C

C/NC

► Coma/Near Coma Scale

CABG

► Coronary Artery Bypass Graft

Cadasil

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Synonyms

Agnogenic medial arteriopathy; Chronic familial vascular encephalopathy; Familial Binswanger's disease; Familial disorder with subcortical ischemic strokes, dementia, and leukoencephalopathy; Hereditary multi-infarct dementia

Short Description or Definition

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is a rare autosomal dominant, adult-onset inherited cerebral vascular disease, characterized by migraines, recurrent transient ischemic attacks, and strokes leading to cognitive decline and dementia.

Current Knowledge

Clinical Manifestation: The typical age of onset is 30–500 years of age. The initial clinical manifestation often begins with migraines with aura, and slowly progresses to transient ischemic attacks (TIA), and recurrent strokes. The migraine attacks are often reported to be particularly long lasting or severe and may even include symptoms of hemiplegia. The strokes are typically lacunar, occurring in subcortical white matter or basal ganglia and, in some cases, occur in the brain stem and spinal cord. Recurrent subcortical infarcts lead to cognitive decline, pseudobulbar palsy, motor impairment, psychiatric symptoms (most commonly depression), and ultimately total motor impairment and subcortical vascular dementia. Cognitive decline is most prominently seen as executive dysfunction, slowed processing speed, and reduced attentional abilities. As the disease progresses, memory and other areas of cognitive functioning begin to decline. The total amount of white matter involvement associated with the lacunar lesions and the degree of atrophy predict severity of cognitive and motor impairment.

Cause: CADASIL is caused by mutations or deletions in the *Notch3* gene on chromosome 19, which plays an important role in cell differentiation during development. While the pathogenetic mechanism has yet to be determined, the resulting pathophysiology includes thickening of arterial walls, accumulation of granular osmiophilic material (GOM) in arterial walls (this distinguishes it from arterial hypertension), gradual destruction of vascular smooth muscle cells leading to fibrosis, progressive thickening of arterial walls, and narrowing of the lumen of cerebral arteries. This eventually causes thrombosis, reduced blood flow, destruction of small- and medium-sized arteries, and consequently focal infarcts. MRI typically reveals characteristic periventricular or white matter hyperintensities, as well as infarcts in the basal ganglia and brain stem. The cerebral cortex remains relatively intact.

Diagnosis: Identification of a *Notch3* mutation provides the most certain diagnosis, in addition to the presence of GOM in arterial walls, which can be detected with a skin biopsy. Positive MRI findings are often detected in even asymptomatic individuals. History of particularly severe migraines may be the earliest indication.

Treatment and prognosis: Currently, there is no cure or disease-modifying therapy for CADASIL, and only symptom management and supportive care are available for

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affected individuals. Death typically occurs 10–20 years after the onset of symptoms. Angiography and anticoagulants should be avoided as they may provoke cerebrovascular accidents.

Cross References

- ▶ Binswanger's Disease
- ▶ Lacunar Stroke
- ▶ Small Vessel Ischemic Disease

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CAGE Questionnaire

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Definition

The CAGE is one of a number of brief screening instruments designed to help in the detection of alcohol misuse. The instrument's name is an acronym for questions about *C*utting down on drinking, *A*nnoyance at other's concern about drinking, feeling *G*uilty about drinking, and using alcohol as an *E*ye-opener in the morning. The CAGE can be rapidly administered in an interview format or written format and consists of the following four items: (1) "Have you ever felt that you should cut down on your drinking?"; (2) "Have people annoyed you by criticizing your drinking?"; (3) "Have you ever felt bad or guilty about your drinking?"; and (4) "Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover (eye-opener)?" Individual item responses are scored in a binary fashion ("0" = "no" and "1" = "yes"). The total score can range from 0 to 4. A score of 2 or greater is typically considered a positive finding, that is, an indication of alcohol misuse.

Historical Background

The CAGE was developed by Ewing and Rouse (1968) for detection of "alcoholism" and was first introduced as a formal screening instrument 2 years later (Ewing & Rouse, 1970). Final item selection for the CAGE was based on examination of 130 randomly selected, general hospital patients; items selected were those that resulted in a "minimal" set of items that "usefully divided" responders into two groups, patients with "alcoholism" (confirmed by physician impressions and chart study) and patients without indication of "alcoholism" (Ewing, 1984).

Over the years, several modifications to the original instrument have been recommended, including placing a time frame reference on the four questions (e.g., in the last year), as well as adding questions regarding frequency of use, tolerance, and perceived history of drinking problems (Bradley, Kiviahan, Bush, McDonell, & Fihn, 2001; National Institute on Alcohol Abuse and Alcoholism, 1995; McQuade et al., 2000).

Psychometric Data

Test–retest reliability of the CAGE over a 7-day period was 0.80 in a psychiatric sample and 0.95 in a nonclinical sample (Teitelbaum & Carey, 2000); mean CAGE score changes for each group across this same time period were nonsignificant. Convergent validity has been reasonably good, though somewhat variable, with reported correlations ranging from 0.48 to 0.70 with other alcohol-screening instruments (see Dhalla & Kopec, 2007 for review).

A number of studies have examined the validity of CAGE in detecting alcohol misuse. Aertgeerts, Buntinx, and Kester (2004) performed a meta-analysis of 10 studies of the utility of the CAGE in detecting alcohol abuse or alcohol dependency in general clinical populations using criteria from the Diagnostic and Statistical Manual for Mental Disorders (DSM-III-R' APA, 1987). With a cutoff

score at ≥2, pooled sensitivities across studies were 0.87 in hospital inpatients, 0.71 in primary care patients, and 0.60 in ambulatory medical patients; specificities were 0.77, 0.91, and 0.92, and positive predictive validities were 0.57, 0.74, and 0.82, respectively. Aertgeerts et al. (2004) concluded that the CAGE is only of "limited value" as a screening instrument using a cutoff score of ≥2 and recommended that additional information about alcohol use patterns be obtained from any patient who gives one positive answer on the instrument.

Dhalla and Kopec (2007) provided a more recent review of the CAGE validity literature, considering, in part, the sensitivity, specificity, and positive predictive value associated with different cutoff scores. The authors concluded that the CAGE has "adequate validity" in screening medical and surgical inpatients, psychiatric inpatients, and ambulatory medical patients for alcohol misuse and that use of a cutoff score of ≥2 affords the best combination of sensitivity, specificity, and positive predictive value. At the same time, they cautioned that the CAGE has not performed well with Caucasian women, prenatal women, and college students.

Fiellin, Carrington, and O'Conner (2000) also provide a noteworthy review of validity studies, concluding that in primary care settings, the CAGE questionnaire was more effective in identifying patients with alcohol abuse or dependency than in detecting patients with at risk, hazardous, or harmful drinking.

Clinical Uses

The CAGE has enjoyed wide popularity in primary care settings as an alcohol-screening instrument for a number of reasons, as it is brief, easy to administer, and can be incorporated into more extensive interviews or questionnaires (Bradley et al., 2001). However, support for its validity across different populations has been mixed. It appears to perform best when used to screen for alcohol abuse or dependence in clinical populations and less well in detecting current hazardous drinking. Caution should be used especially when interpreting results for nonclinical respondents (i.e., general population) as well as for women and college students, as the CAGE has been noted to under-perform in these populations. In addition, as several authors have recommended, a positive finding on the CAGE, regardless of cutoff score level (i.e., ≥1 or 2), should be followed up by further examination of alcohol consumption patterns, using established standard criteria.

Cross References

- ▶ Alcohol Abuse
- ▶ Alcohol Dependence
- ▶ Alcoholic Brain Syndrome
- ▶ Alcoholism
- ▶ Blood Alcohol Level
- ▶ Fetal Alcohol Syndrome
- ▶ Korsakoff's Syndrome
- ▶ Michigan Alcoholism Screen Test
- ▶ Substance Abuse
- ▶ Substance Abuse Disorders
- ▶ Wernicke–Korsakoff's Syndrome

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CalCAP

► California Computerized Assessment Package

Calcarine Cortex

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Synonyms

Primary visual cortex; Striate cortex

Structure

This is the term for the primary visual cortex, V1, or Brodmann's area 17. The area encompasses the medial surface and a small lateral surface of the occipital lobe, and is within the calcarine sulcus. It is located at the most posterior portion of the cerebral hemisphere. The optic radiations terminate in layer IV of the calcarine cortex. Layer IV is rather thick and is subdivided into sublaminae. The calcarine cortex is also sometimes referred to as the striate cortex because of a strip of myelin (Stria of Gennari) that can be visualized. The area is organized in a retinotopic fashion; that is, there is point-to-point representation from the retina to the cortex. For instance, fibers in the right half of each retina (perceives information in the left visual field) project to the right calcarine cortex, and fibers in the left half of each retina (perceives information in the right visual field) project to the left calcarine cortex.

Function

From a functional perspective, the calcarine cortex is important in determining orientation, spatial-frequencies, and color properties of the visual stimulus. The information is then projected to other areas in the occipital lobe for further visual analysis. This occurs via transmission to the dorsal (where) and the ventral (what) streams to the parieto-occipital association cortex and occipitotemporal association cortices, respectively.

Illness

The calcarine cortex is perfused by the calcarine artery, which is a limb off the posterior cerebral artery. Our visual system has evolved such that damage to one eye does not result in blindness in one visual field. Damage to either the right or left calcarine cortex results in a contralateral field cut, a hemianopia. Macular sparing usually occurs because of collateral blood supply from branches of the middle cerebral artery. Isolated lesions of the primary visual cortex results in discrete blind spots (scotomas) in the corresponding area of the visual field.

Cross References

► Calcarine Fissure
► Calcarine Sulcus
► Occipital Lobe
► Scotoma
► Unimodal Cortex
► Visual Cortex

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Calcarine Fissure

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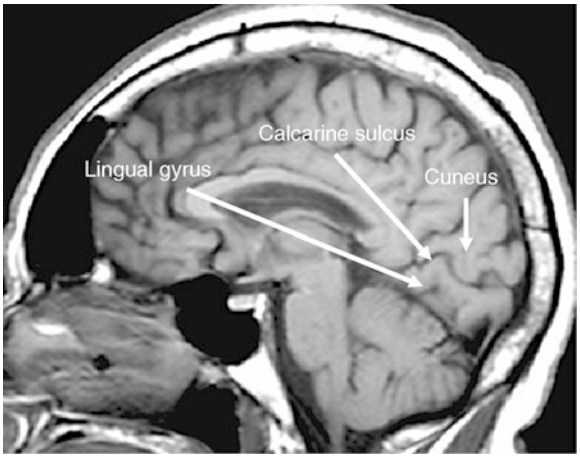
Synonyms

Calcarine sulcus



Calcarine Fissure. Figure 1 Mid-sagittal view of the brain (MRI) showing calcarine fissure separating the cuneus and the lingual gyrus of the occipital lobe

Definition

The calcarine fissure is a deep sulcus located on the medial surface of the occipital lobe (see Fig. 1). The superior (*cuneus*) and inferior (*lingual gyrus*) banks of this sulcus represent the primary cortical projection area for vision. Visual information that is first received in the upper portions of the retina is represented along the upper banks of the fissure (cuneus), while that derived from the lower retina projects to its lower bank. Thus, lesions confined to the upper bank (cuneus) will result in contralateral inferior visual field defects, while damage to the lower bank (lingual gyrus) will produce upper field deficits. Lesions destroying tissue along both the upper and lower banks may result in a contralateral homonymous hemianopia.

Cross References

- ▶ Cuneus
- ▶ Lingual Gyrus
- ▶ Visual System

Calcarine Sulcus

- ▶ Calcarine Fissure

California Computerized Assessment Package

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Synonyms

CalCAP

Description

The California Computerized Assessment Package (CalCAP; 1999) presents a series of brief reaction time tasks designed to assess speeded information processing and psychomotor functioning. The Standard CalCAP consists of 4 Simple and 6 Go–No Go reaction time subtests that require 20–25 min to complete. The individual reaction time measures were designed to assess a number of cognitive domains, including speed of processing (reaction time), lexical discrimination (real word vs. nonsense word), rapid visual scanning, form discrimination and matching, working memory (1-back and 2-back tasks), and sustained and divided attention. Each task includes a practice session that must be completed with 100% accuracy to proceed to the main 2-min procedure, a method that ensures that subjects understand the task instructions and that minimizes practice effects in longitudinal study designs. Subjects are asked to focus on a display field and respond only to specific types of visual stimuli. For example, in the form discrimination task, they are asked to press a key only when two out of three non-nameable figures are identical.

The core measures of reaction time, hits and false-positives, and signal detection parameters allow investigators to measure accuracy and general speed of processing, as well as relative differences among conceptually distinct decision tasks. Four iterations of a Simple reaction time routine at various points in the test allow direct measurement of the subject's ability to maintain focus throughout the 20-min testing procedure. The computer scores each task using age- and education-specific norms derived from 641 men ranging from 21 to 58 years of age, with a mean education of 16 years. Final scores are available immediately in tabular and graphical formats. Additional norms have been derived for elementary school children (3rd, 5th, and 6th grades), women, and older individuals. The CalCAP

also includes a 4-subtest language-independent abbreviated battery weighted toward measures of speeded processing and working memory. These test batteries are well suited for collecting reliable information on psychomotor functioning in a brief period of time, and can be used effectively for assessing changes over time. Stimulus materials are available in English, Spanish, French, Norwegian, Danish, and Flemish.

Historical Background

The CalCAP was originally developed by Dr. Eric Miller in the late 1980s as an automated substitute for traditional neuropsychological testing. The Standard battery, developed in consultation with Dr. Paul Satz and Dr. Ola Selnes, was designed to measure aspects of language and visuoperceptual skills, working memory, speeded information processing, attention, learning and memory, visual scanning, and reaction time. While the reaction time tasks that comprise the Standard CalCAP battery do assess many of these areas of cognitive functioning, studies have shown that the tasks correlate only modestly (0.2–0.4) with traditional neuropsychological measures (Miller, Satz, & Visscher, 1991). Factor analyses using the CalCAP and other neuropsychological test batteries show that the reaction time indices form two primary factors best characterized as simple reaction time and decision speed. These factors are distinct from traditional neuropsychological measures and most researchers and clinicians now use the CalCAP and other reaction time measures primarily as indices of these types of speeded information processing and attention. The abbreviated version of the CalCAP specifically excludes measures designed to look at visuoperceptual and language skills and focuses primarily on these core indices of reaction time, working memory, and attention.

Psychometric Data

All the subtests in the CalCAP have high internal consistency reliability (0.77–0.96). Six-month test–retest reliability for the Go–No Go paradigms is comparable with levels seen for conventional neuropsychological procedures (0.43–0.68), but there is less evidence of the practice effects commonly seen with psychomotor measures. The reduced practice effects may be due to the requirement that all subjects complete practice trials with 100% accuracy before proceeding to the actual test. Unlike the Go–No Go paradigms, the simple reaction time measures have relatively low test–retest reliability (0.20–0.29), suggesting that the psychomotor skills measured by the simple reaction time tasks vary considerably depending on state variables such as mood, attention, fatigue, and time of day (Miller, 2008).

CalCAP reaction time correlates most highly with age, and, to a lesser extent, with years of education. Two studies of gender effects on CalCAP reaction time have shown no differences between men and women on any of the CalCAP indices (Berg, 1994). The psychometric properties of the CalCAP are unstable for 3rd graders, but essentially the same as found in adults by the 5th grade (Budzinski, 1994).

Clinical Uses

The CalCAP has been widely used in cross-sectional and longitudinal studies of HIV/AIDS (Miller et al., 1991; Gonzalez et al., 2003), drug abuse (Chang et al., 2002; Volkow et al., 2001), depression (Stordal et al., 2004), epilepsy (Hessen, Lossius, M. I., Reinvang, I., & Gjerstad, 2008), traumatic brain injury (Waterloo, Ingebrigtsen, & Romner, 1997), and hyperbaric oxygen treatments (Hjalmarsen, Waterloo, Dahl, Jorde, & Viitanen, 1999; Van Hoof, Coomans, De Becker, De Meirleir, & Cluydts, 2002). It is particularly sensitive to the psychomotor changes seen in these disorders and, unlike most psychomotor tasks, has minimal practice effects, making it particularly appropriate for research paradigms such as clinical trials and epidemiologic studies that require repeated testing.

The cognitive functions assessed by the CalCAP program are best described as timed psychomotor skills requiring focused or sustained attention. Impaired reaction time across multiple measures is usually indicative of generalized motor slowing or slowed information processing. Impaired reaction time on specific measures, particularly when coupled with scores outside of normal bounds on true-positive responding, is suggestive of a more specific functional deficit, such as language skills (lexical discrimination task), visuoperceptual deficits (form discrimination), or working memory (1-back and 2-back tasks).

The standard CalCAP program classifies subjects as "outliers" if they perform two standard deviations or lower on two or more of the tasks. Using these criteria, approximately 10% of subjects are classified as outliers. This base rate of 10% includes individuals with premorbid conditions such as prior head injury, learning

disability, preexisting neurologic conditions, as well as individuals who are simply on the low end of normal functioning.

Cross References

- ▶ Attention/Executive Functions
- ▶ Continuous Performance Tests
- ▶ Information Processing Speed
- ▶ Speed of Information Processing
- ▶ Visual–Motor Function

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California Verbal Learning Test (California Verbal Learning Test-II)

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Synonyms

CVLT

Description

The California Verbal Learning Test-II (CVLT-II) is the latest (2000) version of a widely used verbal learning and memory test. The current version contains recalling and recognition of two lists of words over immediate and delayed trials. List A includes 16 words and requires the examinee to recall the list over five trials. List B (interference), which is also 16 words, is administered after List A for one trial. Short-delay free recall and cued recall are administered after List B. A 20-min delay follows the short-delay recalls, followed by nonverbal testing. Long-delay recall, long-delay-cued recall, and yes/no-recognition trials of List A follow the 20-min delay. A revision of the CVLT-II is the addition of a forced choice recognition after a 10-min delay following the yes/no trial.

Another revision of the CVLT-II is the inclusion of a short form (nine words) and an alternate form. The short form was created for the purpose of screening or for patients who may have more severe brain damage and may feel overwhelmed by the long form. This short form takes 15 min to administer and also has a 15-min delay. The alternate form has been statistically equated with the standard form.

The CVLT-II was normed from a national standardization sample of 1,087 of ages 16–89, which was demographically matched to the US population. It takes 30 min to administer and includes 30 min of delay. The test is administered with paper and pencil format, with the examiner recording the responses. The responses are

scored using the Comprehensive Scoring System, which computes raw scores and standardized scores for over 50 learning and memory variables. The raw scores can be scored manually, although there are limits to how much information can be derived.

The CVLT-children's (CVLT-C) version can be administered to children aged 5–16 and can assist professionals in diagnosing memory impairment secondary to learning disabilities, attention-deficit disorder, mental retardation, and other neurological disorders and psychiatric problems. The administration is similar to the format of the CVLT-II, although forced choice recognition is not included.

Historical Background

The first edition of the CVLT was originally published in 1987 from the work of Delis, Kramer, Kaplan, and Ober (1987). They created one of the first tests to incorporate principles from cognitive science to measure learning and memory. This test provided a measurement of not only what an individual remembers, but how they remember and what errors are made. This refuted previous beliefs that memory dysfunction was limited to recall and recognition.

Psychometric Data

CVLT-II: Split-half reliability coefficients based on splitting immediate-recall trials ranged from 0.89 to 0.94. Coefficient alphas based on word category scores across immediate-recall trials ranged from 0.71 to 0.82. The split-half reliability based on the number of times each word was correctly recalled across immediate-recall trials ranged from 0.68 to 0.79. The CVLT-II is correlated with the Wechsler Abbreviated Scale of Intelligence (WASI).

CVLT-C: The internal consistency reliability across five trials ranged from 0.84 to 0.91. Reliability coefficients for across-semantic category consistency ranged from 0.64 to 0.80.

Clinical Uses

The CVLT-II manual states that the test measures "multiple aspects of how verbal memory occurs or fails to occur, therefore providing a comprehensive assessment of each patient's profile of verbal learning and memory strengths and weaknesses." The manual reports a review of the literature of clinical populations from 1987 to 1999, summarizing the key findings of CVLT performance across various clinical populations, which include anterior temporal lobectomy, Korsakoff's syndrome, Alzheimer's disease, Huntington's disease, Parkinson's disease, head injury, schizophrenia, depression and other affective disorders, chronic alcohol and drug abuse, posttraumatic stress disorder, systemic medical disease (HIV, chronic fatigue syndrome, Lyme disease, eosinophilia myaligia syndrome, cardiac transplant candidates), insufficient effort, and predictor for everyday functioning.

Cross References

- ▶ Buschke Selective Reminding Test
- ▶ Children's Memory Scale
- ▶ Hopkins Verbal Learning Test
- ▶ Rey Auditory Verbal Learning Test
- ▶ Rivermead Behavioral Memory Test
- ▶ Selective Reminding Test
- ▶ Wechsler Abbreviated Scale of Intelligence
- ▶ Wechsler Memory Scale All Versions

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California Verbal Learning Test – Children's Version

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Synonyms

CVLT-C

Description

Overview of Test Procedures

The California Verbal Learning Test – Children's Version (CVLT-C) is a relatively brief, individually administered measure designed to assess the strategies and processes involved in learning and remembering verbal materials (Delis, Kramer, Kaplan, & Ober, 1994). Like the adult versions of this measure (CVLT; Delis, Kramer, Kaplan, & Ober, 1987; CVLT-II; Delis, Kramer, Kaplan, & Ober, 2000; ▶ California Verbal Learning Test (California Verbal Learning Test-II), the CVLT-C adopts a process-oriented approach for parsing a variety of learning and memory components, which facilitates the identification of distinct memory profiles associated with clinical disorders.

The CVLT-C uses words presented as part of two shopping lists (i.e., a "Monday list" and a "Tuesday list") and measures both recall and recognition of the words over a number of trials. The use of a "shopping-list" format makes the procedure more relevant for a child and provides information regarding how the child approaches an everyday memory task. Specifically, in the first five trials, the child is asked to recall words from the Monday list (List A), which consists of 15 words, with five words from each of three semantic categories (i.e., fruits, clothing, and toys). An interference Tuesday list (List B) of 15 words is then presented on one trial. Words from the Tuesday list are divided equally across three semantic categories (i.e., fruits, furniture, and desert). The Tuesday trial is followed by short-delay free- and cued-recall trials of the first (Monday) list. For cued-recall trials, the examinee is asked to recall words from the three semantic categories contained in List A. A 20 min delay occurs next, during which nonverbal measures should be administered. After the delay interval, long-delay free-recall, long-delay cued-recall, and yes/no recognition memory trials of the Monday list (List A) are administered.

With these procedures, the CVLT-C evaluates not only the level of a child's memory performance, but also the learning strategies used and types of errors made (Delis et al., 1994). Numerous learning and memory variables are quantified on the CVLT-C, including level of total recall and recognition on all trials, type of learning strategy employed (e.g., semantic clustering and serial clustering), serial position effects, degree of vulnerability to proactive and retroactive interference, retention of information over short and longer delays, enhancement of recall performance by category cuing and recognition testing, perseveration and intrusion errors in recall, and false-positive errors in recognition memory (Table 1 for CVLT-C variable definitions).

Standardization Sample

The CVLT-C was normed on a sample of 920 children from 12 age groups ranging from 5 through 16 years of age. The sample was stratified based on data from the 1988 U.S. Census (for additional information regarding the sample characteristics, see the test manual; Delis et al., 1994). In addition, normative data for 4-year-olds was provided by a study by Goodman, Delis, and Mattson (1999).

Score Conversions and Contrast Scores

Raw scores for the various CVLT-C indices are converted into an age-based *T* or *z* scores (see test manual for description of procedures used to derive these standardized scores; Delis et al., 1994). Although the test manual includes tables for converting the numerous raw scores to standardized scores, these scores are computed automatically using the CVLT-C computer scoring software. The standard score for List A trials 1–5 total is presented in a *T*-score metric, with a mean of 50 and a standard deviation of 10. The remaining standardized scores are presented in a *z*-score metric, with a mean of 0 and a standard deviation of 1.

Examination of contrast scores (i.e., *z*-score differences between two variables) allows for the assessment of particular learning and memory processes. For example, comparing List B (Tuesday list) recall with List A (Monday list) Trial 1 recall provides a measure of a child's susceptibility to proactive interference. As another example, contrasting List A long-delay free recall with List A short-delay free recall can be used to identify rapid forgetting across delays (Delis et al., 1994; Delis et al., 2000; Donders & Minnema, 2004).

