health characteristics (disease activity, length of disease, medication use), immune activity (autoantibodies, proinflammatory cytokines), and behavioral factors such as depression, pain, and fatigue (Hanly et al., 2007; Kozora, 2008). A number of neuroimaging techniques including magnetic resonance imaging, magnetic resonance spectroscopy, diffusion tensor imaging and functional imaging have been helpful in identifying and understanding brain abnormalities in SLE (Hanly et al., 2007). The most common MRI abnormalities include diffuse periventricular hyperintensity infarcts, hemorrhage, cerebral atrophy, and small focal lesions. To date, white matter pathology identified by MRS as well as DTI in SLE patients is associated with attentional and executive dysfunction (Kozora, 2008).

A number of studies have suggested that psychosocial factors such as depression and anxiety occur in approximately 50% of SLE patients (Cohen, Roberts, & Levenson, 2004). Psychiatric disorders are multifactorial and acute confusional state, anxiety disorder, mood disorder, and psychosis have all been identified as NP manifestations in the revised nomenclature (The American College of Rheumatology nomenclature and case definitions for neuropsychiatric lupus syndromes, 1999). Depression and anxiety have been reported in 24–57% of studies of SLE patients (Hanly et al., 2007). Depression is the most common emotional disturbance, but there is considerable debate regarding the diagnosis, prevalence, and etiology of depression in SLE.

Evaluation

Given the wide range of potential cognitive deficits in SLE, evaluation of all domains of cognitive testing is encouraged including overall intelligence, complex attention and visuomotor speed, learning and memory of visual and verbal material, executive functioning and higher problem solving, language ability, visuospatial functions, and sensory and motor skills. For research, a brief battery of tests were recommended by the American College of Rheumatology, and reliability and validity have been established in a prior study (Kozora, Ellison, & West, 2004). Measures of depression, fatigue, and pain were also recommended by this committee.

Treatment

There are few formal treatment procedures empirically validated for cognitive dysfunction in SLE. Corticosteroid treatment for neuropsychiatric features (and cognitive dysfunction) of SLE has been prominent for some time (Denburg et al., 2004). However, a variety of mechanisms underlie NP and cognitive change, and this influences available treatment options. For example, if the cognitive deficit is associated with vasculopathy and the presence of antiphospholipid antibodies, then anticoagulation or antiplatelet drugs may be relevant. Guidelines for pharmacological management of NPSLE syndromes can be found in Hanly et al. (2007). Few rehabilitation studies for cognitive dysfunction in SLE exist. Promising results obtained from psychoeducational treatment aimed at improving memory, self-efficacy, and the ability to perform daily activities in SLE have been reported (Harrison et al., 2005).

References and Readings

Bluestein, H. G. (1992). The central nervous system in systemic lupus erythematosus. In R. G. Lahita (Ed.), *Systemic lupus erythematosus* (2nd ed., pp. 639–655). New York: Churchill Livingstone, Inc.

Cohen, W. S., Roberts, W. N., Jr., & Levenson, J. L. (2004). Psychiatric aspects of systemic lupus erythematosus. In R. G. Lahita (Ed.), *Systemic lupus erythematosus* (4th ed., pp. 785–825). New York: Elsevier Inc.

Denburg, S. D., Laroque, L., & Denburg, J. A. (2004). Cognitive dysfunction in systemic lupus erythematosus. In R. G. Lahita (Ed.), *Systemic lupus erythematosus* (pp. 827–843). New York: Churchill Livingstone, Inc.

Hahn, B. V. H. (2007). Overview of pathogenesis of systeic lupus erythematosus. In D. J. Wallace, & B. H. Hahn (Eds.), *Dubois' lupus erythematosus* (pp. 46–53). Philadelphia, PA: Lippincott, Williams & Wilkins.

Hanly, J. G., Kuznetsova, A., & Fisk, J. D. (2007). Psychopathology of lupus and neuroimaging. In D. J. Wallace, & B. H. Hahn (Eds.), *Dubois' lupus erythematosus* (7th ed.). Philadelphia, PA: Lippincott, Williams & Wilkins.

Harrison, M. J., Morris, K. A., Horton, R., Toglia, J., Barsky, J., Chait, S., et al. (2005). Results of intervention for lupus patients with self-perceived cognitive difficulties. *Neurology, 65*(8), 1325–1327.

Hochberg, M. C. (1997). Updating the american college of rheumatology revised criteria for the classification of systemic lupus erythematosus. *Arthritis and Rheumatism, 40*(9), 1725.

Kozora, E. (2008). Neuropsychological functioning in systemis lupus erythematosis. In J. E. Morgan, & J. H. Ricker (Eds.), *Textbook of clinical neuropsychology* (pp. 636–659). New York: Taylor and Francis.

Kozora, E., Ellison, M. C., & West, S. (2004). Reliability and validity of the proposed american college of rheumatology neuropsychological battery for systemic lupus erythematosus. *Arthritis and Rheumatism, 51*(5), 810–818.

Rus, V., Maury, E. E., & Hochberg, M. C. (2007). *The epidemiology of systemic lupus erythematosus Dubois' lupus erythematosus* (7th ed.). Philadelphia, PA: Lippincott, Williams & Wilkins.

The American College of Rheumatology nomenclature and case definitions for neuropsychiatric lupus syndromes. (1999). *Arthritis and Rheumatism, 42*(4), 599–608.

West, S. G. (2007). Neuropsychiatric lupus. In D. J. Wallace, & B. H. Hahn (Eds.), *Dubois' lupus erythematosus* (7th ed.). Phildelphia, PA: Lippincott, Williams & Wilkins.

Systemic Therapy

► Chemotherapy