Historical Background

In the past, the clinical neuropsychological assessment of memory relied on tests designed primarily to assess the presence or absence of memory dysfunction per se. This goal was accomplished by scoring memory tests only in terms of the level of correct recall or recognition. However, over the past 25 years or so, numerous studies from cognitive neuropsychology have documented qualitatively

California Verbal Learning Test – Children's Version. Table 1 CVLT-C variable definitions

Variable	
List A total	Total number of words recalled across the five learning trials
List A1	Number of words recalled from the first trial
List A5	Number of words recalled from the fifth trial
List B	Number of words recalled from the interference list (List B)
List A short-delay free recall	Number of words recalled from List A immediately after the presentation of the interference list (List B)
List A short-delay cued recall	Number of words recalled from List A with semantic cueing
List A long-delay free recall	Number of words recalled from List A after a 20 min delay
List A long-delay cued recall	Number of words recalled from List A with semantic cueing after a 20 min delay
Semantic clustering	Number of consecutively recalled words from the same semantic category (i.e., consecutive words from the same semantic category), which reflects the extent to which the subject has actively imposed a semantic organization on the list of words
Serial clustering	Number of consecutively recalled words in the same order as they were presented (i.e., a serial cluster)
Primacy %	Percentage of words correctly recalled from the beginning of List A (first four words)
Middle %	Percentage of words correctly recalled from the middle of List A (middle seven words)
Recency %	Percentage of words correctly recalled from the end of List A (last four words)
Learning slope	Average number of new words acquired across the five List A immediate-recall trials (e.g., a score of 1 means that the examinee learned approximately one new word per trial)
Consistency %	Percentage of target words recalled once on each of the four learning trials that were also recalled on the very next trial; reflects ability to maintain a consistent learning strategy
Perseverations	Total number of target words repeated within a trial (called "repetitions" on the CVLT-II; Delis et al., 2000)
Free-recall intrusions	Total number of extra-list intrusions on all the free-recall trials
Cued intrusions	Total number of extra-list intrusions on all the cued-recall trials
Total intrusions	Total number of extra-list intrusions made on free- and cued-recall trials
Recognition hits	Total number of target words correctly identified on recognition testing as belonging to List A (yes/no format)
Recognition discriminability	Accuracy of distinguishing target from distracter words on recognition testing; calculated using signal detection methods
False positives	Total number of distractor words incorrectly identified as belonging to List A during recognition testing
Response bias	Tendency to favor "yes" or "no" responses on recognition testing (positive scores reflect a "yes" bias)

distinct patterns of memory dysfunction associated with various neurological and psychiatric populations. These findings emphasize the multicomponential nature of learning and memory, and, as such, the importance of developing clinical memory tests that allow for the evaluation of both qualitative and quantitative features of memory performance. Informed by principles of learning/memory from cognitive science and cognitive neuroscience, and modeled in part after the Rey Auditory Verbal Learning Test (Rey AVLT; Rey, 1964), the CVLT-C, like its adult counterpart (► California Verbal Learning Test (California Verbal Learning Test-II)), was developed to facilitate the quantification of multiple learning and memory parameters (Delis et al., 1987; Delis, Freeland, Kramer, & Kaplan, 1988; Delis et al., 1994; Delis et al., 2000).

Psychometric Data

Reliability and Validity

The reliability of the CVLT-C was assessed using measures of internal consistency and test–retest reliability (Delis et al., 1994). The internal consistency coefficient for the five trials of the Monday list ranged from .84 to .91 (with a mean of .88) and the across-semantic-category consistency coefficient ranged from .64 to .80 (with a mean of .72). Test–retest stability (median retest interval of 28 days) ranged from .90 to .17, with higher test–retest correlations typically occurring for measures of overall level of performance (e.g., total words recalled during the five learning trials and long-delay free recall). Practice effects were fairly large for certain conditions. For example, trials 1–5 total recall improved by an average of five words for 8-year-olds, by six words for 12-year-olds, and by nine words for 16-year-olds. The average improvement on List B, the short-delay, and long-delay trials ranged from one to two words across all the groups.

Evidence supporting the validity of CVLT-C comes from factor analyses of the original standardization sample and studies of CVLT-C performance in various neurological and neurodevelopmental populations. As described in the test manual, exploratory principal components analysis of the primary CVLT-C performance indices produced a six-factor solution that closely paralleled the adult version (CVLT; Delis et al., 1987). In addition, Donders (1999) employed confirmatory factor analysis to reanalyze the CVLT-C standardization sample, and found a five-factor solution that included attention span (List A, Trial 1; List B; middle region recall), learning efficiency (List A, Trial 5; semantic clustering; recall consistency), free delayed recall (ahort-delay free recall, long-delay free recall), cued-delay recall (short-delay cued recall, long-delay cued recall), and inaccurate recall (total intrusions, recognition false positives).

Clinical Uses

The CVLT-C was designed to assess subtle to severe verbal learning and memory deficits in child clinical populations. The extant literature indicates that the CVLT-C has utility for characterizing the learning and memory deficits associated with a wide-range of clinical disorders (for review, see Strauss, Sherman, & Spreen, 2006). In addition, CVLT-C performance has been shown to be predictive of long-term educational outcomes in children who have suffered a traumatic brain injury (Miller & Donders, 2003).

Because the reliability characteristics of the primary CVLT-C performance indices (e.g., the total words recalled during the learning trials) are stronger than for the "process" or strategy-use variables (e.g., semantic clustering), it has been recommended that clinicians place greater emphasis on the main performance variables and interpret process-variables cautiously (Strauss et al., 2006). In order to evaluate learning and memory in other formats (e.g., story memory) or modalities (visual), the CVLT-C can be complemented with measures from other standardized children's memory assessment batteries (e.g., Children's Memory Scale and Wide Range Assessment of Memory-Second Edition).

Cross References

- ▶ California Verbal Learning Test (California Verbal Learning Test-II)
- ▶ Children's Memory Scale
- ▶ Rey Auditory Verbal Learning Test

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Callosal Apraxia

► Alien Hand Syndrome
► Apraxia

Callosal Disconnection Syndrome

► Split-Brain

Cambridge Neuropsychological Testing Automated Battery

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Synonyms

CANTAB

Description

The Cambridge Neuropsychological Testing Automated Battery (CANTAB) is a neuropsychological measure developed at the University of Cambridge and currently marketed by Cambridge Cognition Limited (CCL). The CANTAB is a theoretically derived instrument, predominantly measuring non-language functions. The current version is a Windows-based program operating on a PC platform utilizing interactive touch screen technology. CANTAB tests are described as having sufficient sensitivity to discern changes in cognitive functioning brought about by CNS disorders and medications.

Historical Background

Originally created in the late 1980s to diagnose dementia in elderly individuals (Fray et al., 1996), the CANTAB is used, and has been studied, across age groups and with patients presenting with both psychiatric and neurological conditions.

Current Knowledge

The CANTAB includes two products: CANTABeclipse and CANTABelect. The CANTABeclipse is composed of 22 tests grouped in specific functional dimensions measuring executive functioning, visual memory, attention, semantic/verbal memory, and decision making/response control. A sixth functional category labeled "induction" is comprised of two very short tests whose purpose is to familiarize examinees with the general idea of responding by touching the screen and to identify those examinees who cannot reliably participate in the examination. Details of each subtest are described in Table 1. The CANTAB offers users flexibility by providing subtests that can be selected for administration based upon the specific clinical question. All subtest stimuli are delivered nonverbally, although adequate receptive language functions are necessary to understand task demands. The publisher regards the majority of CANTAB tests to be independent of language and culture.

CANTABelect is described as a "bespoke service" geared toward conducting pharmaceutical clinical trials. In this case, CCL configures a battery to meet the specific research needs and requirements of the investigator.

Research supporting the use of the CANTAB in the evaluation of brain–behavior relations is reasonably extensive, with many peer reviewed articles available for review on the company's website www.cantab.com. Studies are available supporting the validity and utility of the CANTAB in measuring the impact of pharmacotherapy on neuropsychological performance, and in the assessment of Attention Deficit Hyperactivity Disorder, affective disorders, schizophrenia, senile dementia of the Alzheimer's type and Parkinson's disease. Although considerable research is available attesting to the usefulness of the CANTAB with adult populations, it has not been studied to the same extent in the assessment of brain-behavior relations and their developmental evolution among children.

More recent is the investigation of the CANTAB as a tool to assess the neuropsychological functioning of children. Luciana (2003) noted that because the format and test items can be used in the assessment of all age groups, the test allows for complex cognitive functions to be measured from the time they developmentally emerge to the point where they begin to diminish. DeLuca and colleagues (2003) studied the development of executive capacities during childhood and beyond. Using a sample of participants ranging in age from 8 to 64 years, they found functional gains in the efficiency of working memory capacity, planning, and problem solving abilities between ages 15 and 19 and again from ages 20–29 years. In

Cambridge Neuropsychological Testing Automated Battery. Table 1 CANTAB tests

Subtest	Functional category	Skill measured
Motor screening	Induction	Capacity to undergo CANTAB assessment
Big little circle	Induction	Rule following
Delayed matching to sample	Visual memory	Forced choice recognition
Paired associates learning	Visual memory	Episodic memory
Pattern recognition memory	Visual memory	Recognition memory for patterns
Spatial recognition memory	Visual memory	Recognition memory for spatial locations
Intra-extra dimensional set shift	Executive function	Attentional set shifting
One touch stockings of Cambridge	Executive function	Planning and working memory
Stockings of Cambridge	Executive function	Planning and motor control (Tower of London task)
Spatial span	Executive function	Working memory capacity
Spatial working memory	Executive function	working memory/strategic thinking
Choice reaction time	Attention	Alertness and motor speed
Reaction time	Attention	Simple reaction time
Match to sample visual search	Attention	Visual search and discrimination
Rapid visual information processing	Attention	Continuous performance task
Simple reaction time	Attention	Alertness and motor speed
Graded naming test	Semantic/verbal memory	Lexical access/naming
Verbal recognition memory	Semantic/verbal memory	Immediate and delayed verbal memory
Affective go/no-go	Decision making/response control	Information processing
Cambridge gambling task	Decision making	Decision making and risk taking
	Response control	
Information sampling task	Decision making	Pre-decisional contemplation
	Response control	
Stop signal task	Decision making	Inhibition of prepotent responses
	Response control	

contrast, the developmental vulnerability of executive skills was suggested by a decline in CANTAB performance among subjects in the 50–64 year old sample (Luciana & Nelson, 2002).

Reliability studies have been completed internally by Cambridge Cognition which described test-retest reliability coefficients ranging from 0.4–0.87. Those findings are consistent with stability coefficients reported by Lowe and Rabbitt (1998). Luciana (2003) reported consistency coefficients ranging from .73 for a measure of reaction time latency to .95 for performance on the self-ordered search task.

Limitations of CANTAB include its exclusion of language based measures, thus leaving the examiner to supplement the battery with additional tests to assess relevant brain-behavior issues such as those involving laterality (e.g., auditory-verbal memory). CANTAB's cost will also be prohibitive to many private practitioners and organizations.

Software requirements described by the publisher include Microsoft Windows® 2000 or Windows® XP operating systems, with minimal hardware requirements being a PC with 800 MHz Pentium III processor, 256 MB of RAM, 100 MB free disk space, sound card and speakers, CD-ROM drive, serial port and USB port. A touch screen is also required, which Cambridge Cognition offers to supply.

Future Directions

Pharmaceutical companies have been using CANTAB internationally in multi-site clinical trials for more than 20 years. The company is in the process of developing a version of CANTAB for the health care market.

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Cancellation Tests

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Synonyms

Vigilance tests

Description

Cancellation tests are designed to measure sustained and selective attention.

Historical Background

Numerous variations of cancellation tests have been developed to assess sustained, selective attention, and spatial inattention following hemispheric lesions. The patient is typically required to scan through an array of stimuli and find (cancel) specific target stimuli (e.g., bells).

Psychometric Data

There is relatively little normative data for cancellation tests, and most comparative studies show that normal individuals make few, if any, errors (Weintraub, 2000). Standardized versions of cancellation tests, such as the Ruff 2 & 7, show good test–retest reliability. In addition, on the Ruff 2 & 7 younger adults performed better than older adults and performances improved with higher levels of education. There were no gender effects (Mitrushina, Boone, Razani, & D'Elia, 2005).

Clinical Uses

Cancellation tests have been shown to be sensitive in detecting deficits in attention related to right hemisphere lesions, traumatic brain injuries, schizophrenia, and AIDS-related cognitive impairment. Cancellation tests are particularly useful in assessing visuospatial neglect. Unlike normal individuals who begin searching for target stimuli on the left side of the page and systematically move rightward, neglect patients begin on the right using a disorganized search pattern omitting many targets on the left side of the page (Weintraub, 2000). By decreasing the attentional demands of a letter cancellation test by reducing the number of nontarget stimuli, right hemisphere damaged patients showed decreased left-sided neglect, suggesting that the neglect syndrome is the result of an attentional bias (Kaplan et al., 1991).

Cross References

► Attention
► Neglect and Hemi-inattention
► Neglect Syndrome
► Vigilance

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CANTAB

► Cambridge Neuropsychological Testing Automated Battery

Capacity

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Synonyms

Competency

Definition

In the most basic sense, capacity refers to the ability to make decision(s) with regard to oneself. Specifically, this refers to one's ability to understand and appreciate the consequences of one's actions. Legal capacity remains in effect until death, unless a court rules that one is "incapacitated." If a person is ruled by a court of law to be legally incapacitated, this can remove all or part of a person's right to make decisions. Specifically, one can be deemed incapable of managing financial affairs, but ruled capable of making medical decisions, for example. If a person is ruled to lack full legal capacity, then they are prohibited from entering into a contract, giving a power of attorney, creating a will, or consenting to medical treatment. A ruling of legal incapacity typically results in the appointment of a guardian or conservator to make decisions for the person. There are several types of capacity that are relevant to forensic neuropsychology including: capacity to consent to treatment, to manage financial affairs, and others.

Cross References

► Testamentary Capacity

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Capacity Limitations

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Definition

The constraints on processing of internal and external stimuli dependent upon the structure of, and the processes related to, the cognitive system.

Historical Background

Fundamental research in attention emerged in the 1950s with information processing theories. D.E. Broadbent published an influential book in 1958, *Perception and Communication*, which was in turn influenced by the contemporary work of communication theorists in engineering. Broadbent used the communication system as a metaphor for relationships within the human brain. Each brain system (i.e., sensory, memory, response generation) was conceptualized in terms of a communication system, with an information source, a transmission channel(s), and a receiver. The channel by which a message is transmitted has a capacity, which limits the rate by which information can be transmitted. A property of communication systems is that the transmission of information within a system can never exceed the capacity of the channel divided by the information source output. This theoretical conceptualization sets the stage for future research, describing the limits in sensory and cognitive systems and mechanisms to deal with the processing of information in these systems.

Current Knowledge

In response to the observation that one is not able to attend to all stimuli in the environment at the same time, hypotheses have developed to explain the limitations on the capacity for cognitive processing, particularly in the areas of attention and memory (Broadbent, 1971; Miller, 1956; Shiffrin & Schnieder, 1977; Kahneman, 1973). Cohen (1993) organized attentional capacity in terms of structural and energetic limitations. Structural capacity limitations can be thought of in terms of

optimal processing characteristic of the system such as channel capacity, working-memory capacity, processing speed, and temporal–spatial characteristics of the system. These structural elements are influenced by variables such as performance required on more than one task (e.g., dual task): task complexity, memory demand, high or low rate of target event occurrence, and task duration. Energetic capacity limitations are considered to be natural or imposed variations within an individual that includes arousal, motivation, and generated effort. A clear indicator of the influence of energetic factors on performance is performance variability over time.

Researchers of cognition have used the debate over whether particular processes are limited or unlimited in capacity as a fruitful ground for study. For example, theories of serial and parallel processing have emerged in both the attention and memory literature in support and rejection of capacity limited processes. Although not strictly coupled, serial processing is often considered in the context of limited capacity, whereas parallel processing is thought to be unlimited in capacity. This coupling bears out in visual search studies that have found preattentive processes that are thought to be parallel and unlimited in capacity, versus focal attentive processes that are serial and limited in capacity (Duncan & Humphreys, 1989; Treisman & Gelade, 1980; Wolfe, 1994). Similarly, in memory research, Jacoby (1991) distinguished automatic from controlled processing, with similar parallel and unlimited capacity (automatic) versus serial and limited capacity (controlled) distinctions.

Future Directions

A relatively recent emphasis has been placed on the interaction of neural and cognitive limitations with factors such as arousal, motivation, and effort.

Cross References

- ▶ Attention
- ▶ Attention/Executive Function
- ▶ Parallel Processing
- ▶ Serial/Sequential Processing
- ▶ Short-Term Memory
- ▶ Span Test
- ▶ Working Memory

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CAPD

▶ Central Auditory Processing Disorder

Capgras Syndrome

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Synonyms

Reduplication delusion; Reduplicative paramnesia

Short Description or Definition

Delusions are defined as false beliefs based on incorrect inference about external reality, and firmly sustained in spite of the opinions of others or contrary evidence (American Psychiatric Association, 1987).

Capgras syndrome (Capgras & Reboul-Lachaux, 1923) is a type of reduplication delusion involving the belief that persons well-known to the patient, such as family members, have identical doubles or are imposters. The double or imposter is sometimes perceived as differing

slightly in some physical characteristic from the "genuine" person, but the patient may have difficulty verbalizing the precise nature of this "difference."

Categorization

Several variations of Capgras syndrome have been identified. *Doppleganger* or *subjective doubles* is the belief that the patient himself has a double or impersonator (Christodoulou, 1978). *Fregoli syndrome* is the belief that a person is capable of taking on the appearance of others, while retaining his/her own psychological identity (Courbon & Fail, 1927). *Intermetamorphosis* is the belief that people are changing in both physical and psychological identity to become another person (Courbon & Tusques, 1932).

Capgras syndrome must be distinguished from purely perceptual or hallucinatory disturbances, and from generalized disturbance of cognition. That is, in order to be properly and convincingly diagnosed as a delusion, the disturbance must involve a mistaken belief (not merely a misperception) and must be persistent (not a transitory effect of confusion). For example, in *autoscopy*, the patient experiences a second self, as in the subjective doubles variant of Capgras. However, the phenomena differ in that the double is actually *seen* in autoscopy, rather than believed to be active elsewhere, as in subjective doubles. The prosopagnosic person may fail to recognize his wife, whereas the Capgras patient will insist that the present person is an imposter and that the "real" wife is somewhere else. Patients in confusional states or dementia may express strange beliefs, but the beliefs typically change from hour to hour and do not persist once the confusion resolves. Thus this problem should not be termed delusional.

Patients with Capgras syndrome are usually described as forthcoming and cooperative. Although they insist that their delusional beliefs are true, they often admit to puzzlement or bemusement regarding aspects of the delusion. Rather than escalate their defenses by becoming hostile, they are more likely to confabulate an explanation. For example, a patient with a delusion of duplication (Malloy, 1991) was asked how she could have two sets of children with identical names. She appeared momentarily puzzled and then stated, "My husband was in the Navy, and we moved around a lot; it was hard to keep that straight."

Epidemiology

Dohn and Crews (1986) observed that Capgras syndrome was frequently overlooked in psychiatric patients and found that the delusion actually had a 15% incidence in their sample of adult inpatients previously diagnosed as having schizophrenia. Several researchers have found that about 25% of Alzheimer patients display delusions involving misidentification of people (e.g., Mendez, Martin, Smyth, & Whitehouse, 1992). Large-scale epidemiological studies are lacking to date.

Natural History, Prognostic Factors, Outcomes

About 58% of Capgras patients who receive adequate neurodiagnostic workups are found to display primary psychiatric disorder, uncomplicated by demonstrable neurologic disease (Dohn & Crews, 1986). Although psychological factors can be important in the production of delusions, a critical review of the literature by Malloy and Richardson (1994) demonstrated that delusions can also result from identifiable neurologic disease, from generalized disturbances to focal lesions.

They found that Capgras and its variants have been reported in association with a variety of systemic diseases and diffuse neurologic disorders. Systemic etiologies have included: Metabolic disturbances such as myxedema, pseudohypoparathyroidism, anemia, hepatic dysfunction, B12 deficiency; intoxication and reactions to drugs including cocaine, chloroquine, disulfiram, digoxin, and lithium; cerebral infections such as encephalitis and AIDS; subarachnoid hemorrhage; migraine; post-ECT confusion; minor head trauma; and degenerative dementia.

In terms of focal lesions such as tumors and stroke, that review demonstrated that the right hemisphere or bilateral lesions were invariably found on neuroimaging, with no exclusively left-hemisphere lesions. EEG and neurologic exam findings also implicated right-hemisphere pathology. In terms of specific localization, 72% of cases with CT or MRI scans had right frontal, temporal, or frontotemporal involvement. Neuropsychological testing documented spatial, executive, and nonverbal memory problems, consistent with the right frontotemporal localization on neuroimaging studies.

Neuropsychology and Psychology of Capgras Syndrome

Capgras and its variants represent either underidentification or overidentification of the object of the delusion (Vie, 1930). Thus, in Capgras syndrome, the patient

mistakenly perceives the person as unfamiliar due to *underidentification*. In Fregoli syndrome, on the other hand, the patient misperceives diverse persons as the same person due to *overidentification*.

Feinberg and Shapiro (1989) emphasized the importance of the right temporal lobe in producing misidentification delusions. They reviewed the evidence from stimulation and seizure studies indicating that the right temporal lobe plays an important role in producing the experience of familiarity. Cutting (1991) has put forth a similar argument regarding the role of the right hemisphere in identification.

Crucial factors in the persistence of delusions may be the length of time the perceptual distortion continues, and the ability of the patient to correct the misperception on the basis of new information. Frontal lesions may impact on the latter self-corrective function, making it impossible to resolve conflicting information, resulting in unconcern and confabulation when the conflicts are confronted (Joseph, 1986). Alexander, Stuss, and Benson (1979) were the first to report a Capgras delusion clearly related to a specific neurologic structural lesion, involving the right hemisphere with predominantly frontal and temporal lobe damage. They noted the importance of frontal damage in Capgras, both in terms of the inability to resolve conflicts, and confabulation of a second persona.

Psychodynamic or functional interpretations for the development of delusions are not incompatible with this neuropsychological explanation.

Evaluation

Careful clinical interview with both patient and family members will help to elicit evidence of Capgras delusions. In the course of family disputes and initial evaluations, patients may learn to minimize or deny their delusions. Demented patients may forget instances of misidentification. Hence, family informants may be extremely helpful. The literature is replete with case reports positing a psychodynamic explanation for Capgras, but with no workup to rule out neurologic etiology. Since Capgras is commonly associated with neurologic illness; full workup including neuroimaging and neuropsychological testing is essential. Neuropsychological testing often reveals deficits in frontal/executive and visuospatial functions (Malloy & Richardson, 1994). Facial recognition and memory should be tested to rule out alternative explanations for the patient's problems, such as prosopagnosia.

Treatment

In degenerative dementia, duplication delusions are usually transitory phenomena, occurring in the early-to-middle stages and disappearing when cognitive deficits become severe. In other etiologies such as cerebrovascular disease, they often occur acutely and persist for many months or years. Prognosis appears to vary with the type of delusion and the underlying etiology.

Many underlying systemic causes (e.g., infections, toxic reactions, metabolic disturbances) of duplication delusions are readily treated, resulting in elimination of the delusion. For example, Santiago, Stoker, Beigel, Yost, and Spencer (1987) reported resolution of Capgras following treatment of underlying thyroid disease. Spontaneous resolution of Capgras delusions has also been reported (Ruff & Volpe, 1981).

On the other hand, Joseph (1987) described a patient whose chronic psychosis and intermittent psychotic misidentification of the Capgras and intermetamorphosis types were refractory to neuroleptic treatment. Upon administration of a trial of clorazepate, complete remission of psychotic symptoms was achieved for the first time in 19 years, but these recurred when the patient discontinued her clorazepate.

The effectiveness of psychological interventions may vary with the type of delusion, and the degree to which neurologic factors are involved. Unfortunately, there has been no systematic treatment follow-up research, and data are limited to uncontrolled case studies.

Cross References

► Reduplicative Paramnesia

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Capital Punishment

► Death Penalty

CARB

► Computerized Assessment of Response Bias

Carbamazepine

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Generic Name

Carbamazepine

Brand Name

Tegretol, Carbatrol

Class

Anticonvulsant

Proposed Mechanism(s) of Action

Carbamazepine is a use-dependent blocker of voltage-sensitive sodium channels, it interacts with the open channel conformation of voltage-sensitive sodium channels, it interacts at alpha pore-forming subunit of voltage-sensitive sodium channels, and inhibits release of glutamate.

Indication

Complex partial seizures, generalized tonic–clonic seizures, mixed seizure patterns, and trigeminal neuralgia pain.

Off Label Use

Glossopharyngeal neuralgia, bipolar disorder, psychosis, schizophrenia, and personality disorders.

Side Effects

Serious

Aplastic anemia, agranulocytosis, Stevens Johnson Syndrome, cardiac problems, induction of psychosis or mania, and increased frequency of seizures.

Common

Sedation, dizziness, confusion, headache, nausea, and vomiting.

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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

Carbon Monoxide Poisoning

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Synonyms

Carboxyhemoglobinemia

Short Description

Carbon monoxide (CO) is a colorless, odorless gas generated during the combustion of carbon-containing fuels. When inhaled into the lungs, it readily competes with oxygen for binding sites on hemoglobin. The affinity of carbon monoxide binding to hemoglobin is more than 200-fold greater than that of oxygen. Thus, at atmospheric concentrations as low as 0.1%, carbon monoxide will achieve a 50% saturation of the hemoglobin, resulting in carboxyhemoglobinemia and a significant reduction in the blood oxygen levels.

Acute carbon monoxide poisoning, for example, following exposure to automobile exhaust (which generates about 5% to 7% carbon monoxide) will rapidly saturate the hemoglobin and cause death within minutes with virtually no prior symptoms.

Epidemiology

Exposure to low concentrations of carbon monoxide consequent to the operation of faulty furnaces or gas-powered engines is the leading cause of accidental poisoning in the United States. Exposure to high concentrations of carbon monoxide is the most common form of intentional poisoning in the United States.

Exposure to carbon monoxide is the most common form of poisoning worldwide. With increased awareness of its potential dangers, the majority of these exposures can be prevented.

Natural History, Prognostic Factors, Outcomes

Compared to normal oxyhemoglobin (i.e., when oxygen binds to hemoglobin), carboxyhemoglobin is bright red; thus, an early sign of carbon monoxide poisoning is a cherry-red skin color.

Acute exposure to lower concentrations of carbon monoxide will result in a slowly developing hypoxia that triggers peripheral vasodilation. Paradoxically, when there is a slow increase in carboxyhemoglobin saturation, compensatory changes in respiratory rate may lag. Thus, the symptoms of dizziness, weakness, headache, and nausea will precede fainting. Once unconscious, increased respiration and tachycardia will be followed by convulsions and coma, and death will ensue as the carboxyhemoglobin climbs and remains above 50% saturation.

Chronic exposure to low concentrations of carbon monoxide can occur in heavy smokers as well as in individuals whose occupations involve protracted exposure to exhaust fumes. The binding of carbon monoxide to hemoglobin is fully dissociable though these individuals may manifest carboxyhemoglobin saturation at 10%, which is 20-fold higher than normal. Symptoms associated with chronic exposure include headache, fatigue, nausea, difficulty in concentrating, and impaired memory (Kao & Nanagas, 2005).

Neuropsychological Assessment

The Carbon Monoxide Neuropsychological Screening Battery (CONSB) has been developed to screen for cognitive impairment following acute carbon monoxide poisoning (Messier & Myers, 1991). The CONSB battery begins with an assessment of general orientation of the patient, and this is followed by the Digit Span, Trail Making, Digit Symbol, Aphasia Screening, and Block Design tests. Use of this battery in an emergency room setting allows for the early detection of cerebral impairment in the exposed individuals.

Evaluation

The long-term consequences of acute carbon monoxide poisoning may, in part, be consequent to the damage to

the blood–brain barrier during the period of hypoxic coma. Following apparent recovery from the acute exposure, a delayed toxicity with abrupt onset may appear with symptoms of confusion together with motoric symptoms resembling Parkinson's disease (incoordination, muscular weakness, and muscular rigidity). These neurological signs reflect extensive damage to the basal ganglia and white matter of the brain with a demyelination that spares the neuronal axons. Neuroimaging scans will most likely appear normal 24 h after exposure; lesions may begin to appear 2 weeks later. Functional changes associated with these later-appearing lesions are variable but attempts have been made to relate these to the severity of the initial exposure. Acute exposure resulting in carboxyhemoglobin saturation of 25% or more will result in later onset cognitive impairments in as many as 50% of the patients. The cognitive deficits include agnosia, aphasia, and apraxia as well as impaired memory, impaired executive function, and a general decrease in intellect. In addition, visuomotor performance deterioriates. Finally, late-appearing changes in affect are frequently reported as are obsessive–compulsive tendencies and anxiety (Hopkins & Woon, 2006).

Treatment

Effective treatment entails removal from the source of the carbon monoxide and providing oxygen; when breathing air, the half-life of carboxyhemoglobin is about 5 h and this time can be reduced to 1.5 h with oxygen supplementation. Indeed, supplementation with oxygen immediately after exposure remains as the most effective strategy for reducing the severity of the later-appearing neural and functional consequences following carbon monoxide poisoning (Prockop & Chichkova, 2007).

Cross References

► Neurotoxins

References and Readings

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Carboxyhemoglobinemia

► Carbon Monoxide Poisoning

Cardiac Ultrasound

► Echocardiogram

Career Counseling for Individuals with Disabilities

► Vocational Counseling

Career Maintenance

► Job Retention

Carotid Angiography

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Synonyms

Angio

Definition

Angiography is the evaluation of the blood vessels of the central nervous system and associated

cervicocerebral vasculature applying radiography to the simultaneous injection of intravascular contrast media. Femoral or axillary nonselective approaches can be used to catheterize the aortic arch or selective means employed to catheterize the carotid artery. Contrast media is then injected through the catheter and X-rays taken. Digital subtraction, computed tomography (CT) scanning, and MRI techniques too can be applied at this point. Obstructions, stenosis, aneurysms, and A-V malformations can all be identified. Finer and more selective views are realized by using microcatheters. Some of the disease entities studied include ischemic cerebrovascular disease, aneurysms, vascular malformations, neoplasms, and brain injuries. Two special carotid lesions could be evaluated with carotid angiography. Arterial dissections develop when blood is extravasated within the arterial wall itself narrowing the arterial lumen. The carotid artery between C2 and the base of the skull is frequently a target for the formation of a pseudoaneurysm. Pain, bruits, with cranial nerve palsies will be present. Angiography will be useful. Another condition where carotid angiography is useful is the carotid-cavernous fistula – a high pressure, high-flow connection between the carotid artery and the cavernous sinus veins. Patients will present with visual loss, a bruit, proptosis.

Current Knowledge

Can be part of the evaluation process of patients with cerebrovascular disease.

Cross References

- ▶ Angioma
- ▶ Glioma
- ▶ Hemangioma
- ▶ Hemiplegia

References and Readings

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Carotid Artery

▶ Internal Carotid Artery

Carotid Endarterectomy

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Synonyms

CEA

Definition

CEA is an elective surgical procedure that removes plaque from the lumen of the carotid artery. After anesthesia, the surgeon clamps the carotid artery proximal and distal to the stenosis temporarily. The surgeon may place a shunt proximal to the clamp to reroute blood to the brain. Next, an incision is made over the area of blockage. The plaque is scraped out and separated from the inner lining of the artery. After it is removed, the artery is sutured together, the clamps are removed, and any bleeding is stopped. Finally, the skin incision is closed.

Current Knowledge

The arch of the aorta gives off three branches: the innominate, the left common carotid, and the left subclavian. The innominate artery, also known as the brachiocephalic artery, gives rise to the right subclavian artery and the right common carotid artery. The common carotid arteries bilaterally split into internal and external carotid arteries. The right and left internal carotid arteries, along with the vertebral arteries, branches of the subclavian artery, are the major blood vessels supplying the brain.

As plaque builds in the carotid artery, atherosclerosis, or hardening of arteries, develops. Carotid stenosis, or narrowing of the carotid artery, may also develop and is most common at the origin of the internal carotid artery or less commonly, at the distal common carotid artery.

If the stenosis is severe, it may result in decreased perfusion of brain tissue, and consequently ischemia. Another mechanism by which ischemia can develop is by the development of a thrombus over the plaque. When this occurs, an embolus may break off and may result in the occlusion of a vessel distally.

A stroke may develop if the brain is deprived of its blood supply, resulting in a sudden loss of neurologic function. However, transient ischemic attacks (TIA) may be the diagnosis if symptoms, such as loss of sensory or motor function in the extremities, inability to comprehend or initiate speech, or loss of vision in one eye described a shade coming down over one eye (amaurosis fugax), last for a few minutes to hours and resolve completely in twenty-four hours. Patients with a history of stroke or TIA are at increased risk of a subsequent stroke and should be further evaluated with imaging studies. According to the North American Symptomatic Carotid Endarterectomy Trial (NASCET), patients with symptomatic carotid artery stenosis of greater than 70% have a 26% risk of recurrent stroke over two years.

The diagnosis of a TIA is made by carotid duplex ultrasound. This type of imaging bounces high-frequency sound waves off blood vessels and the blood within the lumen to determine blood flow and any abnormalities within the vessels themselves. Other modalities useful in the diagnosis of a TIA include computed tomography angiography (CTA), magnetic resonance angiography (MRA), and angiography. In each of these, a contrast dye, or gadolidium in the case of MRA, is injected into the arteries. This helps identify any areas of poor blood flow and determine the degree of stenosis.

If there is greater than 70% blockage of the carotid artery, a CEA is recommended if the patient is symptomatic with a history of TIA or nondisabling stroke, or even if the patient is asymptomatic. However, a CEA should not be performed during a stroke or TIA or if the patient experiences a stroke that leaves him severely disabled. If there is less than a 70% blockage and the patient is asymptomatic, a CEA is not recommended. In this case, the patient should be treated medically with a baby aspirin daily. In addition, other risk factors that may cause further damage to the vessels, such as smoking, diabetes, hypertension, and hypercholesterolemia, should be treated medically. A CEA is only recommended for 50–69% stenosis if the patient has had recurrent TIAs unresponsive to medical management.

For patients that are high risk for the CEA, other surgical interventions, such as angioplasty and stenting, can be considered. During this procedure, a catheter is inserted through the groin and is guided until it reached the site of stenosis. After the location of the catheter is confirmed, a balloon inflates and attaches a metal-mesh stent into the artery. The balloon is then deflated and the catheter is gently removed. However, long-term studies have not proven this method to be superior to a CEA.

Cross References

- ▶ Ischemia
- ▶ Magnetic Resonance Angiography
- ▶ Stroke

References and Readings

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CARS

- ▶ Childhood Autism Rating Scales

CAS

- ▶ Cognitive Assessment System

Case Coordination

- ▶ Case Management

Case Management

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Synonyms

Case coordination; Case planning

Definition

"Case management" is a widely used (and often misused) term that has become commonplace in health care and human services. In its most professional use, case management is a collaborative process that assesses, plans, implements, coordinates, monitors, and evaluates the options and services required to meet the patient's health and/or human service needs. It is characterized by advocacy, communication, and resource management and promotes quality and cost-effective interventions and outcomes.

Current Knowledge

In recent years, case management has become a freestanding profession, but it is actually an overlay upon one's core profession. The certified case manager (CCM) must first hold a professional license or certification in a recognized health care or human service profession before he or she is eligible for case management certification. The core profession must involve a postsecondary degree in a field that promotes the psychological, medical, or vocational well-being of the person being served. The license or certification that follows the degree in one's core profession must be one that is required by law for independent practice without the supervision of another licensed professional. Most CCMs are graduate-trained in rehabilitation counseling, rehabilitation or occupational health nursing, or clinical social work.

The essential functions of a case manager include assessment and coordination; development, monitoring, implementation and evaluation of the rehabilitation plan; and achievement of outcomes. It is worth noting that case managers are managers in a very real sense. Generally speaking, management theory states that the primary functions of a manger are to plan, organize, direct, and control. This is precisely what case managers do but not to patients or "cases." People with disabilities often find the term, "case manager," to be pejorative as in, "I am not a case and I will not be managed." Case managers do not manage patients, but instead, they manage the rehabilitation process. Indeed, the planning, organizing, directing, and controlling of the rehabilitation process from the initial point of contact to the realization of outcomes is the ultimate mission of the case manager. For example, life-care planning (► Life Care Planning) is often depicted as the ultimate case management plan because of its inherently comprehensive nature. It is hard to conceive of a successful life-care planner who is not a professional case manager.

Another appropriate way to understand case management is to review the core body of knowledge for CCMs. These include: case management concepts, principles, and strategies; psychosocial and support systems; health-care management and service delivery; health-care reimbursement; and vocational concepts and interventions. Case management requires one to go well beyond the direct patient services of one's core profession and intervene in the systems, services, and supports available in the community to achieve intended outcomes. For example, it is not unusual for a case manager to work with multiple stakeholders in a single case including payers, service providers, family members, attorneys, employers, advocacy organizations, and, of course, the patient. The case manager sees all of these parties as important and is keenly aware that any single individual in this system has the wherewithal to completely sabotage a well-conceived plan if neglected or marginalized. In essence, the case manager is not only the primary architect of the rehabilitation plan, but must also sell that plan to all of the key stakeholders in order to maximize the plan's implementation and outcomes.

"Case management" is among the most misused terms in health care. Because it is unprotected by most state departments of professional regulation, the term is often used in reference to discharge planners, utilization reviewers, claims adjusters, and a host of paraprofessionals. However, CCMs are professionally prepared and evaluated along the lines described above. They are also held to strict scope-of-practice guidelines, ethics, and continuing education requirements.

Cross References

► Life Care Planning
► Recommendation

References and Readings

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Case Planning

► Case Management

CAT Scan

► Computed Tomography

Catastrophic Condition

► Catastrophic Reaction

Catastrophic Reaction

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Synonyms

Catastrophic condition

Definition

This term coined by Goldstein (1948) describes acute distress, agitation, and disorganized behavior occurring when overwhelmed by a situation with which the person cannot cope, typically following brain injury or other neurological impairment. Rapid and extreme anxiety, depression, and frustration can result when a person becomes over-stimulated or overwhelmed with a task that has become too difficult or is perceived as being too difficult. Typically, persons experiencing catastrophic reactions are not fully aware of their increasing lability and cannot respond to logic and reason, but they benefit from decreased demands and calm reassurance. Catastrophic reactions were initially thought to be associated only with dominant hemisphere damage and accompanying language impairment, but they can follow any neurological problem that interferes with adaptive coping and executive functioning.

Cross References

► Agitation
► De-Escalation
► Executive Functioning
► Frustration Tolerance

References and Readings

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Catatonia

► Catatonic Behavior

Catatonic Behavior

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Synonyms

Catatonia

Short Description

Catatonic behavior is characterized by marked disturbances in psychomotor movements that occur within the context of a psychiatric or medical condition. According to the Diagnostic and Statistical Manual of the Mental Disorders–Fourth edition (DSM-IV TR) catatonic features are identified, or the catatonic subtype is recognized, when there are at least two of the following:

- Motoric immobility as shown during catalepsy (including waxy flexibility) or stupor
- Excessive motor activity (apparently purposeless and independent of external stimuli)
- Extreme negativism (motiveless resistance to instructions, maintenance of rigid postures against attempts to be moved) or mutism
- Peculiarities of voluntary movement as shown during posturing (voluntary assumption of inappropriate or bizarre postures)
- Stereotyped movements, prominent mannerisms, or prominent grimacing
- Echolalia (repetition of a word or phrase just spoken by another) or echopraxia (repetitive imitation of movements of others).

Categorization

The DSM-IV TR lists three conditions during which catatonic features may be observed:

1. Schizophrenia – Catatonic Subtype (295.20)
2. Mood Disorder – Catatonic Features Specifier
3. Catatonia due to a General Medical Condition (293.89)

   Catatonic subtype of schizophrenia: This is a subtype of schizophrenia characterized by marked psychomotor disturbance that involves at least two of the six symptoms identified above.

   Mood disorder – Catatonic features specifier: The specifier "With Catatonic Features" can be applied to the current (or most recent) episode in major depressive disorder and bipolar I or II disorder when at least two of the six symptoms listed above are present.

   Catatonia due to a general medical condition: When evidence is present that catatonic behavior, presenting as at least two of the six symptoms listed above, is the direct physiological consequence of a general medical condition, the syndrome is specified as "Catatonic Disorder Due to…" and the medical condition is listed.

Diagnostically, since catatonic behavior has numerous disease causes (Gelenberg, 1976), care must be taken to distinguish the above-listed conditions from other syndromes, which may present with similar features. Specifically, DSM-IV TR notes that a separate diagnosis is not given if the catatonia occurs exclusively during the course of a delirium. Additionally, catatonic-like symptoms may occur in the context of one of the medication-induced movement disorders, such as neuroleptic-induced parkinsonism or neuroleptic malignant syndrome, or as a feature of akinetic parkinsonism, malignant hyperthermia, locked-in syndrome, stiff-person syndrome or elective mutism (Taylor & Fink, 2003).

Epidemiology

Catatonia is currently rare in North America and Europe; however, reports of its prevalence vary widely among studies. Bleuler's initial estimates of catatonic symptomatology among schizophrenia patients were significantly higher than those that are found today. Numerous surveys of catatonia among psychiatric patients using duration criteria of several to 24 h have yielded prevalence rates ranging from 7.6% to 38% (Taylor & Fink, 2003). When including studies in which systematic rules of examination are used and catatonia rating scales are applied this range narrows from 7% to 15% (Fink & Taylor, 2006). Although numerous reports of catatonia among children and adolescents have been documented, particularly in autistic and pervasive developmental disorders population, the syndrome appears to be more common in adults with prevalence rates for children and adolescents ranging from approximately 1–17% (Weder, Muralee, Penland, & Tampi, 2008).

Natural History, Prognostic Factors, Outcomes

Catatonia was first formally described in the late nineteenth century by the German physician, Dr. Karl Ludwig Kahlbaum, as a disorder characterized by mood symptoms, stupor, confusion, and eventually, dementia. Additionally, he noted unusual motor symptoms and carefully documented the character and course of the illness. Subsequently, Kraepelin classified patients who exhibited symptoms of catatonia within his *dementia praecox* disorder and Bleuler, adopting this formulation, interpreted catatonic symptoms as features of his newly named "schizophrenia." This model of catatonia as a

subtype within schizophrenia persisted through early and recent versions of the DSM. Only with the publication of DSM-IV was consideration given to catatonia occurring within the context of other psychiatric and medical conditions. This was due to important articles published in the 1970s, which established co-occurrence of catatonia and mood disorders (Abrams & Taylor, 1976) and emphasized the association between catatonia and neurological or general medical conditions (Gelenberg, 1976). Several reports have also suggested the presence of a genetic form of catatonia not captured by DSM-IV TR criteria (Taylor & Fink, 2003). Thus, the conceptualization and classification of catatonia continues to evolve.

An additional area of controversy involves categorization. Advocates have argued for DSM reclassification of catatonia into a syndrome rather than a subtype of schizophrenia. They claim that the current model fails to adequately recognize catatonia in other psychiatric illnesses and limits treatment of catatonic behavior to protocols focusing on antipsychotic drugs. Fink and Taylor (2003, 2006) have therefore proposed a change in nomenclature, suggesting that catatonia be reclassified separately from other syndromes, akin to delirium or dementia, with three syndrome subtypes (*nonmalignant*, *delirious*, and *malignant*) and four specifiers (secondary to: *mood disorders, general medical conditions or toxic states, neurological disorders, or psychotic disorders*). However, this reclassification system remains controversial.

Numerous risk factors for catatonia have been reported including history of perinatal infections, epilepsy, medication effects, and frontal or basal ganglia diseases (Weder et al., 2008). Additionally, various aspects of comorbid psychiatric conditions including age of onset and severity of symptoms are considered risk factors and contribute significantly to prognosis. Although highly variable, overall prognosis appears to be relatively better for recurrent catatonia and mood disorders with catatonia as compared with catatonic schizophrenia. One review study indicated that while acute treatment prognosis is excellent, long-term prognosis depends on the underlying condition that elicited catatonia (Taylor & Fink, 2003).

Neuropsychology and Psychology of Catatonic Behavior

Although the pathophysiology of catatonia remains poorly understood, both the GABA and dopaminergic neurotransmitter systems have been implicated in numerous psychopharmacological studies. Neuroimaging studies have not been conclusive and have revealed various cortical and subcortical brain regions to be associated with the illness, a finding that may explain observed clinical differences in symptom presentation (Weder et al., 2008). Neuropsychological evaluation of catatonic psychiatric inpatients as compared with a psychiatric control group revealed relatively poorer performance on measures of working memory and those visual–spatial abilities related to right parietal functioning. Additionally, significant differences between catatonic patients and healthy controls were observed on attentional and executive tests associated with frontal functioning. These results suggest attentional-motor and frontoparietal dysfunction in catatonia (Northoff, Nagel, Danos, Leschinger, Lerche, & Bogerts, 1999).

Evaluation

Although DSM-IV requires two of the six symptoms of catatonic behavior listed previously in order to establish presence of catatonia, definitions of catatonia, as well as its basic signs and symptoms, are still the subject of debate and no authoritative set of criteria has been universally accepted (Caroff & Ungvari, 2007). Several rating scales have been developed to assess for the presence of catatonia; however, the number of signs and symptoms that are included range from 10 to 40 for almost all instruments (Taylor & Fink, 2003). Several reviews have listed the most commonly listed signs and symptoms of the syndrome as mutism, posturing, negativism, staring, rigidity, echophenomena, stereotypes, grimacing, and perseveration (Weder et al., 2008). In terms of the number of symptoms required to justify a diagnosis, minimum thresholds vary among scales. While some scales resemble the DSM-IV TR in requiring the presence of 2 or 3 symptoms out of a list of 11 or 12 (Lohr & Wisniewski, 1987; Rosebush, Hildebrand, Furlong, & Mazurek, 1990), other instruments assign greater weight to certain cardinal symptoms over other secondary symptoms (Bush, Fink, Petrides, Dowling, & Francis, 1996). Other scales group symptoms by category (i.e., motor, affective, and behavioral) and require a symptom from each category in order to establish a diagnosis (Northoff et al., 1999). Most authors accept a range from several to 24-hours duration as being necessary in order to definitively establish the presence of catatonia (Weder et al., 2008). Attempts have been made to identify individual clusters within the catatonia syndrome based on discrete symptom-groups and duration of illness (acute/chronic); however, further research in this area is

necessary before the existence of subtypes can be established (Weder et al., 2008).

Treatment

Treatment for catatonic symptoms differs depending on the underlying cause. Catatonia is currently viewed by most clinicians from a predominantly biological framework (Penland, Weder, & Tampi, 2006). Generally, benzodiazepines such as lorazepam are considered to be the first treatment of choice. If patients fail their initial trial, the use of bilateral electroconvulsive therapy is usually considered. Although these are the best-studied treatments of the syndrome, the efficacy of other approaches, such as transcranial magnetic stimulation, continues to be explored. Catatonic schizophrenia may be treated by a variety of pharmacotherapeutic and psychotherapeutic methods. Hospitalization may be necessary to protect the patient's safety and supportive psychotherapy and family education may help patients and their families adjust to problems created by the illness. Other supportive services as sheltered workshops and special education may also be helpful. When catatonic symptoms are due to a mood disorder, episodes may be treated with mood stabilizers or antidepressant medications. Catatonic symptoms caused by a medical disorder require correct diagnosis of the underlying medical condition, followed by appropriate treatment. For example, levodopa and amantadine (symmetrel) have shown some effectiveness in reducing catatonic symptoms due to postencephalitic Parkinson's disease. Hospitalization and careful supervision of persons with catatonic symptoms may be necessary to ensure that they do not harm themselves or others and to prevent malnutrition, exhaustion, or fever.

Cross References

- ▶ Delusion
- ▶ Mood Disorder
- ▶ Schizophrenia

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Catecholamines

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Synonyms

Adrenergic agonists; Direct- and indirect-acting adrenergic receptor agonists; Dopamine agonists; Dopaminergic agonists; Sympathomimetic amines/drugs/agents/compounds

Definition

Catecholamines are a class of biologically active water soluble hormones comprising *catechol* and *amine* compounds. They are derivatives of the amino acid tyrosine. Tyrosine is converted from phenylalanine as a function of the hydroxylation by the enzyme phenylalanine hydroxylase, and can also be directly ingested from dietary proteins (Catecholamines, 2002; Catecholamines, 2008; Catecholamines, 2009a, b).

Catecholamines are sympathomimetic amines, a group of compounds including dopamine, epinephrine, and norepinephrine whose molecular structure is similar to that of a larger class of neurotransmitters, the monoamines. The specific molecular structure of catecholamines is a benzene ring with two hydroxyl groups, an intermediate ethylamine to the side and an amine terminal group. The chromaffin cells in the adrenal medulla and sympathetic nervous system postganglionic fibers are the primary site of catecholamine production (Hoffman, 2004).

During times of physiological and/or psychological stress neurons in the central and peripheral sympathetic nervous systems secrete catecholamines via hypothalamic and adrenomedullary activation. Catecholamine toxicity, also referred to as catecholamine dump or storm can occur from trauma, causing over-stimulation, excessive production and circulation of catecholamines in the central nervous system, and/or lesions of nuclei affecting the sympathetic nervous system in the brainstem.

Catecholamines affect metabolic rate, temperature regulation, smooth muscle functions, cardiovascular and nervous systems. Epinephrine and norepinephrine autonomically prepare the body for crisis such as cold, fatigue, danger, and shock. Dopamine is produced as an intermediate process in the synthesis of epinephrine and is critical to the regulation of many neurological processes, including smooth fine and gross motor movements, energy regulation, motivation, executive functioning and formal thought regulation. Sympathomimetic drugs used to treat conditions associated with these biological functions mimic the actions of epinephrine, norepinephrine or dopamine (Sympathomimetic drug, 2008; Venes, Thomas, Egan, Houska, 2001). Such drugs include those used to treat a variety of abnormalities associated with central and sympathetic nervous system processes (e.g., blood pressure, cardiac anomalies, premature labor, glaucoma, bronchitis, asthma, emphysema, blood glucose metabolism, movement disturbances and psychosis). In addition, numerous disorders involve catecholamine disturbances of congenital, genetic, or familial origin such as dopamine-β-hydroxalase deficiency, paraganglioma syndrome, and tetrahydrobiopterin deficiency, but also those with unknown precipitating mechanisms, including chemodectina, neuroblastoma, and pheochromocytoma (Catecholamines, 2002).

Catecholamine-Related Conditions

Benign Familial Tremor/Essential Tremor is an inherited autosomal dominant condition suggestive of excessive systemic adrenergic activity. It is the most common involuntary movement disorder and tends to develop during middle adulthood. Anxiety can precipitate and exacerbate the amplitude of the tremor. It is generally treated with beta blockers such low doses of propranolol and with lesser efficacy with Atenolol and anxiolytics such as alprazolam (Kaufman, 2007).

Chemodectoma (nonchromaffin paraganglioma) – Benign chemoreceptor system tumors. The most common types are glomus jugulare tumor and carotid body tumor.

Dopamine-β-hydroxalase Deficiency – Severe orthostatic hypotension of a congenital nature caused by the inability to generate the enzyme, dopamine-β-hydroxalase.

Familial Paraganglioma Syndrome – Genetic slow-growing chromosomal (11q23) benign tumors primarily of the head and neck. These unusual conditions are inherited from the father. Disfiguring facial swelling, hearing loss, tinnitus, pain, persistent cough, or other head/neck anomalies as a function of cranial nerve damage from the tumor.

Neuroblastoma, produces catecholamines, and following brain tumors is the second most common solid tumor in childhood. Testing for the catecholamine metabolites, vanillylmandelic acid and homovanillic acid, are identified in the urine.

Pheochromocytoma is a benign tumor that secretes norepinephrine and epinephrine. It originates in sympathetic paraganglia or adrenal medulla.

Tetrahydrobiopterin deficiency is a genetic condition that results in the inability to manufacture the enzymes required to synthesize catecholamines. This leads to a deficiency of the neurotransmitters, norepinephrine, epinephrine, and dopamine (Catecholamines, 2002; Myers, 2006).

Mechanisms of Action

The adrenergic or sympathomimetic drugs act at sympathetic postganglionic terminal by activating the catecholaminergic hormones, epinephrine (adrenaline), norepinephrine (noradrenaline), and/or dopamine in various ways. Some compounds have cross-reactivity such that they act on catecholamines by more than one mechanism of action (Sympathomimetic drug, 2008).

Directly activating adrenergic postsynaptic receptors (Direct-Acting Adrenergic Agonists) include α-adrenergic agonists and β-adrenergic agonists. There are five types: α_1, α_2, β_1, β_2, and β_3. Adrenergic *agonists* stimulate one or more of these receptor types. Adrenergic *antagonists* inhibit one or more of the actions of these receptors. Some agents have stimulating and blocking actions on different receptors simultaneously. Receptor selectivity

Catecholamines. Table 1 Sympathomimetic Action & Function

Primary neurotransmitter actions	Mechanism of action	Examples of compounds	Clinical uses
Alpha (α) adrenergic agonists	Direct-acting alpha-adrenergic agonists – Promote vasoconstriction, mydriasis, body tissue-building, inhibition of endogenous testosterone release	Alpha (α_1, α_2) – adrenergic agonists	Used to treat glaucoma by decreasing the production of aqueous fluid; as vasopressors, nasal decongestants and to dilate the pupil for eye exams
α_1 Agonists	α_1 Agonists – stimulate phospholipase C activity	Oxymetazoline, phenylephrine	Used to treat anemias caused by deficient production of red blood cells, aplastic anemia, hypotension, shock, paroxysmal supraventricular tachcardia
α_2 Agonists	α_2 Agonists – inhibit adenylyl cyclase activity	Clonidine – (α_2 and imidazoline-I1 agonist)	α_2 Agonists reduce sympathetic nervous system activation. They are used to treat opiate and alcohol withdrawal symptoms, as antihypertensives, gestational hypertension, to decrease peripheral resistance and as sedatives. More recently they are being used to treat neuropathic pain, sleep hyperhidrosis, and off-label, to counter the side effects of stimulant medications
		Guanfacine – (affinity for preference for α_{2a} adrenoceptor)	
		Methyldopa is approximately 50% absorbed from the gut, metabolized in the intestines and liver and alpha-methylnorepineprine, its metabolite, stimulates central α_2- receptors	
Mixed α, β_1, β_2 agonists	Undetermined or mixed alpha- beta-stimulation	Epinephrine, amidephrine, ergotamine, norepinephrine, synephrine	Mixed alpha- beta- agonists such as epinephrine are used to treat anaphylaxis, acute asthmatic reactions, cardiac arrest, open-angle glaucoma and nasal congestion. It stimulates alpha, beta$_1$ and beta$_2$ adrenergic receptors, increasing cardiac output, dilating bronchials, skeletal muscle vasulature, and pupils dilation due to constriction of dilatory muscles
β Antagonists (beta blockers) – block the action of epinephrine and norepinephrine	Direct-acting beta-adrenergic agonists – stimulate adenylyl cyclase activity and open calcium channels	Cardiac stimulants	Beta blockers are used to treat heart block, ventricular tachcardia, cardiac arrest, asthma, chronic bronchitis, emphysema, hypovolemic and septic shock, low cardiac output or hypoperfusion, congestive heart failure
β_1 Agonists	Direct-acting β_1-adrenergic agonists	Albuterol, dobutamine	Asthma, Chronic Obstructive Pulmonary Disease (COPD), sinus congestion

Catecholamines. Table 1 (Continued)

Primary neurotransmitter actions	Mechanism of action	Examples of compounds	Clinical uses
β_2 Agonists	Direct-acting β_2-adrenergic agonists – stimulate postsynaptic β_2 receptors of the intracellular enzyme, adenylyl cyclase. Closes calcium channels, relaxing smooth muscle	Metaproterenal, formoterol, salmeterol, terbutaline	A moderately selective β_2 – agonist. It acts on smooth muscle in the lungs, uterus, and skeletal muscle vasculature. Used to treat asthma, COPD, premature labor
			The enzyme intracellular adenyl cyclase catalyzes the conversion of ATP to cAMP. Elevated cAMP levels are associated with bronchial smooth muscle relaxation and regulation of mast cells activation
Mixed β antagonists		Isoproterenol (β_1 and β_2 agonist)	Cardiogenic shock, bradyarrhythmias, heart block, cardiac arrest, brochospasm associated with anesthesia, asthma, chronic bronchitis, emphysema, hypovolemic and septicemic shock
COMT-inhibitors	Indirectly inhibiting epinephrine and norepinephrine metabolism	Entacapone, tolcapone	Adjunctive Parkinson's treatment
Dopamine agonists	Stimulate production and release at D_1 receptors	Bromocriptine, apomorphine, fenoldopam	Hypertensive crisis through vasodilation in coronary, renal, mesenteric and peripheral arteries
		Pramipexole – (pre- and postsynaptic $D_{2\ and\ 3}$ receptor agonism)	Pramipexole used to treat idiopathic parkinsonism signs. It stimulates caudate neurons by D_3 agonism
	Prolongs dopaminergic activity when combined with COMT-inhibitors and dopa decarboxylase inhibitors (e.g., benserazide or carbidopa)	Levodopa (l-Dopa)	Parkinsonianism – Levodopa's efficacy is as a prodrug. It is a precursor of catecholamines
Dopamine Reuptake Inhibitors (DRIs)	Increase norepinephrine release at central noradrenergic neurons. Release of dopamine at mesolimic areas in high doses.	Cocaine, Methylphenidate Amphetamines	Cocaine used recreationally and highly subject to abuse. Ophthalmically used as local anesthetic.
			Amphetamines and methylphenidate are used primarily to treat ADHD, narcolepsy, occasionally in geriatric depression, post-stroke and cancer to increase alertness and counteract chemotherapy lethargy. All DRIs and stimulant-acting drugs elevate mood, increase focusing ability and physical energy in most, increase respiration and heart rate, blood pressure and decrease appetite. They can cause euphoria, rebound depression and anxiety particularly if over-used or used inappropriately to treat depressive disorders

Catecholamines. Table 1 (Continued)

Primary neurotransmitter actions	Mechanism of action	Examples of compounds	Clinical uses
Dopamine antagonists	Blockade of dopamine (more D_2 than D_1 receptor affinity).	Antipsychotic agents	Schizophrenia, Bipolar I, mania
	Moderate α adrenergic and histaminic antagonism.		
Monoamine Oxidase Inhibitors (MAOIs)	Stimulate production and release of catecholamines in CNS	Phenelzine (Nardil)	Treatment resistant depression, atypical depression, panic disorder
	Chronic use downregulates α_2 or β adrenergic and serotonin receptors	Tranylcypromine (Parnate)	
Norepinephrine reuptake inhibitors	Prevent re-uptake of norepinephrine at postsynaptic receptors	Most non-SSRI antidepressants: Tricyclic antidepressants (TCAs – mixed catecholamine stimulation and inhibition) Atomoxetine (Strattera) – serotonin and norepinephrine reuptake inhibition Duloxetine (Cymbalta) Venlafaxine (Effexor) – selective norepinephrine reuptake inhibition	Atypical depression, dysthymia, anxiety, ADD, post traumatic stress disorder
Norepinephrine-dopamine reuptake inhibitor	Increase availability of monoamines, dopamine and norepinephrine, due to inhibiting re-uptake, causing a stimulating effect	Bupropion, pyrovalerone, mazindol (also classified as a TCA), phenethylamines	ADHD, depression, chronic fatigue, obesity
Norepinephrine releasers	Stimulate production and release of catecholamines	Mirtazapine (classified as a tetracyclic and a noradrenergic specific serotonergic antidepressant – NaSSA)	Depression, mixed anxiety-depression with sleep disruption
		Mianserin is a tetracyclic antidepressant	

Catecholamines. Table 1 (Continued)

Primary neurotransmitter actions	Mechanism of action	Examples of compounds	Clinical uses
Stimulants	Stimulate direct production and release of catecholamines, particularly norepinephrine and dopamine from storage vesicles	Amphetamine salts, methylphenidate, dexamphetamine, dextroamphetamine, phentermine	ADD/ADHD – increases alertness, concentration, focus, attention, executive functions, endurance mentally and physically. Diminishes verbal and physical impulsivity
			Narcolepsy – to help maintain alertness
			Major depressive disorder – occasionally used to augment antidepressant treatment
			Weight management – phentermine is used to suppress appetite
	Also increase norepinephrine and dopamine levels via reuptake inhibition and binding to the MAO protein transporter		Schedule IV controlled substances in the US
	Stimulate production and release of catecholamines	Modafinil, adrafinil	Narcolepsy – to maintain alertness, and counteract abnormal states that diminish alertness
			Night shift work – to counteract fatigue
Over-the-counter stimulants	Synthetic and plant-based herbal preparations with significant abuse potential thought to stimulate release of catecholamines	Ephedrine, pseudoephedrine, MDMA, cocaine, ephedra, Ma huang, guarana	To suppress appetite, increase alertness, enhance athletic performance
			Cocaine, a tropane derivative, is made from South American coca bush leaves. It is occasionally used legally as an ophthalmic local anesthetic, but no longer prescribed for its stimulant properties

Aminoff (2004); Hoffman (2004); Kaufman (2007); Katzung (2004); Psychoactive drug (2009); Stimulant (2009); Sympathomimetic drug (2008); Williams and Turner (2008).

addresses strength of affinity toward a particular receptor rather than absolute specific selectivity of one receptor only.

Direct-acting dopamine agonists are used to treat hypertensive crisis and parkinsonian signs and symptoms.

Indirect-acting, norepinephrine transporter antagonists such as ephedra, cocaine, and amphetamines. They block and reverse the activity of the norepinephrine transporter proteins. These proteins functionally terminate the effects of norepinephrine and to a lesser degree, dopamine, by removing these neurotransmitters from the synapse and repackaging them in their vesicles for later use (Katzung, 2004).

Indirectly inhibiting epinephrine and norepinephrine metabolism such as COMT-Inhibitors (e.g., entacapone and tolcapone) and MAOIs such as Parnate and Nardil (Aminoff, 2004) that raise the level of all the catecholamines in the blood. Monoamine oxidase (MAO) is the enzyme primarily responsible for metabolism of epinephrine and norepinephrine. If the enzyme is inhibited, it prevents the removal of epinephrine and norepinephrine from the system which induces sympathomimetic effects. Similarly, with COMT-Inhibitors, the enzyme catechol-*O*-methyl transferase (COMT) is an intracellular enzyme in the postsynaptic neuron that degrades epinephrine and norepinephrine. This enzyme effectively deactivates them by inducing *S*-adenosyl methionine (SAM) to add a methyl group to the catecholamines. The COMT-Inhibitors prevent this deactivation by blocking the breakdown/reuptake process, and enabling the utility of the catecholamines (Catechol-O-methyl transferase, 2009).

Stimulating production and release of catecholamines as do MAOIs and amphetamines. These agents are known to increase cardiac output and blood pressure as a function of constricting peripheral blood vessels which increases the force and rate of cardiac muscle contractions. They can

also lead to elevations in blood lipid levels due to fat catabolism and blood glucose levels due to glycogenolysis of skeletal and hepatic muscle (Sympathomimetic drug, 2008). As discussed above, monoamine oxidase (MAO) is the enzyme primarily responsible for degrading catecholamines. It has a half-life of several minutes. MAOIs and psychoactive stimulants bind to the enzyme, inhibiting the break down of the catecholamines, and largely for this reason the effects of amphetamines last longer than those of cocaine or crack. Amphetamines are among the adrenergic agents with dual mechanisms of action or cross-reactivity. The production of all the catecholamines, dopamine, epinephrine, and norepinephrine is stimulated and the reuptake, particularly of norepinephrine, is inhibited.

Specific Compounds and Properties

All sympathomimetic amines can be conceptualized to be part of a broad group of stimulant drugs due to their sympathetic nervous system and/or central nervous system activating properties. However, due to multiple mechanisms of action sympathomimetics are classified accordingly and as a function of their use in the treatment of specific conditions (Table 1).

Cross References

- ▶ Adrenergic Agonists
- ▶ Dopamine
- ▶ Epinephrine
- ▶ Monoamine Oxidase
- ▶ Norepinephrine
- ▶ Sympathomimetic Amines

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Categorical Data

- ▶ Qualitative Data

Category Fluency

- ▶ Controlled Oral Word Association Test
- ▶ Verbal Fluency

Category Test

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Synonyms

BCT; CT; HCT

Description

The Category Test (CT) was first published by Ward Halstead (Halstead & Settlage, 1943). Over the years, there have been several versions of the test, all retaining the essential format of the original and the goal of measuring concept formation ability. All versions involve the presentation of visual images of different shapes to the examinee. The examinee is to determine which underlying concept is presented by the image and choose a number from 1 to 4 to identify the concept. For example, the examinee may choose the number corresponding to the number of items in the image, the proportion of the image that is drawn continuously, etc. The examinee is given feedback after each choice to indicate if he or she was correct in their decision. By a process of utilizing this feedback to monitor performance, the examinee then learns the concepts. The number of errors is used to determine the overall performance.

The original version of the test was administered by way of a drum with images on it and a slit for examinees to view them. Feedback was given with a bell (correct) or buzzer (incorrect). Sometime during the 1940s or 1950s (the exact date being unclear), the test was reduced from its original 360 items and 9 subtests to 208 items and 7 subtests. The final subtest asks the examinee to recall previously presented concepts in order to respond correctly, constituting a memory component to the test. More recent versions have included a projection of images using slides, a card format, a self-administered version, a booklet version, and various computer versions. The various versions are available from different publishers: Slide projector version (Reitan Labs), Booklet version (Psychological Assessment Resources), and Computer versions (Psychological Assessment Resources and Mental Health Systems). Feedback has varied by version and includes the original bell or buzzer, computer approximations of the bell or buzzer, or examiner feedback of "correct" and "incorrect." Test administration time is 30–45 min.

Historical Background

Halstead (1940) reported the preliminary findings of studies dating back to the 1920s using concept formation tests in which brain-damaged participants grouped objects into categories. It was clear from subjective analyses that brain damage affected this ability. Halstead hypothesized that his test measured abstraction and the process of developing sets or frames of reference on which to base approaches to new and unfamiliar stimuli. Although Halstead found his test to discriminate frontal lobe-damaged patients from others, this finding was never validated by other researchers. Various researchers have, however, identified the CT as one of the most sensitive measures of brain injury in the Halstead Reitan Battery (Choca, Laastch, Wetzel, & Agresti, 1993).

The existence of various versions of the CT has raised the issue of comparability of these newer forms with the original CT. To date, there has been no research indicating effects of CT test apparatus on the performance of examinees. Numerous studies demonstrated comparable results, regardless of the test format. Kupke (1983) pointed out that the modification of a test apparatus in a purely cognitive task, such as that presented by the CT, should have little effect on performance. However, this analysis would only pertain to versions that retain the original test stimuli and administration instructions. A self-administered version of the test that drastically changed instructions and allowed the examinee to view earlier responses does not meet this requirement. Also, various short forms of the CT have not been studied sufficiently to determine their comparability to the original length test.

A number of investigators have attempted to determine if the CT can generate more scores than just the number of errors. Factor analytic studies have indicated that there may be different abilities tapped by the test, prompting research into the possibility of developing scores for different abilities. Scales for spatial positional reasoning, proportional reasoning, perseveration, loss of set, and memory, have all been proposed, along with a measure of effort. Recent research has found concurrent validity for a number of these measures, and efforts to standardize them for clinical use continue (Minassian et al., 2003).

Psychometric Data

Demonstrated test–retest validity of the CT ranges from 0.60 in normal populations to 0.96 for brain-damaged participants. A problem in identifying retest stability for the CT has to do with the substantial learning aspect of the test. Although there has been no adequate analysis of expected gain on re-administration of the test, reliability data suggest an improvement of about 15 points for normal individuals and 25 points for brain-damaged individuals, with a retest interval of 2–3 months. Internal consistency, perhaps a better marker of reliability for this test, has been shown to be very high at 0.97, as is split half reliability at 0.98.

Numerous studies have demonstrated the CT's ability to discriminate brain damaged from normal examinees, with analyses indicating statistically significant discrimination at the 0.05 to 0.001 levels. However, utilizing only the suggested cut-off score of 50 errors to separate groups has resulted in a false positive rate of as high as 18%. The need to correct for age and education has been firmly established, as these variables account for a significant variance of CT error scores. Including these corrections significantly reduces classification errors (Heaton, Grant, & Matthews, 1991).

The original intent of Halstead to utilize the CT as a measure of frontal lobe functioning has not been validated by researchers. Also, the CT has been found to be relatively ineffective in lateralizing damage to the brain. The test, as is the case with many neuropsychological instruments, is significantly impacted by severe psychiatric disturbance and inadequate effort.

Clinical Uses

The category test is generally grouped in most textbooks in the field with other measures of "executive functioning." This concept can be loosely defined as the ability to utilize feedback to organize and plan one's approach to deal with a novel problem-solving task. Taking this definition, the CT seems to qualify as a measure of this ability. In fact, clinicians who use the test typically include it in their battery of tests as an executive functioning measure. This approach is supported by the test format and some research, but should not involve reliance on the CT as the sole measure of executive functioning. As previously discussed, the test does not identify only frontal lobe dysfunction, but is sensitive to any type of brain injury.

Unfortunately, the only validated and normed score for the CT is the total error score. The original cut-off of 50 errors should not be used without also looking at age and education adjusted scores. The sex of the examinee does not appear to have a significant effect on test performance, and although norms are frequently broken down by sex, this demographic variable can largely be ignored. The clinician can view CT performance as indicative of the overall cortical integrity of his or her patient. There is also some evidence that the test assesses fluidity of thinking and may be related to rehabilitation outcome.

Ongoing research will hopefully provide the clinician more information concerning his or her patient's abilities related to perseveration, attention, and possibly other aspects of their performance on the CT.

Cross References

- ▶ COWA
- ▶ Ruff Figural Fluency
- ▶ Tower of London
- ▶ Trail Making Test
- ▶ Wisconsin Card Sorting Test

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Caudate Nucleus

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Definition

The caudate nucleus is an arcuate mass deep into the cortical hemispheres forming a part of the basal ganglia. Apart from being involved in the smooth orchestration of

motor actions, it has been recently implicated in other functions like learning, memory, emotion, and language.

Nomenclature

Striatum/Neostriatum = Caudate Nucleus (nucleus caudatus) + Putamen
Corpus striatum (Dorsal division) = Caudate Nucleus + Putamen + Globus Pallidus

Current Knowledge

Anatomy

The caudate nucleus has a globular head, tapering body, and down curving tail. The head lies in the floor and lateral wall of the anterior horn of the lateral ventricle, whereas the body runs in the floor. Medially, the caudate nucleus abuts the thalamus; in the floor of the ventricle, this junction forms a groove known as the sulcus terminalis that lodges the stria terminalis of the choroids plexus. The corpus callosum runs above the head and body. The caudate nucleus is separated from the lentiform nucleus by the anterior limb of the internal capsule.

Histology

The caudate nucleus and putamen show similar histology. Small and large neurons (in a 20:1 ratio) with spherical or ovoid dendritic fields populate the neostriatum making connections within the nucleus or with the globus pallidus. Most small neurons contain GABA and either enkephalin or substance P. Enkephalinergic neurons express D2 dopamine receptors whereas those with substance P have D1 receptors. Large neurons with spiny dendrites contain acetylcholine esterase (AChE) and choline acetyltransferase (CAT).

Circuitry

Corticostriate, thalamostriate, and nigrostriate fibers form the major input to the striatum. Cortical areas involved in motor planning and execution project to the caudate and putamen. Striatal neurons relay to the globus pallidus that sends information to the thalamus (VA/VL), which in turn feeds back to cortical motor areas forming the "direct pathway" that increases motor activity. An indirect pathway, involving the subthalamic nucleus, decreases motor activity. The activity of these pathways is further modulated by dopamine from the nigrostrial tract and ACh from the interneurons.

Physiology and Pathophysiology

The caudate nucleus is mainly involved in motor planning and execution of smooth movement. Degeneration of the caudate is associated with dyskinesias and involuntary movements. Caudate pathology manifests in choreiform movements (brisk, jerky, and purposeless), as seen in Sydenham's chorea (minute caudate hemorrhages and capillary emboli post streptococcal infection in children) or Huntington's disease (an adult onset degenerative hereditary disorder). Athetosis (slow, sinuous, and aimless movements involving distal musculature) is also seen in striatal pathology. Loss of nigrostriatal input in Parkinson's disease leads to akinesia and rigidity characteristic of the disease.

Recently, the caudate nucleus has been implicated in functions other than motor planning. Using functional imaging, Crinion et al. (2006), have shown activation of the caudate nucleus during language processing in bilingual persons. Initial degeneration in the caudate is thought to be responsible for the dysphoria in early Huntington's disease (Paradiso et al., 2008). The caudate nucleus has also been theorized to envision positive emotional events in the near future (D'Argembeau et al., 2007) and provide complimentary information of the outcomes of actions (Lau & Glimcher, 2007).

Cross References

- ▶ Globus Pallidus
- ▶ Putamen
- ▶ Thalamus

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Causal Modeling

► Path Analysis

Cavernoma

► Angioma, Cavernous Angioma

Cavernous Hemangioma

► Angioma, Cavernous Angioma

Cavernous Venous Malformation

► Angioma, Cavernous Angioma

CBCL

► Child Behavior Checklist

CBCT

► Cognitive Behavioral Couples Therapy

CCT

► Children's Category Test

CDR

► Clinical Dementia Rating

CEA

► Carotid Endarterectomy

Ceiling Effect

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Synonyms

Ceiling level

Definition

A ceiling effect is observed when an examinee obtains very high or maximum scores on a particular test. This may lead to an underestimation of the examinee's true ability level because the test does not allow for higher levels of performance to be assessed.

Current Knowledge

A ceiling effect in a large number of participants in a normative sample may prevent the examiner's ability to gauge a particular individual's personal ceiling, or highest level of performance, when compared with others in the standardization sample because the maximum level of performance within the sample cannot be assessed.

For example, on a list-learning memory test that contains only four words, the majority of examinees will most likely recall most or all of the words. Four "bits" of information (in this case, words in a list) place very little demand on the average person's memory ability. To say that any particular examinee who learns all four words is demonstrating intact memory abilities may underestimate that examinee's actual memory span. Adding at

least five more words would allow for an investigation of that individual's upper limit of learning or memory capacity.

Cross References

► Floor Effect
► Testing the Limits

Ceiling Level

► Ceiling Effect

Center for Epidemiological Studies–Depression

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Synonyms

CES-D

Description

The Center for Epidemiological Studies – Depression Scale (CES-D; Radloff, 1977) is a 20-item self-report measure that assesses the presence and severity of depressive symptoms over the previous week. The CES-D has been used with adult and adolescents. Individuals completing the Scale rate each item on a four-point Likert scale ranging from 0 (*rarely or none of the time*) to 3 (*most or all of the time*). Four items require reverse scoring. Administration time is approximately 5 min. Responses are summed to obtain a total score of 0–60, with higher scores indicating a greater frequency of symptoms. Scores of 16 or higher were suggested to identify subjects with depressive illness (Radloff, 1977). Work by Pandya, Metz, and Patten (2005) supports the use of this cut-off in those with multiple sclerosis (MS). Earlier work by Zich, Attkisson, and Greenfield (1990) suggest that alternate cut-off scores may be more appropriate with medical populations.

Historical Background

In 1971, the National Institute of Mental Health (NIMH) began development of the CES-D to measure depressive symptoms in epidemiological research (Brantley, Mehan, & Thomas, 2000). Selection of original components of depressive symptomatolgy (e.g., depressed mood, worthlessness) was based on factor analytic studies and clinical literature (Radloff, 1977); specific items were chosen from established measures. Following minor revisions, items were added to the NIMH structured interview and used in a large-scale study (Brantley et al., 2000).

Follow-up studies support use of the CES-D with diverse populations, including those with neurological compromise. Recent efforts have also focused on creating shorter versions of this measure (e.g., Iowa, Boston) to facilitate use and decrease participant burden. Kohout and colleagues (1993) found that when assessing older adults (65 and older) shorter versions adequately evaluated the same symptom dimensions as the original measure. Findings from Carpenter et al. (1998), who pooled data from 832 women (6 population), support use of the Iowa form over the Boston.

Psychometric Data

Psychometric properties of the CES-D were initially established with members of the general population (household interview survey) and those receiving services in a psychiatric setting (Radloff, 1977). Validity (content, construct, and criterion) was confirmed via item choice and patterns of correlations with clinical ratings and alternate self-report measures. CES-D scores differentiated between psychiatric inpatients and members of the general population (Radloff, 1977): 70% of the patients versus 21% of the general population scored above 16. Internal consistency, based on coefficient alpha and Spearman-Brown split-halves method, is generally high – approximately 0.85 in the general population and approximately 0.90 in the patient sample. Consistent with the expectation that shorter test-retest intervals would produce higher correlations, test-retest reliabilities ranged from 0.32 (12 months) to 0.67 (4 weeks). Follow-up studies appear to support Radloff's (1977) original

reliability and validity statistics (Brantley et al., 2000). A four factor structure (depressed affect, positive affect, somatic and retarded activity, and interpersonal) was initially reported (Radloff, 1977), and has been supported by subsequent confirmatory analyses. However, findings suggest that among minority populations alternate models may provide a better fit (Brantley et al., 2000).

Clinical Uses

The CES-D is a widely accepted measure of depressive symptoms that has been used with a range of populations, including those with a history of neuropsychological impairments secondary to disease (e.g., MS), acquired insult (e.g., stroke), or injury (e.g., traumatic brain injury, TBI). The CES-D has been relied on to document the incidence and severity of depression in individuals with MS, including those who received disease-modifying treatments (Patten, Fridhandler, Beck, & Metz, 2003; Chwastiak et al., 2002), and to assess for potential adverse psychiatric side effects of Interferon treatment (Patten & Metz, 2001). It has also been used to explore the relationship between depression and cognitive complaints in individuals with MS (Maor, Olmer, & Mozes, 2001), and study whether depression may be a risk factor for increased mortality after stroke (Gump et al., 2005).

Confirmatory factor analysis supports the use of this measure for those with mild to moderate TBI (McCauley et al., 2006); acceptable reliability of the CES-D with individuals with TBI has also been documented (Bush, Novack, Schneider, & Madan, 2004). Moreover, Bay, Hagerty, and Williams (2007) argue that the CES-D's psychometric properties and ease of use render it an appropriate measure for depression screening in persons with TBI. The CES-D has also been used in longitudinal research on the incidence of depression following TBI (Dikmen et al., 2004) and in studies of depression in caregivers of individuals with TBI (Rivera et al., 2007).

Of note, although findings from the CES-D may be useful in identifying certain symptoms of depression, the measure only addresses mood during the week prior to administration and, therefore, does not permit the clinician to assign a formal diagnosis of a mood disorder. As such, in a clinical setting it may be best used in conjunction with additional findings (e.g., structured clinical interview, additional measures) to diagnose depressive disorders and assess treatment needs.

Cross References

- ▶ Beck Depression Inventory
- ▶ Geriatric Depression Scale
- ▶ Hamilton Rating Scale of Depression
- ▶ Structured Clinical Interview for DSM-IV (SCID)
- ▶ Zung Self-Rating Depression Scale

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Center for Medical Home Improvement

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Membership

The Center for Medical Home Improvement (CMHI) is a nonprofit organization affiliated with Crotched Mountain. (Crotched Mountain is a charitable organization employing more than 900 people, whose mission is to serve individuals with disabilities and their families, embracing personal choice and development, and building communities of mutual support. It provides specialized education, rehabilitation, community and residential support services for more than 2,000 individuals living in New England and New York.) It is founded in 1993 by Dr. Carl W. Cooley, Medical Director and Ms. Jeanne W. McAllister, R.N., M.S., M.H.A., Director. Along with the founders, Dr. Cooley and Ms. McAllister, the CMHI is staffed by Lora Council, M.D. (Quality Consultant), Lori Keehl-Markowitz, R.N., B.S.N. (Program Manager), Leah Reed (Program Coordinator), and Kathleen Sherrieb, R.N., M.S., M.P.H. (Data Management Consultant).

Major Areas or Mission Statement

The mission of the CMHI is to promote high-quality primary care in the medical home and secure health policy changes critical to the future of primary care. The CMHI defines the medical home "as a community-based primary care setting which provides and coordinates high quality, planned, family-centered health promotion, acute illness care and chronic condition management" (chronic condition management acknowledges that children, adults, and their families may require more than the usual well child and/or adult, preventive care, and acute illness interventions. It involves explicit changes in the roles of providers and office staff aimed at improving).

1. Access to needed services
2. Communication with specialists, schools, and other resources
3. Outcomes for children and families

Landmark Contributions

The "story" of the medical home extends from the 1935 Social Security Legislation Act that called for Maternal and Child Health (MCH) Title V Programs to "locate, diagnose, and treat crippled children" to today's emphasis on providing planned primary health care in the context of the families and communities in which patients live. Community-based care in a medical home is increasingly accepted as one of the means of achieving optimal outcomes for children, youth, adults, and their families. The term "medical home" was coined in 1967 by the Council on Pediatric Practice, a subgroup of the American Academy of Pediatrics, and has been described with a variety of applications and definitions since then. The CMHI began its efforts in 1993 with a capacity building endeavor for pediatric practices serving children with special healthcare needs. In 1997, the CMHI developed and implemented an improvement model with multiple New Hampshire, Vermont, and Maine healthcare teams. These teams (physician leader and practice-based care coordinator) partnered with patients and families to redesign primary care services. In 2003, the CMHI published the Medical Home Index (MHI) as a validated measurement to assess quality and monitor improvements in a primary care practice (this tool is now available for adult care). Practice improvement activities in multiple states have consistently demonstrated sustained increases of greater than 30% in MHI scores overall. A companion Medical Home *Family* Index and Survey used by these practices has shown significant improvement in clinical, functional, satisfaction, and cost outcomes and an increase in the use of written and portable care plans. During 2001–2004, the CMHI spread its model to ten additional practices in Vermont and New Hampshire and partnered with the National Initiative for Children's Healthcare Quality (NICHQ) in two national medical home learning collaboratives. These efforts "spread" the CMHI's model to 20 state Title V Programs, 40 primary care practices, and multiple family advocacy organizations. The CMHI and NICHQ

adapted the widely recognized Chronic Care Model as the "Care Model for Child Health in a Medical Home."

A health resources and services administration (HRSA) systems grant was awarded to the CMHI in 2004 and a contract for a medical home outcomes research study. Early findings from this research show that enhanced chronic condition management and care coordination, as outlined in the MHI, are associated with reductions in emergency room use, hospitalizations, and visits to specialists. Starting in 2007, the CMHI Medical Director, Dr. Carl Cooley began convening and chairing a Medical Home Work Group to design and develop a pilot program to test a new process of primary care medical home-based, longitudinal care of children with genetic conditions identified through newborn screening including the potential use of registries, planned care methods, decision support mechanisms, comanagement with specialists, and application of family-and-patient-centered practices. This project is called the New England Genetics Collaborative (NEGC) and is funding through the University of New Hampshire's Office of Sponsored Research. A similar medical home project entitled "Leadership Education in Autism Spectrum Disorders" (LEASD) will begin in the fall of 2008. The CMHI will work in partnership with Dartmouth Medical School (DMS) and the University of New Hampshire Leadership in Education in Neurodevelopmental and Related Disabilities (LEND) program to assure that all children in New Hampshire are screened for autism and spectrum disorders at 18 and 24 months to assure early diagnosis and access to early intervention and family-centered care for optimal development outcomes. The providence for the medical home model has gained substantial momentum that in 2007, the American Academy of Pediatrics, American Academy of Family Physicians, and the American College of Physicians as well as Family Voices and the National Association of Pediatric Nurse Practitioners endorsed the medical home as the model for twenty-first-century primary care (www.pcpcc.org). States are looking for better ways to effectively support the healthcare needs of its citizens and the organizational and operational needs of those providing health care. However, there is a looming crisis in primary care that raises concerns about current and future capacity, recruitment, and reimbursement. In response to the current struggle inherent to primary care, the CMHI, in partnership with New Hampshire Special Medical Services regularly convenes the New Hampshire Council on the Future of the Primary Care Medical Home with explicit goals to:

1. Build and spread awareness of the medical home model of primary care
2. Develop supports for primary care practices to improve their "medical homeness"
3. Align statewide efforts toward an investment in the future of primary care

Following initial Council meetings, the New Hampshire Endowment for Health funded the CMHI to convene and lead the New Hampshire Primary Care Task Force. This group will issue consensus statements crafted to further detail the resources and supports necessary to develop and provide relationship-centered primary care. The Task Force will comment on what they would like a future medical home pilot effort to look like in New Hampshire. The CMHI plans to develop and provide technical assistance to help primary care practices pass the National Committee for Quality Assurance (www.ncqa.org) medical home recognition measure, a requirement to enter a medical home pilot demonstration and receive enhanced payment. The CMHI is working with the New Hampshire Citizen's Health Initiative and a public private multipayer and stakeholders group to craft New Hampshire's pilot to stimulate a transformative process in primary care for the twenty-first century. In conclusion, the CMHI provides content expertise, education, and consultation focused on medical home development to a variety of states, centers, and initiatives including the American Academy of Pediatrics, the commonwealth fund, various HRSA grantees, and numerous state and regional programs.

Major Activities

2001: Medical Home Index (MHI), pediatric version, a validated measurement to assess quality and monitor improvements in a primary care practice (available at www.medicalhomeimprovement.org). 2001: Developed a companion Medical Home *Family* Index and Survey (available at www.medicalhomeimprovement.org). 2001: Medical Home Improvement Tool Kit (available at www.medicalhomeimprovement.org). 2003: Parent Partners *Creative Forces on Medical Home Improvement Teams*" (available at www.medicalhomeimprovement.org). 2006–current: New Hampshire Council on the Future of Primary Care Medical Home. 2007–current: New Hampshire Primary Care Task Force 2007: Medical Home Practice-Based Care Coordination, Workbook (available at www.medicalhomeimprovement.org). 2007–2008: New England Genetics Collaborative (NEGC), Regional Genetics and Newborn Screening Collaborative. 2008: Leadership Education in Autism Spectrum Disorders (LEASD). 2008: "Extra-Ordinary Care: Improving Your Medical

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Center for Outcome Measurement in Brain Injury (COMBI)

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Membership

The Center for Outcome Measurement in Brain Injury (COMBI) is a collaborative project of 16 brain injury facilities or centers, most of them Traumatic Brain Injury Model Systems (funded by the National Institute on Disability and Rehabilitation Research). The product is an informational web site that is oriented primarily towards clinicians and researchers, but is also freely available to the general public.

Major Areas

The COMBI is an online resource for those needing detailed information and support in regards to outcome measures for persons with brain injury. The measures included in the COMBI are commonly used in the field of brain injury rehabilitation and assessment. The COMBI is a collaborative project of 16 brain injury facilities or centers, with each center contributing information on one or more measures. For most supported measures, there are syllabus and training information, rating forms, background information on validity and reliability, a reference list of published studies, and testing materials. A Frequently Asked Question (FAQ) section is also included, compiled from past training

Center for Outcome Measurement in Brain Injury (COMBI). Table 1 COMBI-featured scales/measures

Agitated Behavior Scale (ABS)
Apathy Evaluation Scale (AES)
Awareness Questionnaire (AQ)
Cognitive Log (Cog-Log)
Coma/Near Coma Scale (CNC)
Coma Recovery Scale-Revised(CRS-R)
Confusion Assessment Protocol (CAP)
Community Integration Questionnaire (CIQ)
The Craig Handicap Assessment and Reporting Technique (CHART)
The Craig Handicap Assessment and Reporting Technique Short Form (CHART SF)
The Craig Hospital Inventory of Environmental Factors (CHIEF)
Disability Rating Scale (DRS)
The Family Needs Questionnaire (FNQ)
Functional Assessment Measure (FAM)
Functional Independence Measure (FIM)(TM)
Glasgow Outcome Scale (GOS)
Extended Glasgow Outcome Scale (GOS-E)
High Level Mobility Assessment Tool (HiMAT)
Level of Cognitive Functioning Scale (LCFS)
Mayo Portland Adaptability Inventory (MPAI)
Mississippi Aphasia Screening Test (MAST)
Neurobehavioral Functioning Inventory (NFI)
The Orientation Log (O-Log)
The Patient Competency Rating Scale (PCRS)
Participation Objective, Participation Subjective
Satisfaction With Life Scale (SWLS)
Service Obstacle Scale (SOS)
Supervision Rating Scale (SRS)

information and submitted questions. The COMBI was designed to provide accessible and consistent information regarding brain injury outcome measures to clinicians and researchers.

Landmark Contributions

The COMBI is coordinated by Santa Clara Valley Medical Center and is supported by funds from the National Institute on Disability and Rehabilitation Research (NIDRR). It has been a collaborative project of the NIDRR-funded Traumatic Brain Injury Model Systems since 1998.

Major Activities

Currently, the COMBI has 28 featured scales, with 25 scales available for immediate download. Table 1 shows the COMBI-featured scales as of August 2008. Additional information is available about measuring employment and substance abuse after brain injury. The COMBI web site also conducts testing and certification for the Disability Rating Scale (DRS). The COMBI web site (www.tbims.org/combi) receives over 1,200 visitors per day, with well over one million visits in total.

Cross References

- ▶ Agitated Behavior Scale (ABS)
- ▶ Coma/Near Coma Scale (CNS)
- ▶ Coma Recovery Scale (CRS)
- ▶ National Institute on Disability and Rehabilitation Research (NIDRR)
- ▶ Neurobehavioral Functioning Inventory (NFI)
- ▶ Orientation Log
- ▶ Rancho Los Amigos Scale
- ▶ Traumatic Brain Injury
- ▶ Traumatic Brain Injury Model System

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http://www.tbims.org/combi

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Centers for Medicare and Medicaid Service

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Membership

The Centers for Medicare and Medicaid (CMS) served 102 million Americans enrolled in Medicare, Medicaid, and the Children's Health Insurance Program (CHIP) in FY 11.

Major Areas

CMS is part of the Department of Health and Human Services. The primary offices are not located in Washington D.C. but in Baltimore. With a budget of approximately $650 billion, serving almost 90 million beneficiaries, CMS has a dominant role in American health care. The top position in CMS, the Administrator, is nominated by the President of the USA but must be confirmed by the Senate. Most administrators have come to CMS from academia or have been industry lobbyists. The position of Administrator is complex and demanding. Nancy-Ann DePearle, who served as Administrator at the end of the Clinton administration, during one of the few periods in which federal spending for CMS declined, noted that the Administrator has "...many bosses, including 535 members of Congress, the White House, the Inspector General, the US Government Accountability Office (GAO)" (*Health Affairs*: W-5–337).

The Department of Health and Human Services, through CMS, issues regulations and policy clarifications guiding the provision of services for Medicare beneficiaries. All psychologists who provide health-care services to Medicare recipients, particularly neuropsychologists, interact with CMS. Each psychologist who provides services to Medicare beneficiaries must obtain National Provider Identification (NPI) from CMS. Medicare fiscal intermediaries oversee payment for Medicare services including the electronic fund transfer. Each state has a fiscal intermediary overseeing Medicare Part A (inpatient services) and Part B (professional services). CMS regulations now guide the care of all Medicare beneficiaries. In addition, the size and impact of Medicare have made the agency a predictor of many coverage choices by private

insurers. When CMS issues proposed rules in the *Federal Register* (the federal government's daily journal), all providers have the opportunity to comment. Final rules that guide reimbursement by CMS's fiscal intermediaries are then issued.

Landmark Contributions

In July 1965, at the home of President Harry Truman, President Lyndon B. Johnson signed the Social Security Act of 1965. Titles XVIII and XIX of the Act fostered a new view of health care in the USA, responding to more than 20 years of legislative initiatives aimed at creating an insurance program to cover hospitalization, health services, and skilled nursing care for the elderly as well as coverage for low-income children. When implemented in 1966, Medicare (created by Title XVIII) provided coverage for more than 19 million elderly Americans. Medicaid (created in Title XIX), covered almost 15 million people and was administered by the Social Rehabilitation Administration, an agency managing poverty and welfare programs.

The inclusion of Medicare and Medicaid within the Social Security Act reflected political compromises needed for the passage of both programs. Wage and price controls imposed during World War II prompted many employers to offer health-care coverage. This "de facto" wage increase determined that health insurance became a common employer benefit, but one that excluded the elderly and unemployed. By 1960, the inadequacy of health services for older Americans had become a political issue. In order to secure enough votes to pass Medicare, President Johnson created the concept of the "medically indigent" thereby linking welfare to health care for the elderly.

Medicare and Medicaid were administered separately during the Johnson and Nixon administrations. Joseph Califano, Secretary of Health Education and Welfare (HEW) under Jimmy Carter, believed that ineffective oversight of health programs permitted spiraling health costs and health-care spending inflation (Derzon, 2005). Califano knew the separate administration of Medicare and Medicaid reflected the political compromises needed to create coverage for the elderly and the poor through linking coverage for the elderly to welfare. The split administration of Medicare and Medicaid, however, made the two mammoth programs less manageable. Califano moved quickly, in secret and combined Medicare and Medicaid, forming the Health Care Financing Administration (HCFA). HCFA provided oversight to the Medicare program (health insurance for people aged 65 and over, younger people receiving social security disability benefits, and persons with end-stage kidney disease) as well as the Medicaid program (providing medical assistance from state and federal governments for eligible low-income persons). Although the federal government regulates Medicaid, state governments actually operate the program). HCFA also provided federal oversight for quality control initiatives.

Between 1989 and 2000, the number of Medicare claims rose from 70% to more than 800 million claims. Despite this staggering volume, Medicare's administrative costs were only 2%. HCFA was viewed as having a bias toward providers. In 2001, the Bush administration, believing that HCFA was perceived as bureaucratic, unresponsive, and biased toward physicians and hospitals, sought a new image for the agency. In an effort to improve the agency's image, the Bush administration changed the name to the Centers for Medicare and Medicaid – CMS (Scully, 2005), heralding an effort to streamline the many administrative processes overseen by CMS. Despite the new name, CMS continued the responsibilities of HCFA: Medicare, end-stage renal disease, quality, and coordination of Medicaid with the states.

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Centigray

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Synonyms

cGY

Definition

Centigray (cGY) is the preferred measurement of absorbed radiation and is equivalent to one-hundredth (10-2) of a gray, or 1 rad. The gray measures the deposited

energy of radiation. The daily dose of radiation is also referred to as a fraction, since each dose is a percentage of the cumulative prescribed dose. The dose is given in sublethal fractions, which protracts the dose to facilitate the occurrence of repair kinetics. Hence, radiation oncologists use fractions to take advantage of the differential recovery rates for normal and neoplastic tissue, thereby permitting repopulation of normal cells and inducing radiosensitivity via increased oxygen to the remaining tumor cells. Fractionated dose radiotherapy enhances the treatment efficacy by targeting the cancer cells while mitigating the damage to healthy tissue. This is determined using a therapeutic ratio, which compares the damage to both cancerous and healthy cells (Potters, Timmerman, & Larson, 2005). Moreover, the biological effects on the relevant tissue vary according to the radiation type and intensity.

Cross References

► Radiation Therapy

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Central Auditory Processing Disorder

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Synonyms

Auditory perceptual disorder (APD); Auditory processing disorder (APD); CAPD; Sensory integration-C/APD (SI-C/APD)

Short Description or Definition

Central Auditory Processing Disorder (CAPD) is *not* a neuropsychological diagnosis. It refers to an amalgam of symptoms diagnosed by audiologists and/or speech and language specialists using the audiology code of ICD-9 code 388.4 (Abnormal Auditory Processes).

The American Speech-Language Hearing Association (ASHA, 2005) defines CAPD as difficulties in the perceptual processing of auditory information in the central nervous system (CNS). It presents in the form of poor performance in one or more of the following abilities or skills: sound localization and lateralization, auditory discrimination, auditory pattern recognition, temporal aspects of audition (including temporal integration, temporal discrimination/gap detection, temporal ordering, and temporal masking), auditory performance in competing acoustic signals (including dichotic listening), and auditory performance with degraded acoustic signals.

Categorization

Neither CAPD nor SI is listed in the DSM-IV or DSM-IV-TR manuals which provide diagnostic codes for psychologists, psychiatrists, and speech and language specialists. When individuals from these fields assess problems that are language based, they use language tests and assign diagnostic codes associated with specific language disorders (e.g., ICD-9: 315.3, Developmental Speech or Language Disorder; ICD-9: 389.9, Unspecified Hearing Loss).

While the diagnosis of CAPD has a 50-year history, there is much controversy about its legitimacy (Carneol, 2008). In part, this stems from its extensive comorbidity with a variety of impairments in attention/concentration, language, memory, IQ, academic achievement, and other behavioral/emotional disorders. Children with the CAPD diagnosis typically demonstrate poor listening skills, difficulty filtering linguistic information from background noise, problems following oral instructions, poor auditory discrimination skills, distractibility/inattentiveness, and often need additional time to complete tasks (Bloom & Hynd, 2008). In addition, there is no compelling research that reliably provides specific neurobiological bases for CAPD when compared to findings that link specific brain regions with language-based learning disabilities and Attention Deficit Hyperactivity Disorder (Bloom & Hynd, 2008). Together, these factors have called into question the validity of CAPD as a distinct diagnosis.

It is important to understand the complicated neurophysiological constructs of hearing to better understand the etiological basis of CAPD. That is, one must know how sound travels from the outer ear through the middle

and inner ear structures through the eighth auditory nerve to Heschl's gyrus in the left temporal lobe of the brain. At that point, the brain begins to recognize nerve impulses as sound and then initiates interpretation of these impulses as either speech or nonverbal auditory stimuli. These stimuli are then analyzed bilaterally by auditory stimulus networks distributed within the brain (Carneol, 2008).

Some researchers have proposed CAPD subgroups such as Auditory Decoding Deficit (primary left auditory cortex dysfunction), Prosodic Deficit (nonprimary right auditory cortex and associated areas dysfunction), and Integration Deficit (corpus callosum dysfunction) (Bellis, 2003; Bellis & Ferre, 1999; Katz, 1992). These subtypes may help clinicians design intervention strategies specific to an individual's presenting problems However, these models are theoretical in nature and not universally agreed upon (ASHA, 2005; Jutras et al., 2007).

Epidemiology

No truly authoritative population or prevalence studies regarding CAPD are available (Castrogiovanni, 2008). Chermak and Musiek (1997) estimate a prevalence rate of 2–3% in children, with the disorder occurring twice as frequently in males than females. Cooper and Gates (1991) estimate that the prevalence in older adults is 10–20%.

Natural History, Prognostic Factors, and Outcomes

The etiology of CAPD is unknown. CAPD may present as a developmental or acquired disorder. The maturation of the central auditory pathway may be delayed (Bamiou, Musiek, & Luxon, 2001). Prenatal or perinatal factors (e.g., hyperbilirubenemia, anoxia or hypoxia, ototoxic drugs, Rh incompatibility, prematurity/low birth weight, birth complications, maternal diabetes, or infections), ear infections, heavy metal exposure, cerebrovascular disorders, epilepsy, Lyme disease, and traumatic brain injury may be risk factors for the constellation of symptoms that characterize the CAPD diagnosis (Bamiou, Musiek, & Luxon, 2001; Musiek & Chermak, 2009; Riccio & Hynd, 1996).

It is not clear if problems in auditory processing resolve with age, if children are able to compensate for auditory processing deficits, and what factors may contribute to outcome trajectories (e.g., comorbid ADHD, LD) (Bloom & Hynd, 2008).

Neuropsychology and Psychology of CAPD

The immense overlap of CAPD symptoms with other developmental disorders such as ADHD continues to fuel debate among psychologists, neuropsychologists, audiologists, and speech-language pathologists over the existence of this diagnosis. While a definition of CAPD as a unimodal disorder is a useful tool in the conceptualization of the symptoms associated with it, the notion of CAPD as separate and distinct from language-based LD and ADHD has not been well documented in the literature.

In a sample of 30 9–13-year-olds, approximately 50% of children diagnosed with CAPD also met diagnostic criteria for ADHD (Riccio, Hynd, Cohen, Hall, & Molt, 1994). Based on ratings from audiologists and pediatricians, Chermak, Tucker, and Seikel (2002) identified exclusive behavior sets characterizing CAPD and ADHD. CAPD was not characterized by hyperactivity and difficulties with impulse control.

Some individuals with CAPD may have difficulties with speech and language. Varying combinations of auditory processing deficits may be associated with different functional deficits in speech and language. Differential diagnosis between CAPD and language disorders can be quite challenging due to shared symptomatology. For example, a core deficit in phonological processing underlies dyslexia. Similarly, children with CAPD may have difficulty discriminating and manipulating the phonetic aspects of auditory input. It has been suggested that auditory processing problems in developmental dyslexia are specific to the encoding of speech, whereas CAPD is characterized by a general dysfunction encoding all auditory stimuli.

Academic difficulties are often a characteristic of children who carry a CAPD diagnosis (ASHA, 2005). They may have a difficult time encoding/learning spoken information or information presented with background noise. They are more likely to have behavioral, emotional, and social difficulties secondary to poor communication skills (ASHA, 2005). The preceding, combined with associated learning difficulties, can compromise self-esteem and contribute to emotional withdrawal, somatization, conduct disorders, depression, anxiety, and interpersonal problems (Riccio, Cohen, Garrison, & Smith, 2005).

Evaluation

Since CAPD is an auditory deficit, an audiologist is the professional best-qualified to diagnose CAPD (ASHA, 2005). Although psychologists and speech-language pathologists (SLPs) may screen for auditory processing difficulties, speech-language and psychological measures should not be used to diagnose CAPD (ASHA, 2005). Screening processes involve systematically observing listening behavior and assessing performance on tests of auditory function (ASHA, 2005). Multidisciplinary assessment including other professionals such as psychologists and SLPs should be conducted to help delineate cognitive communication and language-related factors associated with CAPD, determine the functional impact of CAPD, and guide treatment (ASHA, 2005).

The audiologist's central auditory diagnostic test battery, which aims to examine the integrity of the central auditory nervous system (ASHA, 2005) may include auditory discrimination tests, auditory temporal processing and patterning tests, dichotic speech tests, monaural low-redundancy speech tests, binaural interaction tests, electroacoustic measures, and electrophysiologic measures (ASHA, 2005). Auditory processing tests include elements of attention and memory, but also appear to assess processes not tapped by measures of these constructs (Riccio et al., 2005). A diagnosis of CAPD typically requires performance, two standard deviations below the mean on two or more auditory processing tests. The audiologist must also consider the confounding effects of fatigue, poor attention, memory, and motivation on test performance.

Treatment

Collaboration among educators, school psychologists, speech-language pathologists, audiologists, neuropsychologists, and physicians may be necessary to identify and implement interventions aimed at ameliorating symptoms that resemble those associated with the CAPD diagnosis.

While there is some evidence that methylphenidate may ameliorate symptoms in children with the diagnosis of CAPD, research is inconclusive. More typical interventions for CAPD symptoms include perceptual training, instruction in linguistic and cognitive compensatory strategies, environmental modifications (e.g., preferential seating, use of a stereo system in the classroom, use of area rugs), assistive technology alternatives, and specific educational programming. These would be written in the form of an Individual Education Program (Individuals with Disabilities Education Improvement Act of 2004, 20 U.S.C. 1400; Public Law 108–446) that would provide such accommodations under the category of Other Health Impaired.

Cross References

- ADD
- ADHD
- American Speech-Language-Hearing Association (ASHA)
- Americans with Disabilities Act
- Auditory System
- Cognitive Communication Disorder
- D-amphetamine
- Dyslexia
- Executive Functioning
- Learning Disability
- Section 504 of the Rehabilitation Act of 1973
- Stimulants

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Central Executive

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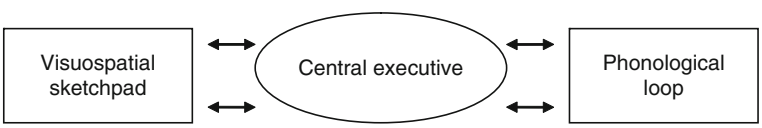
Definition

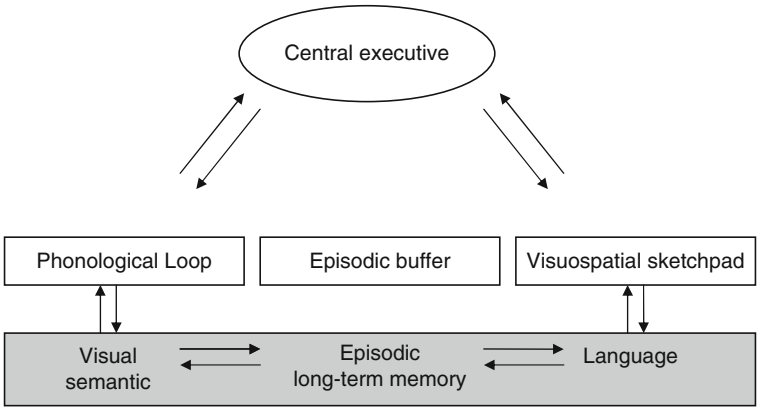
Working memory (WM) has been defined as the "blackboard of the mind" and the "mental sketchpad" (Baddeley, 1986). It enables the online holding and mental manipulation of information. Human beings use WM processes all the time. For example, WM is used to perform rapid mathematical functions in our heads and to understand the inherent meaning in speech and writing. WM is also important for reasoning and problem solving (Baddeley, 1999). Psychologists have posited several types of memory in the intact human mind. Although philosophers have long been theorizing about distinctions within memory, there has been experimental evidence supporting the divisions of memory for only the past 30–40 years (Baddeley, 1999).

Historical Background

Baddeley and Hitch (1974) developed a theory of a WM system that has become the dominant theory in cognitive psychology for the organization of WM (Fig. 1). Baddeley's model addressed weaknesses of previous short-term memory (STM) models, including the failure to address nonverbal processing. Baddeley's model, which has undergone changes throughout the years, is a theoretical concept and was not originally developed to directly correspond with specific neuroanatomical regions, though research that attempts to do so has been completed. The original theory proposed the existence of three components (Fig. 1). Baddeley envisioned this system as consisting of a central executive, which is a limited capacity subsection that controls certain subsystems. The central executive controls 'slave' systems, which are mainly used for the temporary storage of information. Information that is not needed right now, but will soon be needed, can be stored within these 'slave' systems to free up the central executive for other tasks. One of the slave systems is the articulatory or phonological loop, which is a verbal store. Subvocalization of verbal material creates a record in a phonological buffer. The central executive then reads the phonological buffer, and the cycle begins again. For most of this cycle, the central executive is not needed. Another slave system is the visuospatial sketchpad, which is used to rehearse visual or spatial materials. Manipulation of information within either the phonological loop or visuospatial sketchpad does not affect information being retained in the other system; they have been shown to be distinct systems in regard to interference effects (Brooks, 1968). Another component, the episodic buffer (described in detail below), was added in 2000 to address some of the perceived weaknesses of the earlier model. The current multicomponent model describes 'fluid'



Central Executive. Figure 1 Initial three-component model of working memory (WM) (Baddeley & Hitch, 1974)



Central Executive. Figure 2 Multicomponent model of working memory (WM) (Baddeley, 2000)

capabilities (such as temporary storage) and 'crystallized' abilities that are involved in long-term knowledge (Fig. 2).

Phonological Loop. The phonological loop is most similar to the earlier concept of STM (Baddeley, 1992) and is the most studied component within this theory. There are two components within the phonological loop: a phonological store and an articulatory rehearsal system. The phonological store can temporarily hold acoustic material for 1–2 s and is involved in speech perception. The articulatory rehearsal system can maintain material by subvocal repetition and can take visually presented material and register this material by subvocalization. There are several pieces of evidence that have supported the presence of the phonological loop. The simplest piece of evidence is that it appears clear that our verbal store holds only a limited amount of information. The *phonological similarity effect* indicates that sequences of items with similar phonological sounds are more difficult to remember than those with disparate sounds (e.g., "mad can cap man" is more difficult than "pen day cow bar rig") (Baddeley, 1966b). In addition, it demonstrates the tendency for participant errors to be phonologically similar to the correct item (i.e., F for S and B for G). Similarity of meaning does not seem to be important for the phonological store (Baddeley, 1966b), whereas long-term storage is affected by similarity of meaning, but not sound (Baddeley, 1966a). The *irrelevant speech effect* shows that exposure to irrelevant speech either at the same time or directly after stimuli material can disrupt immediate recall of the stimulus; meaningless noise does not disrupt stimuli material. This effect is the same for phonologically similar or dissimilar items (Salame & Baddeley, 1987). Preventing rehearsal (*articulatory suppression*) further decreases performance (Baddeley, 1999); when participants are prevented from rehearsing items by being required to state an irrelevant word (such as the. . .the. . .the), performance declines. There also appears to be a *word-length effect* such that longer words are more difficult to remember, potentially because participants are subvocalizing words, and thus, there is more time for information to deteriorate (Baddeley, 2000).

People frequently use the phonological store in everyday life, subvocalizing when counting and when reading. Adult fluent readers use this component less than poor readers, or individuals learning to read, but show difficulties picking up errors in written text when their subvocalization capabilities are disrupted (Baddeley, 1999). The phonological store may also be important for new language acquisition as well as native language acquisition (Baddeley, 1999); some researchers have indicated that the phonological loop's primary purpose is for language acquisition (Baddeley, Gathercole, & Papagno, 1998).

A deficit in phonological STM appears to stem from a defective phonological store. Articulatory rehearsal appears to be defective in aphasic patients with dyspraxia, as they are unable to carry out the speech motor codes needed for articulation (Waters, Rochon, & Caplan, 1992). Dysarthric patients do not appear to have a deficit in articulatory rehearsal, likely because their deficits are peripheral, not central (Baddeley & Wilson, 1985). Neuroanatomically, based upon lesion studies, the inferior parietal cortex appears to be related to the temporary phonological store, whereas the articulatory rehearsal system uses brain areas necessary for speech production, such as Broca's area and/or the supplementary motor area (Muller & Knight, 2006).

Visuospatial Sketchpad. Similar to the function of the phonological loop for verbal material, the visuospatial sketchpad allows for the maintenance of temporary

representations of visuospatial information. The visuospatial sketchpad is involved in such tasks as visual imagery and mental rotation. Evidence for this system comes from the finding that visuospatial immediate memory can be disrupted by visual tracking, but not by verbal coding (Baddeley, 1999). There seems to be separate subsystems involved in the maintenance of visual information (shape, color) and spatial information, independent of the central executive (Klauer & Zhao, 2004). In addition, there also appears to be a dissociation of this system into two components, a passive store that maintains information, and a more active device that manipulates it (Bruyer & Scailquin, 1998).

The visuospatial sketchpad appears to be principally represented within the right hemisphere (Baddeley, 2000). Within this component, as noted above, past research has indicated that there is a neuroanatomical differentiation between object and spatial information. Using evidence from the parietal and temporal visual streams, it is likely that the parietal lobe (dorsal stream) processes spatial information (i.e., where – the relation of objects across coordinates) while the temporal lobe (ventral stream) processes nonspatial (i.e., what – as in a visual image or object information); this subdivision also possibly extends into dorsal and ventral regions of the prefrontal cortex, and these regions are most relevant for short-term, or working memory processes (Goldman-Rakic & Leung, 2002).

Central Executive. The central executive has been described as a "homunculus," or "little man" who makes decisions as to how the slave systems should be used. It is a limited capacity attentional system that is in charge of the phonological loop and visuospatial sketchpad (Baddeley, 1999). Although the central executive may be considered the most important component of this model, given the apparent complexity of this system, it is difficult to investigate. Baddeley (1986) coined the term dysexecutive syndrome (DES) to describe dysfunctions of the central executive.

Baddeley has compared the central executive to Norman and Shallice's ideas of the supervisory attentional system (SAS; Shallice, 1982), which is used in planning, decision-making, novel situations, and difficult situations. Contention scheduling (CS) is a process that chooses one response and inhibits another, in a "crude and fast" way. CS modulates the selection of an action schema when it is routine, or unconscious, selecting the schema with the "strongest triggers." The SAS is a higher level system that is "slow and flexible." It is utilized when a selection is more complex, and adjusts the activation level of the action schemas. Therefore, the SAS can actually "override" the CS when the CS fails or no known schema exists (Shallice, 1982).

Additional attentional processes that have been ascribed to the central executive include focusing, dividing, and switching attention, as well as serving as the interface between the subsystems described above and long-term memory (Baddeley, 2001). With regard to *focusing* attention, participants in a chess game were impaired by interference from a random digit generation task; this was thought to place a heavy load on the central executive (Robbins et al., 1996). With regard to *dividing* attention, participants with Alzheimer's disease who have long-term memory and attentional deficits are impaired on dual-task paradigms, yet are able to do the same tasks individually (MacPherson et al., 2007). There is a question as to whether *switching* is primarily an executive process, though there also appears to be a strong relationship between the phonological loop and switching.

Research has indicated that the prefrontal cortex, especially the dorsolateral prefrontal region, is critical for executive functioning (Collette et al., 1999; D'Esposito et al., 1995; Owen, Evans, & Petrides, 1996; Salmon et al., 1996). However, more recent studies have indicated that, although the PFC appears essential for executive functioning, other brain regions may also be important for these functions (Baddeley & Wilson, 1988; Baker et al., 1996; Berman et al., 1995; Nagahama et al., 1996).

Episodic Buffer. As mentioned above, the episodic buffer was developed in response to a need for a component to relate between working and long-term memory (Baddeley, 2000). The episodic buffer is envisioned as a temporary storage system controlled by the central executive, as well as an interface between multiple systems. The central executive can retrieve information from the store, and potentially manipulate or modify it. There were multiple weaknesses to the initial three-component theory that this multicomponent model has attempted to address. For example, with articulatory suppression (interference of encoding of numbers) of the phonological loop, short-term span becomes shorter, but does not completely disappear (Baddeley, 1984). In addition, participants can remember more in sentences or in paragraphs than would be anticipated solely based upon the phonological store. There appears to be a distinction between immediate recall and delayed prose recall, with disparate subsystems implicated in each (Wilson & Baddeley, 1988). The episodic buffer appears to be preserved in patients with impaired LTM (Baddeley, 2000), and plays an important role in sending information to and retrieving information from LTM. Although there is minimal research specifically examining the neuroanatomical basis of this component,

it appears likely that the frontal lobes are involved in these processes. Specifically, there appears to be greater right frontal activation for integration of information (Prabhakaran, Narayanan, Zhao, & Gabrieli, 2000).

Baddeley's theory provided a framework for the initial study of working memory processes, addressing weaknesses of earlier models of STM that did not sufficiently account for many important pieces of information. Although there have been additions to the model since its initial conception, Baddeley's ideas continue to be relevant today, and this work has informed, and continues to inform, multiple avenues of research within the field.

Cross References

- ► Attention/Executive Functions
- ► Controlled Attention
- ► Dysexecutive Syndrome
- ► Working Memory

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Central Neurocytoma

► Neurocytoma

Central Venous Thrombosis

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Synonyms

Cerebral thrombophlebitis; Cerebral venous thrombosis; Dural sinus thrombosis; Intracranial venous thrombosis; Sagittal sinus thrombosis; Sinus thrombosis

Definition

Central venous thrombosis is a rare form of stroke that results from thrombosis (blood clot) of the veins in the dura mater that surround and drain blood from the brain.

Current Knowledge

Symptoms may include headache, abnormal vision, seizures, and any of the symptoms of stroke such as weakness of the face and limbs on one side of the body. Diagnosis is usually made by computed tomography (CT/CAT) or magnetic resonance imaging (MRI) scanning, using radiocontrast to demonstrate obstruction of the venous sinuses by thrombus. In about 80% of patients with this condition, it occurs in the setting of a preexisting underlying clotting disorder such as Protein C deficiency, Protein S deficiency, hyperhomocysteinemia, nephrotic syndrome, antiphospholipid antibody syndrome, pregnancy, oral contraceptive use, systemic lupus erythematosus, Wegener's granulomatosis, and sarcoidosis, and infections such as otitis, mastoiditis, and meningitis. For this reason, a search for these conditions should be undertaken.

Treatment relies on the administration of anticoagulants, or rarely, thrombolytic agents. If raised intracranial pressure results from the thrombosis, then surgical placement of a ventriculoperitoneal shunt is considered. Survival rate exceeds 90%, full recovery occurs in 88% of the survivors, and recurrence rate is about 2%.

Cross References

► Anticoagulation
► Antiplatelet Therapy
► Cerebrovascular Disease
► Heparin
► Ischemic Stroke
► Thrombolysis
► Thrombosis
► Venous Thrombosis
► Warfarin

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Cephalalgia

► Headache
► Post-traumatic Headache

CERAD

► Consortium to Establish a Registry on Alzheimer's Disease

Cerebellar Cognitive Affective Syndrome

► Cognitive Affective Syndrome

Cerebellar Hemorrhage

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Synonyms

Posterior fossa hemorrhage

Definition

A cerebellar hemorrhage is a bleeding into the cerebellum, the portion of the brain located posteriorly, that controls balance, coordination, and related functions.

Current Knowledge

It is estimated that 10% of all intracerebral hemorrhages, or about 1–2% of all strokes, are cerebellar hemorrhages. It can be caused by high blood pressure, heavy alcohol consumption, cocaine use, anticoagulant use, clotting disorders, cerebral vascular abnormalities such as arteriovenous malformations and aneurysms, and cerebral amyloid angiopathy. Approximately two-thirds are thought to result from hypertension. Symptoms of the hemorrhage include headaches, especially at the posterior and inferior area of the skull, nausea and emesis, stiff neck, dizziness and vertigo, blurred or double vision, balance and coordination deficits, speech difficulty, and altered consciousness. The onset of symptoms is generally abrupt and dramatic. This is a medical emergency, requiring immediate neurosurgical attention. It is diagnosed using CT or MRI scanning. Treatment relies on reducing intracranial pressure and surgically removing the hemorrhage as quickly as possible.

Cross References

▶ Hemorrhagic Stroke
▶ Intracerebral Hemorrhage
▶ Intracranial Hemorrhage

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Cerebellar Mutism

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Definition

A transient speech disorder typically associated with resection of cerebellar tumors, particularly medulloblastomas involving the cerebellar vermis. Cerebellar mutism can also result from stroke in the cerebrovascular distribution, affecting the cerebellar peduncles and brainstem. Cerebellar mutism forms part of a syndrome of deficits, known as the posterior fossa syndrome (PFS).

Current Knowledge

Symptoms

As part of PFS, cerebellar mutism is associated with decreased or absent speech, irritability, hypotonia, ataxia, and the inability to coordinate voluntary movements, including the volitional motor aspects of speech. Mutism occurs within the first week of surgery (or cerebrovascular event) and its duration may be a matter of days or weeks. It is expected to resolve within 4 months. However, even with the return of functional speech, the quality of vocalizations may lack normalcy in being hypernasal, monotone, high pitched, slowed, and/or sparse.

Pathophysiology

Information obtained from single photon emission computerized tomography (SPECT) studies have supported the theory that cerebellar mutism results from the effects of decreased cerebral and cerebellar blood flow upon cell functioning in particular brain pathways. These cause disruption in the cerebellar modulation of neural circuits that link prefrontal, posterior parietal, superior temporal, and limbic cortices with the cerebellum.

Treatment

The treatment of cerebellar mutism resides in speech–language therapy, with early goals of therapy including the teaching of nonverbal communication as a compensatory strategy as well as the direct strengthening of oral-motor functioning through the systematic practicing of tongue and lip movements. Moreover, since cerebellar mutism occurs within a context of broad cognitive and affective change, a comprehensive assessment of speech and language skills is important to identify and treat language deficits more broadly. Problems in higher-level cognitive–linguistic functioning can include difficulty in areas like planning and initiating communication, verbal fluency, abstract reasoning, and working memory.

Cross References

- ▶ Brain Tumor
- ▶ Cerebellum
- ▶ Cognitive Affective Syndrome
- ▶ Medulloblastoma
- ▶ Posterior Fossa
- ▶ Posterior Fossa Syndrome

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Cerebellum

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Definition

The cerebellum, one of the three major portions of the brain, is involved with the coordination of voluntary movements as well as the control of equilibrium.

Current Knowledge

Embryology

The cerebellum originates from the metenencephalic division of the rhombencephalon, sharing the same embryologic origin with the pons.

Anatomy

The cerebellum consists of three main lobes: anterior (paleocerebellum or spinocerebellum), posterior (neocerebellum or cerebrocerebellum), and flocculonodular (archicerebellum or vestibulocerebellum). These lobes are further divided to ten small lobules: lingula, folium, central lobule, tuber, culmen, pyramid, declive, uvulae, and tonsils.

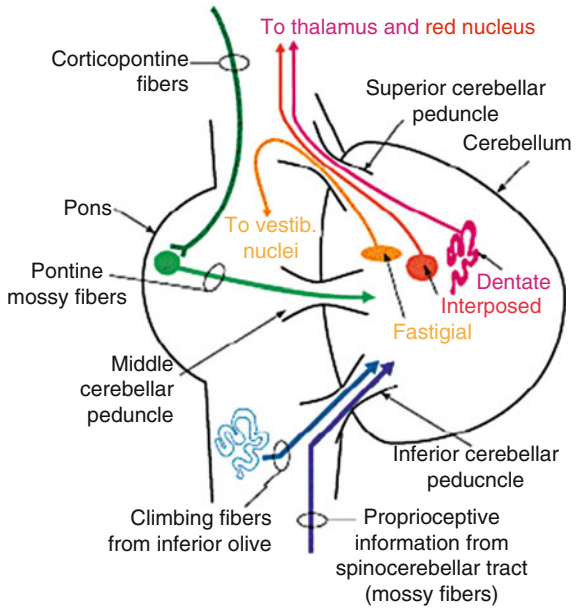
Cerebellar Peduncles

The cerebellar peduncles are bundles of nerve fibers, connecting the cerebellum with the brain stem. There are three bundles: inferior, middle, and superior cerebellar peduncles.

The inferior cerebellar peduncle (ICP) contains the dorsal spinocerebellar tract (DSCT), cuneocerebellar tract (CCT), olivocerebellar tract (OCT), and vestibulocerebellar tract (VCT). DSCT fibers arise from cells in the ipsilateral Clarke's column in the spinal cord (C8-L3). CCT fibers arise from the ipsilateral accessory cuneate nucleus. The largest component of the OCT fibers arises from the contralateral inferior olive. VCT fibers arise from cells in both the vestibular ganglion and the vestibular nuclei, and pass in the inferior cerebellar peduncle to reach the cerebellum.

The middle cerebellar peduncle (MCP) contains the pontocerebellar tract (PCT). These fibers arise from the contralateral pontine gray matter.

The superior cerebellar peduncle (SCP) is the primary *efferent* peduncle of the cerebellum. It contains fibers that arise from several deep cerebellar nuclei. These fibers pass ipsilaterally and then cross at the level of the inferior colliculus to form the decussation of the SCP. From the SCP, these fibers will then continue rostrally to terminate in the red nucleus and the motor nuclei of the ventral anterior (VA) and ventral lateral (VL) thalamus.



Cerebellum. Figure 1

Histology

Cortex

The cerebellar cortex consists of three layers from outermost to innermost: molecular, Purkinje (pyriform), and granular layers. The molecular layer consists mainly of neuropil and is the site of synapses. The majority of cells in this layer are stellate and basket cells in addition to a few neurons. Purkinje cell layer consists of a single layer of large (25 micrometer) pear-shaped neurons. It is considered the largest cells in the nervous system. The granular layer is dense with 3–7 million neurons per cubic mm, consisting of small cells with a granular cytoplasm. Beneath the cortex lies white matter that forms the core of the foliae.

Cells

The cerebellum is formed of several complex cells. Stellate cells project local inhibitory output (taurine). Basket cells also project local inhibitory output (GABA). They feed forward inhibition on Purkinje cells. Purkinje cells are stimulatory, transmitting impulses from the cerebellar cortex via efferent pathways. They project inhibitory out (GABA). Granule cells have axons that run parallel to the longitudinal axis of the lobes. Granule cell excite by way of glutamate. Granule cells are only excitatory in the cerebellar cortex, terminating on Golgi, basket, and Purkinje cells. Golgi cells are intermediary cells located in the granular layer. They receive stimulatory input (GABA) from the granular layer to inhibit granule cells.

Nuclei

Purkinje cells can transmit inhibitory signals to the deep nuclei of the cerebellum. The most lateral nucleus, dentate, receives its input from the OCT and PCT and carries planning information from the posterior parietal area. The interpositus nucleus (globose and emboliform) receives its Purkinje cell input from the OCT and PCT fibers carrying information from primary motor cortex (area 4), DSCT, and CCT. Medially located fastigial nucleus receives input from the DSCT and CCT. Vestibular nuclei receive proprioceptive input from the spinal cord and medullary olive.

Fibers

Climbing fibers go to all parts of the cerebellum. They are not restricted to a particular zone. A climbing fiber sends a collateral synapse to the deep cerebellar nuclei, which is excitatory. The climbing fiber then "climbs up," synapsing on the dendrites of the Purkinje cell. Each Purkinje cell receives input from only one climbing fiber axon, but each climbing fiber axon can split to innervate several Purkinje cells. The climbing fiber-Purkinje cell synapses are excitatory. The OCT terminates directly on Purkinje cells, by-passes granule cells, and causes complex spikes in Purkinje cells.

Mossy fibers are the axons of DSCT, CCT, vestibulocerebellar (VCT) and PCT carrying input to the cerebellar cortex. They terminate and excite granule cells. Each mossy fiber branches profusely in the white matter. Each Purkinje cell receives input from approximately >20,000 mossy fibers and only one climbing fiber.

Parallel fibers are the long axons of granule cells that pass dorsally through the granule and Purkinje cell layers to reach the molecular layer of the cerebellar cortex, where they bifurcate and run parallel to the long axis of the folium. Parallel fibers excite a row of Purkinje cells and in addition to a few basket cells that in turn will inhibit distant Purkinje cells outside the field of excitation.

The aminergic fibers originate in the Raphe nuclei and possess serotonergic input, modulating the granule and molecular layers. This category of fibers also includes those originating from the locus ceruleus possessing noradrenergic input and terminating in all three cortical layers.

Function

The cerebellum receives input from all areas of the central and peripheral nervous systems. Continuous flow of information from the spinal centers and cortical areas are integrated in the cerebellar cortex. The cerebellar output then guides the precision of different cerebral functions namely equilibrium, planned voluntary movements, and muscle tone. The cerebellar modulation of motor control is executed through its inhibitory output to the motor cortex and the descending motor tracts. Guided by visual, proprioceptive, and vestibular spinal input, the cerebellum compares the intended force needed to execute a planned voluntary movement with the appropriate muscle power needed to execute it. It then modulates the tone of the agonist and antagonist muscles through inhibitory input to the motor cortex, the pyramidal and extrapyramidal tracts, aiming at the execution in a precise manner. While this voluntary movement is executed, maintenance of equilibrium regardless of movement or body position is achieved through the cerebellar output to the antigravity muscles and the vestibular centers. Eye movements are also maintained during body movement via extensive connections with the occulomotor nuclei in the brain stem. More recent data has shown that the cerebellum is involved in cognitive, behavioral, and emotional processing, including executive control, attention, working memory, learning, language, pain, and emotion (Strick et al., 2009).

Lesions

Damage to the cerebellar center or to either its inflow or outflow tracts leads to loss of cerebellar fine tuning modulation on different cerebral functions. Vestibulocerebellar lesions can result in disequilibrium, nystagmus, abnormal gait, and recurrent falls. Truncal ataxia and scanning speech are seen with spinocerebellar lesions. Patients with corticocerebellar lesions can display signs of dysmetria, asynergia (decomposition of the voluntary movement), hypotonia, dysdiadochokinesia, and intention tremors.

Cross References

- ▶ Ataxia
- ▶ Dysdiadochokinesia
- ▶ Glutamate
- ▶ Nystagmus
- ▶ Proprioception

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Cerebral Amyloid Angiopathy

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Definition

Cerebral amyloid angiopathy is a syndrome characterized by recurrent spontaneous lobar cerebral hemorrhages of various sizes and in various locations. Each hemorrhage may be asymptomatic or may cause all of the symptoms of lobar hemorrhages resulting from increased intracranial pressure, including severe headache, seizure, stiff neck, and vomiting; altered consciousness; paralysis or weakness and sensory loss; cognitive and language dysfunction, often leading to dementia after multiple episodes.

Current Knowledge

The pathological process that causes this disease is the deposition of a protein, beta-amyloid, in the walls of the arteries of the brain. Interestingly, this protein is identical to the one found in high quantities in the brains of patients with Alzheimer's disease. The incidence of cerebral amyloid angiopathy is difficult to estimate, but is known to increase with advancing age. It is thought to account for 15% of all intracerebral hemorrhages in patients over 60 years and up to one-half of lobar intracerebral hemorrhages in patients older than 70, totaling about 20 per 100,000 per year in that group. Diagnosis is usually made based on the clinical presentation and imaging of recurrent spontaneous lobar hemorrhages, with no other predisposing problems, usually associated with progressive decline in function, and most often associated with dementia. The recurrences can occur simultaneously,

clustered in time, or separated by years. The definitive diagnosis, based on pathological findings, is most typically made postmortem. Treatment is usually supportive, consisting of observation, symptom relief, and rehabilitation.

Cross References

- ▶ Hemorrhagic Stroke
- ▶ Lobar Hemorrhage

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Cerebral Angiitis

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Synonyms

Cerebral vasculitis

Definition

Cerebral angiitis or cerebral vasculitis is a relatively rare disease, characterized by inflammation of the blood vessels inside and leading to the brain. It may be caused either by a primary disease of the blood vessel walls producing inflammation or as a secondary phenomenon resulting from a systemic inflammatory disease such as systemic lupus erythematosus or certain infections.

Current Knowledge

Angiitis that is confined to the brain is relatively uncommon, and is called primary angiitis of the central nervous system (PACNS), isolated CNS vasculitis, primary CNS vasculitis, or granulomatous angiitis of the nervous system. It usually affects small- and medium-sized cerebral blood vessels, but does not involve blood vessels outside of the CNS. Headache and encephalopathy are the most frequent symptoms. Stroke occurs in about 20% of patients. Blood tests reflecting inflammation are usually normal, but magnetic resonance imaging of the brain is abnormal in more than 90% of patients. However, the pattern of abnormal findings is not specific. Cerebrospinal fluid analysis usually reveals elevations in total protein level or white blood cell count. Angiography has a low sensitivity and low specificity. Treatment usually includes cyclophosphamide and prednisone.

Cross References

- ▶ Lupus Cerebritis
- ▶ Moyamoya Disease
- ▶ Vasculitis
- ▶ Vasospasm

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Cerebral Artery

- ▶ Anterior Cerebral Artery
- ▶ Internal Carotid Artery
- ▶ Middle Cerebral Artery
- ▶ Posterior Cerebral Artery

Cerebral Autoregulation

- ▶ Cerebral Blood Flow

Cerebral Blood Flow

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Synonyms

Cerebral autoregulation; Cerebral perfusion pressure

Definition

Cerebral blood flow is the amount of blood that goes through the arterial tree in the brain in a given amount of time.

Current Knowledge

In adults, cerebral blood flow is typically 750 ml per minute, or about 50 ml per 100 grams of brain tissue per minute. This amount is equivalent to about 15% of the total cardiac output. Cerebral blood flow is highly regulated, through "autoregulation," in order to meet the metabolic demands of the functioning brain. If it is too high, it can cause elevated intracranial pressure, which will compress and damage brain tissue. If it is too low, it will fail to meet the demands of the brain, resulting in cerebral ischemia if blood flow is less than 20 ml per 100 grams of brain tissue per minute and in cerebral infarction if blood flow is less than 10 ml per 100 grams of brain tissue per minute. Cerebral blood flow is affected by blood viscosity, blood vessel size, intracranial pressure level, and systemic blood pressure.

Cross References

- ▶ Atherosclerosis
- ▶ Diffusion-Weighted Imaging
- ▶ Ischemic Penumbra
- ▶ Ischemic Stroke
- ▶ Perfusion-Weighted Imaging
- ▶ Transcranial Doppler Ultrasonography
- ▶ Vasospasm

References and Readings

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Cerebral Cavernous Malformation (CCM)

- ▶ Angioma, Cavernous Angioma

Cerebral Cortex

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Synonyms

Cerebrum surface; Cortex

Definition

The cerebral cortex is a structure lying on the outer surface of the vertebrate cerebrum that is responsible for consciousness and higher brain functions.

Historical Background

The cerebral cortex is a structure lying on the outer surface of the vertebrate cerebrum that is responsible for consciousness and higher brain functions, including sensory perception, voluntary movement, language, reasoning, memory, and planning. Cerebral comes from the Latin word cerebrum, meaning brain. Cortex comes from the Latin word for bark, which is typically an outer layer or covering. In large mammals, this structure is folded forming ridges known as gyri and grooves known as sulci. Gyri and sulci normally form in the same relative locations from one individual to another. This folding increases the cortical surface area while allowing for constraints on skull circumference. Abnormal folding of the cortex is associated with neurological deficits. Absent or reduced folding in humans is known as lissencephaly (smooth brain) and is associated with mental retardation and epilepsy (Leventer, Mills, & Dobyns, 2000). The abnormality of small regions of increased folding is known as polymicrogyria (many small ridges), and can also be associated with developmental delay and epilepsy (Piao et al., 2005).

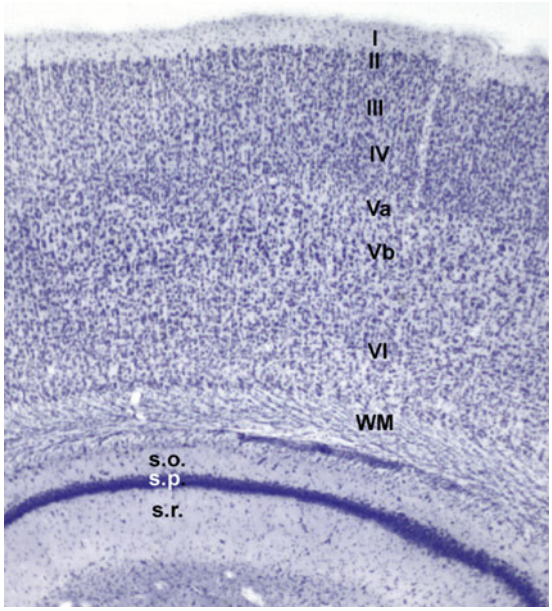
Current Knowledge

Structurally, the cerebral cortex can be divided into four lobes named after the overlying cranial bones: frontal, parietal, temporal, and occipital. Prominent sulci define the borders of these lobes. Primary functions of the lobes

can be ascribed as follows: occipital: vision; parietal: sense of touch (somatosensation) and body position; frontal: planning of action and control of movement; and temporal: hearing, visual identification, and memory. These lobes are present in each of the two hemispheres (left and right).

The cerebral cortex can also be divided on the basis of phylogeny into archicortex, paleocortex, and neocortex (oldest to newest). Archicortex consists of the hippocampus, which is associated with the acquisition of memories. Paleocortex is primarily associated with the function of olfaction. Only mammals have neocortex. In humans, the majority of the cerebral cortex is made up of neocortex. In addition, as a percentage of total brain tissue, humans have more neocortex than other species (see for example, human relative to rat, Swanson, 1995). This is unique to the neocortex, since evolution has not increased the size of other brain structures, such as the cerebellum (Clark, Mitra, & Wang, 2001). This increase in proportion is due to increased surface area, and not to a change in the thickness of the cortex, which is from 1–3 mm thick in all species.

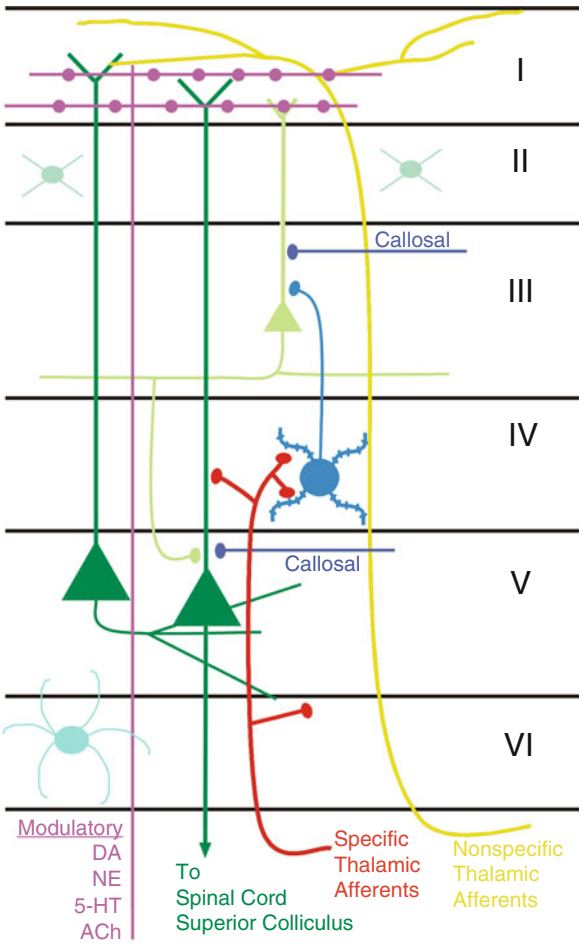
Cortex is made up of gray matter, where cell bodies predominate, and white matter that consists primarily of myelinated axons. All cortex is laminated, but the gray matter of neocortex has six layers (Fig. 1), while that of the older archi-and paleocortices has three layers. Cortical layers are differentiated based on their cellular components. The basic components and lamination of cortex are consistent across phylogeny. Within neocortex, the layers are given names that represent the predominant neuronal cell type. The outermost layer (identified by Roman numeral I), contains mainly dendrites and axons and is called the molecular layer. Layer II is called the external granule layer and consists of small, spherical cells. Layer III primarily contains small to medium pyramidal neurons and is called the external pyramidal layer. Layer IV contains spiny stellate neurons and is called the internal granule cell layer. Layer V contains large pyramidal neurons and is called the internal pyramidal layer. Layer VI has a variety of morphological cell types and is therefore called the polymorphic cell layer. These layers have differential functions that are consistent across different neocortical areas (Fig. 2). Layer I is a modulatory region and receives input from higher order cortical regions. Layers II and III perform intracortical processing, receiving input from the deeper layer IV, as well as from adjacent layers II and III and from the homologous cortical region in the opposite hemisphere. Layer IV is the major input layer and receives specific thalamocortical afferents in sensory areas of cortex. Layer V is the major cortical output, for instance sending the result of motor cortical processing to the motor neurons of the spinal cord. Layer VI provides a return feedback to the thalamus.



Cerebral Cortex. Figure 1 Cortical Lamination. Cresyl Violet stained coronal section from rat, showing neocortex above hippocampus (archicortex). Neocortical layers are indicated by Roman numerals. Layer V is commonly divided into subparts with layer Va containing callosal projection neurons, and Vb containing the largest pyramidal neurons that project to spinal cord and other subcortical locations. Within this section of neocortex, slight differences in cytoarchitectonics can be seen between the somatosensory cortex (Brodmann's area 3) to the right and motor cortex (Brodmann's area 4) to the left. Most obvious is the lack of a clear layer IV within motor cortex. *WM* neocortical white matter; s.o. stratum oriens; s.p. stratum pyramidale; s.r. stratum radiatum, all of the CA1 region of the hippocampus

Different functional regions of cortex are considered to have primary, secondary, and association components. In sensory cortex, the primary cortical area is the region that first receives information about that sense from the periphery (traveling by way of the thalamus). The secondary cortical area is considered "higher order" because the input it receives is the result of cortical processing from the primary cortical area. Association cortex receives input from several different cortical regions.

The general function of the layers is maintained across cortical regions; however, there are slight changes in cell size and packing density (cytoarchitecture) from one



Cerebral Cortex. Figure 2 Diagram of typical excitatory neuronal cell types and connections of neocortex. Layer I, the modulatory cell layer, contains the tufts of deeper lying pyramidal neurons, nonspecific thalamic afferents, and input from brainstem modulatory transmitter systems. Layer II contains primarily small granule cells. Layer III has small pyramidal neurons that perform intracortical processing, sending their axons horizontally within layer III. Layer IV contains spiny stellate neurons that send their output to layer III. Specific thalamic afferents make excitatory synapses within layer IV on the spiny stellate cells as well as on the apical dendrites of deeper lying pyramidal neurons. These specific thalamic afferents have a smaller projection to layer VI, near the border with layer V. Layer V is the major output layer for the cortex, and contains medium and large pyramidal neurons. Layer VI contains neurons with a variety of shapes. Note that inhibitory cells that make up ~20% of the neurons in the neocortex are not shown here. Modulatory: Modulatory neurotransmitters; DA: Dopamine; NE: Norepinephrine; 5-HT: Serotonin; and ACh: Acetylcholine

cortical area to another. These cytoarchitectonic differences were used by Korbinian Brodmann in 1909 to draw boundaries presumed to identify functionally different cortical areas.

Perpendicular to the plane of the cortical layers are functional modules called cortical columns. The idea that a column of cortex represents a fundamental processing unit was brought to light by Vernon Mountcastle of Johns Hopkins University (Mountcastle, 1957). Within a column, neurons tend to have similar response properties. For instance, within somatosensory cortex, the neurons within a column have similar receptive fields (area of the receptive surface that causes the neuron to fire action potentials). Neurons of different columns have nonoverlapping receptive fields. The result of evolution then has been to add cortical columns or additional processing units.

Head trauma, stroke, and tumor may all result in lesions of the cerebral cortex. The function lost will be dependent on the location of the lesion. For example, lesion of Brodmann's area 17 will result in loss of vision, while lesion of Brodmann's area 3 will result in some loss of somatosensation including touch and pain discrimination. Damage to Brodmann's area 4 will result in loss of motor function. Lesions of the frontal cortex can cause severe personality changes. Memory loss is typically associated with cortical lesions. The inability to speak occurs after destruction of Broca's area in the ventral portion of the frontal lobe, typically in the left hemisphere (Brodmann's areas 44 and 45). Incoherent speech or "word salad" results from the destruction of Wernicke's area in the upper portion of the temporal lobe (part of Brodmann's area 22).

Cross References

- ▶ Association Cortex
- ▶ Auditory Cortex
- ▶ Brodmann's Areas of the Cortex
- ▶ Heteromodal Cortex
- ▶ Homotypic Cortex
- ▶ Neocortex
- ▶ Prefrontal Cortex
- ▶ Primary Cortex
- ▶ Secondary Cortex
- ▶ Somatosensory Cortex
- ▶ Striate Cortex
- ▶ Tertiary Cortex
- ▶ Unimodal Cortex
- ▶ Visual Cortex

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Cerebral Dominance

► Hemispheric Specialization

Cerebral Edema

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Synonyms

Cytotoxic edema; Vasogenic edema

Definition

Cerebral edema is an increase in the water content of the brain that leads to brain swelling. It may be divided into two broad categories: vasogenic and cytotoxic.

Vasogenic edema involves a disruption in the blood–brain barrier with leakage of fluid from the intravascular space.

In cytotoxic edema, the blood–brain barrier is intact, and there is an increase in the intracellular fluid compartment.

Cross References

► Cerebral Perfusion Pressure
► Intracranial Pressure

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Cerebral Embolism

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Synonyms

Embolic stroke

Definition

A cerebral embolism is a blood clot (thrombus) that starts from the heart or blood vessel where the clot originates and stops in an artery that leads to or rests within the brain. The result is occlusion of the vessel and obstruction of the flow of oxygen and blood to the brain tissue supplied by that artery.

Current Knowledge

Cerebral embolisms cause about 15–20% of all strokes and about one-quarter of all ischemic strokes. It occurs most

frequently in patients who have known heart disease, including atrial fibrillation and other arrhythmias, valve disease, "mural thrombus" (a blood clot sitting in the left ventricle of the heart), or other conditions. It causes symptoms similar to those of thrombotic strokes, but the presentations of embolic strokes tend to be more abrupt and dramatic. These can include sudden onset of hemiplegia, sensory loss, facial weakness, cognitive deficits, or speech disturbance. Seizures or headaches are relatively common in embolic strokes, and both of these symptoms are relatively rare in ischemic strokes. In addition, there may be multiple diffuse simultaneous neurological findings, which may result from multiple simultaneous emboli, known as "showers of emboli." Usually, management requires addressing the cardiac condition and preventing subsequent emboli by using anticoagulants, in addition to the treatment of the cerebral infarction and its neurological consequences.

Cross References

- ▶ Anticoagulation
- ▶ Echocardiogram
- ▶ Infarction
- ▶ Ischemic Stroke
- ▶ Myocardial Infarction
- ▶ Thrombosis
- ▶ Warfarin

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Cerebral Evoked Potentials

- ▶ Event-Related Potentials

Cerebral Hemisphere

- ▶ Frontal Lobe
- ▶ Occipital Lobe
- ▶ Parietal Lobe
- ▶ Temporal Lobe

Cerebral Hemorrhage

- ▶ Hemorrhagic Stroke

Cerebral Infarction

- ▶ Ischemic Stroke

Cerebral Leukencephalopathy

- ▶ Periventricular Leukomalacia

Cerebral Malformation

- ▶ Arteriovenous Malformation (AVM)

Cerebral Microvasculature

- ▶ Blood-Brain Barrier

Cerebral Palsy

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Synonyms

Static encephalopathy

Short Description or Definition

As defined by the International Workshop on the Definition and Classification of Cerebral Palsy, Cerebral Palsy (CP) is:

- a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behavior, by epilepsy, and by secondary musculoskeletal problems (Rosenbaum et al., 2007).

Categorization

Classification schemes are critical to providers attempting to describe the disorder, measure change in function, and provide prognostic information (Rosenbaum et al., 2007). Historically, classification has focused on two features: (a) tone and (b) body part involvement (Menkes & Sarnat, 2000). Table 1 for a summary.

Spastic forms of CP are more common, with spastic diplegia being the most common (Menkes & Sarnat, 2000). However, there is disagreement regarding classification schemes, largely due to poor reliability (Accardo & Hoon, 2008). Recent attempts to improve classification schemes include: (a) development of standardized examination procedures and diagnostic algorithms and (b) use of the Gross Motor Functional Classification System (GMFCS), which focuses on functional mobility without identifying abnormalities of tone or affected limbs (Rosenbaum et al., 2007).

Epidemiology

Prevalence rates vary from 1.3 to 3/1,000 and are stable across country of origin (Clark & Hankins, 2003). Preterm birth raises the risk of CP, with a recent study reporting a diagnosis of cerebral palsy among 20% of children born at or before 27 weeks of gestation versus diagnosis of 5–6% of children born between 28 and 31 weeks (Ancel et al., 2006). Reviewers note that overall rates of CP are climbing, while rates of CP among full-term infants have remained stable (1.1/1,000). This suggests that increases are largely due to greater survival of preterm infants (Mukherjee & Gaebler-Spira, 2007).

Natural History, Prognostic Factors, and Outcomes

Etiology varies by birth status (preterm or full-term) and type of CP (Menkes & Sarnat, 2000). For preterm infants, the most common causes of CP are intraventricular hemorrhage and/or periventricular leukomalacia. Risk factors for CP in full term infants include prenatal infections, anoxic or ischemic injuries, genetic syndromes, brain malformations, or stroke. Atypical CP with athetoid movements is typically caused by basal ganglia damage secondary to hyperbilirubinema (Mukherjee & Gaebler-Spira, 2007). A variety of symptoms are associated with CP, which vary by severity and type of CP (Menkes & Sarnat, 2000; Mukherjee & Gaebler-Spira, 2007). See Table 2 for a summary.

Neuropsychology and Psychology of Cerebral Palsy

As many as 30–50% of children with CP may have a diagnosis of mental retardation, with increased incidence for children with quadriplegia, more severe motor deficits, full-term birth, and/or a coexisting seizure disorder (Menkes & Sarnat, 2000; Mukherjee & Gaebler-Spira, 2007). Estimates of intellectual functioning can be difficult to obtain, as apraxic speech, visual difficulties, and

Cerebral Palsy. Table 1 Traditional categorization of cerebral palsy

Type	Description
Spastic cerebral palsy	Increased muscle tone with movement
Spastic quadriplegia	Spasticity of upper and lower limbs
Spastic diplegia	Greater involvement of the legs than the arms
Spastic hemiparesis	Greater involvement of one side of the body (more often the right); greater impairment of the arm than the leg
Extrapyramidal cerebral palsy	Involuntary and abnormal muscle movements: dystonia (fluctuating tone and abnormal body postures) and/or athetoid movements (writhing movements in the extremities)
Hypotonic cerebral palsy	Persistent, low muscle tone
Mixed and atypical cerebral palsy	Mixture of spasticity and extrapyramidal symptoms

Cerebral Palsy. Table 2 Symptoms associated with CP

Domain	Symptoms
Secondary muscular and orthopedic symptoms	Delayed development of adaptive motor skills
	Gait abnormalities
	Oral-motor difficulties and problems with speaking and drooling
	Contracture (shortening of the muscle)
	Bone deformities (e.g., hip subluxation/dislocation)
	Scoliosis
	Osteoporosis
	Reduced limb growth
Neurologic symptoms	Seizure disorder
Sensory	Visual problems
	Homonomous hemianopsia (in spastic hemiplegia)
	Strabismus
	Nystagmus
	Visual sequela related to prematurity
	Tactile/perceptual deficits
	Stereognosis
	Poor two-point discrimination (in spastic hemiplegia)
	Neglect of affected side of the body (in spastic hemiplegia)
	Hearing loss
Feeding/gastrointestinal	Dysphagia and aspiration
	Malnutrition requiring gastrostomy
	Gastroesophageal reflux
	Constipation
	Incontinence or difficulty voiding
Dental	Malocclusion
	Poor tooth enamel
Pain and fatigue	Pain associated with primary (e.g., spasticity and contractures) and secondary (e.g., constipation) disease processes
	Pain related to medical procedures
	Fatigue secondary to poor mobility

fine motor deficits can limit participation in traditional tests (Fennell & Dikel, 2001). Little is known about cognitive functioning among children with CP who do not have a diagnosis of mental retardation, reports of deficits in learning (specifically arithmetic), visual-perceptual processing (particularly in spastic diplegia), working memory, and executive functioning are emerging (Blondis, 2004; Jenks et al., 2007).

The empirical literature regarding mental health in children with CP is sparse. Overall, studies of children with physical disabilities suggest that difficulties with adjustment are atypical, with the exception of poor self-concept in areas directly impacted by the physical disability (e.g., attractiveness, social interaction, athletics, academics) (Miyahara & Cratty, 2004). Adolescent girls may be at particularly increased risk for low self-concept (Shields, Murdoch, Loy, Dodd, & Taylor, 2006). Studies of Quality of Life (QOL) suggest lower QOL for children with CP, particularly in areas associated with CP and its sequella (e.g., academics, social interaction) and most notably for children with quadriplegia/more severe CP (Livingston, Rosenbaum, Russell, & Palisano, 2007). Transition to adulthood is understudied, but issues regarding reduced involvement in age-appropriate social activities and roles, employment, and leisure activities are of concern (Liptak, 2008).

Evaluation

Given the broad variability in etiology and the sparse knowledge base regarding neuropsychological functioning in CP, evaluations should be tailored to the child and situation. Prior to the evaluation, medical evaluations clarifying the child's vision and hearing will be critical. Assessment measures should be chosen carefully to avoid underestimates of function due to motor or verbal output problems (e.g., use of nonverbal tests of intelligence that minimize requirements for motor output) (Fennell & Dikel, 2001). In children with marked communication deficits, parents and educators may be interested in possible methods for alternative communication (e.g., picture exchange systems, complex computerized devices), a question which can be addressed through a multi-disciplinary evaluation including a psychologist, speech–language pathologist, and occupational therapist. For children who are less impaired, evaluations should include a broad overview of neuropsychological functions with attention to executive functioning, visual-perceptual processing, and academics. Assessments should include formal or informal evaluation of pain, fatigue, and psychological and behavioral functioning.

Treatment

Multidisciplinary treatment is considered the standard of care for children with CP (Braddom, 2007). Children are involved in medical and therapeutic treatments designed to decrease spasticity and increase function (e.g., botox injections, baclofen pump, surgical interventions, splinting and casting, physical and occupational therapies, early intervention services). Although promising, treatment efficacy is unknown for many of these interventions (Tupper, 2007).

Interventions developed from a rehabilitation psychology perspective are integral to the treatment plan, including behaviorally-based treatments to increase function and assessment and intervention of pain and fatigue. Constraint-induced movement therapy (CIMT) is a promising approach that attempts to overcome learned non-use of the affected limb and to promote use of the affected limb through brain re-organization (Hoare, Wasiak, Imms, & Carey, 2007). Patients are restrained from using the unaffected limb through a sling or glove for 2–3 weeks and behavioral techniques (e.g., shaping, massed practice, scaffolding, and positive reinforcement) are used to encourage use of the affected limb. Also promising are behavioral treatments that address problems with drooling (Van der Burg, Didden, Jongerius, & Rotteveel, 2007). Pain can be life-limiting and may be underrecognized due to communication deficits and atypical pain responses in children with CP (Houlihan, O'Donnell, Conaway, & Stevenson, 2004). Adaptation of common self-report pain assessment scales and/or use of measures that have been developed for children who have communication problems may help the team to adequately evaluate and treat pain (Hadden & von Baeyer, 2005). Fatigue due to increased energy expenditure during daily tasks can also impact function and should be addressed (Berrin et al., 2007).

Cross References

- ▶ Assistive Technology
- ▶ Augmentative and Alternative Communication
- ▶ Constraint Induced Therapy
- ▶ Encephalopathy
- ▶ Hemiparesis
- ▶ Hemiplegia
- ▶ Interdisciplinary Team Rehabilitation
- ▶ Periventricular Leukomalacia
- ▶ Prematurity and Low Birthweight

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Cerebral Perfusion Pressure

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Definition

Cerebral perfusion pressure (CPP) is the net pressure of flow of blood to the brain, which is the difference between the mean arterial pressure (MAP) and the intracranial pressure (ICP).

Cross References

► Cerebral Blood Flow
► Intracranial Pressure

References and Readings

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Cerebral Seizures

► Epilepsy

Cerebral Thrombophlebitis

► Central Venous Thrombosis

Cerebral Vasculitis

► Cerebral Angiitis

Cerebral Venous Thrombosis

► Central Venous Thrombosis

Cerebral Ventricles

► Ventricles

Cerebrovascular Accident (CVA)

► Stroke

Cerebrovascular Dementia

► Multi-infarct Dementia

Cerebrovascular Disease

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Definition

Cerebrovascular disease refers to the group of conditions characterized by disease of the blood vessels that supply blood to the brain. It can occur in the blood vessels that lead to the brain or in the blood vessels inside the brain. While it usually presents with symptoms of a stroke (the group of clinical manifestations of cerebrovascular disease), it can be asymptomatic, in which case it is usually detected either by physical examination or selected imaging techniques.

The term "cerebrovascular accident" (or CVA) is incorrect and should be avoided, as there is nothing accidental about a stroke. The term "cerebrovascular disease" is a more general term than is "stroke," because "cerebrovascular disease" includes asymptomatic or subclinical disease, in addition to the clinically manifest strokes.

Most cerebrovascular disease is obstructive in nature, caused by atherosclerotic plaques that line the blood vessel walls and block the blood flow. If the blockage is only partial, and is not severe enough to impair brain function, then the disease remains asymptomatic. However, if the obstruction is severe enough to reduce blood supply to the extent that brain injury occurs, then symptoms suggesting a stroke ensue. If these symptoms are temporary and completely reversed, the phenomenon is known as a "transient ischemic attack." If the brain injury is irreversible, this causes a completed "stroke," which is defined as the neurological manifestations of disease of blood vessels of the brain. Other forms of cerebrovascular disease also can occur. Inflammation of blood vessel walls ("vasculitis"), bleeding into the cerebral vessel walls ("dissection"), and hemorrhage, or extravasation of blood outside of the vessels themselves and into the brain tissue, also can cause brain damage. These can give rise to strokes.

Cross References

► Atherosclerosis
► Cerebral Angiitis
► Cerebral Embolism
► Dissection
► Hemorrhagic Stroke
► Infarction
► Intracerebral Hemorrhage
► Ischemic Stroke
► Stroke
► Subarachnoid Hemorrhage
► Thrombosis
► Transcranial Doppler Ultrasonography
► Vascular Dementia
► Vascular Malformation
► Vasculitis

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Cerebrum Surface

► Cerebral Cortex

CES-D

► Center for Epidemiological Studies–Depression

CFL Test

- ▶ Controlled Oral Word Association Test
- ▶ F-A-S Test
- ▶ Verbal Fluency

CGY

- ▶ Centigray

Chandler Exterminators v. Morris (1992)

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Definition

The testimony of neuropsychologists is commonly challenged by defense attorneys in cases relating to inferences of subtle brain changes associated with neurotoxic brain injury. In the case of *Schudel v. General Electric* (1995), plaintiffs accepted neuropsychological evidence for brain damage caused by organic solvents and polychlorinated biphenyls (PCBs). However, the federal appeals court from the Ninth Circuit ruled that neuropsychological testimony is limited only to damages and cannot determine physical causation. The court opined that determination of causation is relegated to medical doctors (MDs) or left to the discretion of the jury to make connections between neurocognitive deficits presented and exposure to toxins. In the case of *Chandler Exterminators v. Morris* (1992), the Georgia Supreme Court ruled in favor of the trial court's decision to prohibit neuropsychological testimony that proposed a link between neurotoxicants and impaired neuropsychological test scores. In response to this case, Georgia legislature wrote a new law permitting neuropsychologists to provide testimony related to causation of brain injuries in Georgia. Challenges to specific neuropsychological tests and test batteries occur with some degree of frequency, and they should be taken seriously by all parties involved, especially neuropsychologists. These types of challenges question the scientific basis of one or more of the expert's methods, measures, or conclusions. Challenges to specific methods are brought under *Frye v. United States* (1923) and *Daubert v. Merrell Dow* (1993) rulings.

Cross References

- ▶ *Daubert v. Merrell Dow*

References and Readings

Chandler Exterminators Inc. v. Morris, 200 Ga. App. 816 (1992).
Schudel v. General Electric, 120 F. 3d 991 (1995).

Chapple v. Ganger

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Historical Background

In the cause of *Daubert v. Merrell Dow* (1993), it was ruled that for scientific testimony to be admissible, it has to be: (a) scientifically valid, and (b) relevant to the case at hand. The court provided a list of guidelines intended to aid in the determination of scientific validity (e.g., peer reviewed, fasifiability, acceptable error rate, etc.). The *Daubert* ruling along with subsequent related rulings (e.g., *General Electric v. Joiner,* 1997, *Kumho Tire v. Carmichael,* 1999), generated significant debate among psychologists and neuropsychologists, and many other disciplines. Specifically, Reed (1996) viewed the *Daubert* ruling to necessitate the utilization of commercially available fixed batteries only, such as the Halsteid–Reitan Battery. However, most neuropsychologists employ a flexible battery approach; thus, contradicting Reed's assertions implying that most neuropsychologists would not be suited for involvement in forensic work. In support of his conclusion, Reed referenced the case of *Chapple v. Ganger (1998)*, a brain injury claim. Review of the judge's written decision in *Chapple v. Ganger* outlined that all

neuropsychological testimony (even *partial* HRB protocols from two other neuropsychologists) was admitted into evidence and the fact that the judge had placed more emphasis on testimony from a fixed battery advocate was completely unrelated to the determination of the test battery. Indeed, there was no *Daubert* challenge to a flexible test battery approach. In this particular case, only the testimony of a vocational specialist who conducted no testing and provided no evidence in the form of a peer-reviewed study to support his claims, was subject to a *Daubert* hearing from the defense.

References and Readings

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Charles Bonnet Syndrome

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Synonyms/Associated Terms

Bonnet syndrome

Short Description or Definition

Charles Bonnet syndrome (CBS) is characterized by the following features (Eperjesi & Akbarali, 2004):

1. The presence of well-formed, complex, repetitive or persistent visual hallucinations
2. Full or partial retention of insight into the unreal nature of the hallucinations
3. Absence of hallucinations in other sensory modalities (e.g., auditory, olfactory)
4. Absence of delusions

Categorization

The images associated with CBS are often rich in detail, and their clarity frequently contrasts sharply with sufferers' blurred perception of real objects (Menon, Rahman, Menon, & Dutton, 2003). They are sometimes referred to as "pseudo-hallucinations" to indicate that the person experiencing them is aware that the images are not real. Hallucinations may vary greatly in terms of color, clarity, movement, and bizarreness (Plummer, Kleinitz, Vroomen, & Watts, 2007). Nevertheless, common themes and figures have been described, including humans and animals, extended landscapes, and ornate structures (Plummer et al., 2007). Ffytche and Howard (1999) classified their patients' hallucinations into eight categories (Table 1).

While the clinical validity of this classification system has not yet been established, it does share some similarities with known functional and anatomical networks within the visual association cortex (Plummer et al., 2007).

Epidemiology

1. *Prevalence*: Once considered rare, CBS is becoming increasingly more common (Rovner, 2006). The larger number of reported CBS cases may be related to the growing population of older adults and the more common occurrence of visual disorders such as age-related macular degeneration, glaucoma, and cataracts (Rovner, 2006). It has also been argued that past prevalence estimates of CBS were spuriously low due to a

Charles Bonnet Syndrome. Table 1

Hallucination Category	Description
Tessellopsia	Regular, overlapping patterns
Hyperchromatopsia	Hyperintense, vivid, brilliant colors
Prosopometamorphopsia	Facial distortions
Dendropsia	Branching forms
Perseveration	True percept that persists after the individual looks away
Illusory visual spread	Spread of a non-hallucinated pattern
Polyopia	Multiple copies of a percept
Micropsia/macropsia	Miniaturized/"larger than life" images

general lack of awareness of the syndrome in the medical community, as well as patients' reluctance to disclose their hallucinatory symptoms for fear of being labeled psychotic or demented (Plummer et al., 2007). One estimate suggests that the prevalence of complex visual hallucinations in patients with visual impairment is between 11% and 15% (Menon et al., 2003).

2. *Age of onset*: CBS may occur at any age, but it is more common in the elderly. Average age of onset tends to be in the 70s and 80s (Plummer et al., 2007). The increased prevalence of CBS in older adults is likely related to the greater incidence of sudden visual loss and/or isolation in this age group (Menon et al., 2003).

Natural History, Prognostic Factors, Outcomes

Historical background: Charles Bonnet syndrome is named after the eminent Swiss philosopher and naturalist who first described this phenomenon in 1760 (Hedges, 2007). In a book entitled *Essai Analytique sur les Facultiés de L'Ame* [Analytical Essays Concerning the Faculties of the Mind], Charles Bonnet described how his cognitively intact, 89-year-old grandfather with failing eyesight began to experience well-formed visual hallucinations, which he was aware were not actually physically present. Interestingly, Charles Bonnet began to have similar experiences later in his own life. At the age of 22, he began to experience severe eye pain and progressively worsening loss of vision that made it difficult for him to use a microscope, and he ultimately turned to more abstract philosophical pursuits and theoretical questions in biology. In his retirement, he experienced formed visual hallucinations associated with many of the common attributes of the syndrome that shares his name, including blindness, intact cognition, and occurrence in quiet and reflective settings. Another native of Geneva, George de Morsier, proposed in 1967 that visual hallucinations in older men without mental deficiency be designated the syndrome of Charles Bonnet.

Current thinking/prognostic factors: The question of whether visual impairment is necessary for the development of CBS has been the matter of debate. Some argue that CBS is almost invariably associated with impaired vision, and it may occur whenever sensory input to the brain is decreased sufficiently to allow release phenomena. Other researchers report that visual dysfunction, though common, is not mandatory for diagnosis, and note that CBS has been found in individuals with intact vision (Terao & Collinson, 2000).

Charles Bonnet Syndrome. Table 2

Factors Favoring the Recurrence of Hallucinations
Dimly lit conditions
States of drowsiness
Physical and social isolation

Charles Bonnet Syndrome. Table 3

Factors that May Help Relieve Hallucinations
Rapid blinking
Sustained eye closure
Diversionary activities
Limiting exposure to dim lighting
Walking away
Looking at or approaching images

It has been argued that CBS is more commonly associated with higher degrees of visual impairment, and with bilateral as opposed to unilateral ocular pathology (Menon et al., 2003). It has also been suggested that it is not the specific lesion site or the severity of impairment, but rather the rate of development of the visual impairment that best predicts CBS (Plummer et al., 2007). That is, hallucinations may be more common in the context of sudden or unexpected decrease in visual function (Menon et al., 2003).

Outcomes: The course of CBS can be unpredictable. While the onset is generally sudden, it may also be gradual (Menon et al., 2003). Hallucinations can last from seconds to hours, or even days. Clustering of episodes across days or weeks is not unusual. Three patterns of the syndrome have been described (Menon et al., 2003). The *episodic* pattern, characterized by hallucinations that happen over a period of days to months and then permanently cease, is reportedly the least common. In the *periodic* pattern, phases of hallucinatory activity alternate with phases of remission. The *continuous* pattern, as its name suggests, is characterized by unremitting hallucinations (i.e., no hallucination-free intervals) (Menon et al., 2003). Overall, the duration of CBS can extend from days to years, and spontaneous recurrence after a symptom-free interval is possible. Interestingly, some sufferers have reported permanent remissions of CBS in conjunction with ongoing visual decline (Plummer et al., 2007).

Neuropsychology and Psychology of Charles Bonnet Syndrome

The exact pathophysiology of CBS is unclear. Although Charles Bonnet suggested that the primary pathology was restricted to the eye, subsequent research has suggested that complex hallucinations can occur in the context of visual impairment secondary to pathology anywhere along the central visual pathway, from the orbit to the occipital cortex (Menon et al., 2003). A leading hypothesis is that complex visual hallucinations result from deafferentation of the visual association cortex following lesions among the central visual pathway. Age-related macular degeneration is a commonly cited cause of CBS, but other ocular pathologies include glaucoma, central retinal artery occlusion, and optic neuritis (Plummer et al., 2007). Extraocular and central visual axis pathologies associated with CBS include lesions of the pituitary and optic chiasm, meningioma, and occipital stroke.

Evaluation

The evaluation of CBS should begin with a clinical interview, which should be approached carefully in light of sufferers' frequent reluctance to disclose hallucinatory experiences. The clinician should assess the nature of the hallucinations, the modalities in which they occur, the presence of delusions, and the patient's insight. It is important to note that insight into the illusory nature of the hallucinations may not occur immediately; in fact, there may be a period of initial deception, especially if the perceived images are not uncommon and fit realistically into the patient's surroundings (Menon et al., 2003). Referrals to an ophthalmologist, low-vision specialist, and neuropsychiatrist may be helpful.

A key issue for clinical neuropsychologists is the differential diagnosis of CBS from other causes of visual hallucinations. Conditions belonging in the differential include migraine, occipital seizures, peduncular hallucinosis (usually from rostral brainstem infarct), drug-induced states, psychiatric disease, delirium, and dementia. In particular, CBS may occur in the early stages of dementia with Lewy bodies (DLB), and as cognitive function declines, insight about the unreal nature of the hallucinations vanishes (Terao & Collinson, 2000). Some have suggested the term *Charles Bonnet Plus or CBS plus* to describe visual hallucinations that occur in the presence of a neuropsychiatric disorder (Eperjesi & Akbarali, 2004; Menon et al., 2003).

Treatment

Treatment for CBS may not always be necessary, since visual hallucinations often resolve spontaneously, either in response to improvement or further deterioration of visual function (Menon et al., 2003). In addition, many patients are not distressed by their hallucinations and may even enjoy them. However, if hallucinations are frequent or distressing, the following treatment options are available:

1. *Optimizing visual acuity*: Patients should initially be referred to a low-vision specialist, who may be able to reduce or alleviate hallucinations by optimizing visual function (e.g., via prescription eyeglasses or visual aids). If appropriate, the patient might be considered for surgery (e.g., cataract surgery or neurosurgical procedures). Several reports indicate that improvement of visual function, either spontaneously or by intervention, can effectively decrease or even eliminate hallucinations (Menon et al., 2003).
2. *Supportive treatment*: A key component in the management of CBS is supportive. Patients may derive comfort from sympathetic explanations that their hallucinations are not uncommon, are not necessarily a marker of psychiatric disease, and may represent a release phenomenon in the context of visual impairment. Using the analogy of "phantom visions," similar to a phantom limb syndrome, may be helpful (Rovner, 2006).
3. *Psychotherapeutic strategies*: Psychotherapeutic techniques used for phantom limb pain, including distraction, hypnosis, relaxation training, and cognitive restructuring can help reduce the unpleasant effects of intrusive and upsetting visual hallucinations (Menon et al., 2003). Support or psychoeducational groups are useful settings in which sufferers can meet, obtain reassurance, and be given advice about specific techniques for reducing hallucinations (Eperjesi & Akbarali, 2004).
4. *Behavioral/environmental modifications*: Approaches such as rapid eye blinking, sustained eye closure, minimizing fatigue and stress, and engaging in distracting activities (e.g., listening to the radio and attending to household chores) may help reduce hallucinations. Limiting exposure to dim lighting (e.g., by increasing lighting in the home in the evening) and taking steps to reduce glare may also be helpful. Looking directly at the images, attempting to approach them, and conversing with them have also been reported to stop hallucinations (Menon et al., 2003).

Since solitude and loneliness, particularly during the evening hours, tend to heighten hallucinations, strengthening social networks and increasing the amount of time spent interacting with others may be useful (Plummer et al., 2007).

5. *Pharmacological interventions*: Referral to a specialist for pharmacological therapy may be helpful. A few case studies have reported efficacy of pharmacological agents in alleviating symptoms, such as sodium valproate, olanzapine, and carbamazepine (Plummer et al., 2007). Use of other medications (e.g., risperidone, gabapentin, and diazepam) has also been described (Eperjesi & Akbarali, 2004).
6. *Follow-up*: Since some cases of CBS do go on to develop dementia, it is recommended that clinicians follow patients with complex visual hallucinations carefully over time (Menon et al., 2003). Though most patients experience no practical problems associated with CBS, continuous visual hallucinations can interfere with navigation and driving, and patients' ability to perform daily activities safely should be monitored over time (Menon et al., 2003).

Cross References

- ▶ Dementia with Lewy Bodies
- ▶ Macropsia
- ▶ Micropsia
- ▶ Visual Hallucinations

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CHART

▶ Craig Handicap Assessment and Reporting Technique

CHART Short Form

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Synonyms

CHART-SF; Craig handicap assessment and reporting technique (CHART) short form

Definition

The Craig Handicap Assessment and Reporting Technique Short Form (CHART-SF) is a 19-item measure of handicap or level of societal participation. Released in 1998, the CHART-SF is the short version of the 32-item CHART instrument designed to provide a simple, objective measure of the degree to which impairments and disabilities result in handicaps (societal participation limitations) for adolescents and adults (15 years and older) in the years after initial rehabilitation. Like its precursor, the CHART-SF includes six subscales (physical independence, cognitive independence, mobility, occupation, social integration, and economic independence), which closely reflect the disablement model developed by the World Health Organization, published in 1980 and revised in 2001. Each subscale contains from 2 to 5 questions, which together quantify the extent to which individuals fulfill various social roles. CHART-SF focuses on objective, observable criteria that are easily quantifiable and unlikely to be open to subjective interpretation. Each of the domains or subscales of the instrument have a maximum score of 100 points, which is considered the level of performance typical of the average non-disabled person. High subscale scores indicate less handicap, or higher social and community participation.

Although originally developed for use with persons with spinal cord injury, the CHART and the CHART-SF have proven to be appropriate measures of societal participation that can be used with individuals having a

range of physical or cognitive impairments. The CHART-SF was designed to be administered by interview, either in person or by telephone and takes approximately 5–7 min to administer. There is no set time period for administering the CHART-SF; however, it is recommended that multiple measurements be taken over the course of a person's lifetime to assess the changes with adaptation to the disability and to gain insight into changes in participation, which may occur over time.

The 19-item CHART-SF with subscales closely approximating the subscale scores for the CHART long form is recommended for those populations for whom time is at a minimum.

Current Knowledge

In an effort to reduce the number of items in the original CHART, a short form was developed. A multidimensional analysis was performed which showed that fewer variables were needed to obtain CHART scores. Regression analyses were performed on each subscale with the dependent measure being the scale score and the variables contributing to the subscale acting as the predictor variables. All CHART subscale scores could be reduced by fewer questions to reach 90% explained variance except Economic Self-Sufficiency, which using the main variables could only explain 45%.

Cross References

► Craig Handicap Assessment and Reporting Technique

References and Readings

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CHART-SF

► CHART Short Form
► Craig Handicap Assessment and Reporting Technique

CHEIs

► Cholinesterase Inhibitors

Chelation

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Synonyms

EDTA therapy

Short Description or Definition

Chelation therapy has been used in allopathic and in complementary and alternative (CAM) forms of medicine. Claims that chelation therapy with ethylene diamine tetraacetic acid (EDTA) is an effective technique for controlling and treating cardiovascular disease are not supported by systematic reviews of the literature (Ernst, Pittler, Stevinson, White & Eisenber, 2001; Seely, Wu & Mills, 2005). However, Chelation therapy does appear to be highly effective and is the treatment of choice in treating heavy metal poisoning (Ernst et al., 2001). Recent research also suggests that Chelation therapy may have applications in the treatment of malaria (Mabeza, Lovevsky, Gordeuk & Weiss, 1999). The therapy involves the intravenous administration of EDTA, which binds ions in the blood, and is often used in combination with vitamins, trace elements, and iron supplements.

Categorization

While Chelation therapy has been used for treating lead poisoning and vascular occlusive disease, oral chelation therapy with the α-ketohydroxypyridine chelator 1,2-dimethyl-3-hydroxypyrid-4-one (L1, INN/BAN: deferiprone) has also been used in iron- and aluminum-overloaded patients. A number of iron(III) chelators have also shown antimalarial activity *in vitro* with the proposed mechanism of activity involving the withholding of iron from metabolic pathways of the intra-erythrocytic parasite and the formation of toxic complexes with iron.

History

Chelation therapy began in the early 1950s and was primarily used to treat metal poisoning of the blood. However, both allopathic and CAM practitioners have claimed that the technique can be used to reverse the arteriosclerotic disease process (e.g., peripheral arterial occlusive disease (PAOD)).

Evaluation

Studies examining the efficacy of Chelation therapy have shown mixed results. In one authoritative systematic review, it was stated that proponents of chelation therapy adhere to pathophysiological models of arteriosclerosis, which are inconsistent with current knowledge and practice (Ernst et al., 2001). In a systematic review of chelation therapy in the treatment of malaria, it was concluded that when used via oral administration, its efficacy and low cost make it more accessible than desferrioxamine for the majority of patients needing iron chelation (Mabeza et al., 1999).

Treatment and Mechanisms

Typically, Chelation therapy is administered in multiple sessions with each treatment lasting for over an hour. The putative mechanism underlying chelation therapy is the binding of ions in the blood by EDTA. The therapy is, generally, considered to be effective in heavy metal poisoning. In CAM applications, it has been used as an alternative to bypass surgery, based on the idea that chelation therapy removes harmful plaque build-up in the arteries (unblocking arteriosclerotic arteries) and helps prevent strokes. The mechanism is believed to involve the extraction of calcium out of arteriosclerotic plaques via the chelating mechanism. In a systematic review of randomized, placebo-controlled, double-blind trials, it was concluded that Chelation therapy for PAOD is not superior to placebo, that it is associated with considerable risks and costs, and that it should now be considered obsolete (Ernst et al., 2001). However, it should also be noted that using oral chelation therapy, in doses ranging from 55 to 100 mg kg^{-1} of L1 (α-ketohydroxypyridine chelator 1,2-dimethyl-3-hydroxypyrid-4-one (L1, INN/BAN: deferiprone)), a majority of iron-loaded patients showed urinary iron excretion levels greater than those accumulating from transfusions (15–35 mg d^{-1}) and also reductions in serum ferritin and liver iron to near normal levels (Kontoghiorghes, 1995). Toxic side effects include six cases of reversible agranulocytosis, 0–30% incidence of transient musculoskeletal and joint pains, 0–6% of gastric intolerance, and 0–2% zinc deficiency. In the treatment of malaria, iron chelation therapy with desferrioxamine, which is the only compound of this nature that is available for use in humans, has shown clinical activity in both uncomplicated and severe malaria in humans (Mabeza et al., 1999).

Adverse Side Effects

In one systematic review, adverse effects were characterized as rare but cases of hypocalcemia and a single case of increased creatinine was noted in a patient on the EDTA intervention (Seely, Wu & Mills, 2005). Moreover, it was emphasized that if the treatment is used in lieu of proven therapies, indirect harm to the patient could result. In another systematic review, it was reported that EDTA treatment may be associated with life-threatening adverse effects, such as hypocalcemia and severe kidney damage, in addition to prolonged bleeding and respiratory distress (Ernst et al., 2001).

Cross References

▶ Lead Exposure

References and Readings

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Chemical AIDS

▶ Multiple Chemical Sensitivity

Chemical Hypersensitivity Syndrome

► Multiple Chemical Sensitivity

Chemotherapy

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Synonyms

Systemic therapy

Definition

Chemotherapy is a systemic treatment for cancer, comprising cytoxic agents that target cancer cells. Normally, cells develop and die in an orderly and determined fashion. However, when cancer manifests itself, the cells intractably divide and proliferate. Chemotherapy drugs target these cancer cells by destroying them before they continually multiply and divide. In particular, chemotherapy interferes with or impairs the targeted molecules (e.g., DNA, proteins) during designated cellular stages, such as synthesis or mitosis. The majority of chemotherapy drugs damage or interfere with the replication of DNA and/or RNA, and are used to treat several malignancies, particularly brain tumors, lymphomas, and leukemias. Some of the chemotherapies include: alkylating agents (such as cisplatin, carboplatin, cyclophosphamide, and temozolomide) to treat brain tumors, lymphomas, and leukemias; nitrosoureas (e.g., carmustine and lomustine) are indicated for the treatment of brain tumors and lymphomas; antimetabolites (such as methotrexate) to treat leukemias; anthracycline and related drugs (e.g., doxorubicin), which have toxic effects on the heart; topoisomerase inhibitors (such as topotecan, irinotecan, and etoposide); mitotic inhibitors (e.g., vinblastine and vincristine), which can cause peripheral nerve damage; and corticosteroid hormones, which can be used to kill or slow the growth of cancer cells. There are other chemotherapies that are excluded from these categories, namely L-asparaginase, -hydroxyurea, and -thalidomide. The specific manner in which chemotherapy achieves the intended effect is contingent upon the particular drug(s) employed. However, cytotoxicity usually occurs when the cell attempts to divide and before the repair occurs (Chabner & Longo, 2004). The probability of the intended effect on the targeted molecules reflects the appropriate concentration of drugs, amount or dose and timing of drug administration. Drug absorption, distribution, and penetration are also significant factors inherent in the efficacy of chemotherapy. The precise drug dosage can be complicated because if the amount is too low, it may be ineffective against the tumor. Conversely, if the dosage is too high, patients may suffer from excessive toxicity. Since chemotherapy damages healthy cells during the therapeutic process, the treatment is associated with several harmful side effects, such as myelosuppression. For example, bone marrow, which produces white blood cells, red blood cells, and blood platelets, can be damaged during chemotherapy treatment. In particular, white blood cells and platelets frequently drop transiently after chemotherapy, so that patients are at increased risk for infection and bleeding during and post chemotherapy. Many chemotherapy patients also suffer from nausea and vomiting because the drugs irritate the stomach lining and bowel. Certain chemotherapy drugs also cause alopecia, or hair loss. This condition results from the chemotherapy agent adversely affecting the growth of hair cells, causing them to become brittle and eventually break. Several chemotherapies also result in anorexia, severe loss of appetite, and significant weight loss. Fatigue, diarrhea, and constipation are also very common side effects from cancer and chemotherapy. Additionally, specific chemotherapy agents can cause stomatitis, a condition that results in sores manifesting inside the mouth or throat. Chemotherapy is most often given intravenously, whereby a thin needle is inserted into a patient's vein on the hand or lower arm. Intravenous chemotherapy can also be delivered through catheters, ports, and pumps. The treatment is frequently given in cycles (i.e., specified treatment periods) that reflect alternating rest periods. This is necessary because patients require substantial relief to permit the body to recuperate, build healthy new cells, and restore strength. Treatment regimens may be given daily, weekly, or monthly and are based upon a drug's efficacy or toxicity. Chemotherapy is usually administered before (neo-adjuvant) surgery or post (adjuvant) surgery. Neoadjuvant chemotherapy is intended to decrease the primary tumor's size. This potentially mitigates the harmful effects of surgery or radiotherapy and enhances the efficacy of chemotherapy.

Adjuvant chemotherapy may also reduce the probability of tumor resistance to drug therapy in the event of disease recurrence (Chabner & Longo, 2004). Furthermore, adjuvant chemotherapy is effective at destroying residual cancer cells that have spread to distal parts of the body (i.e., metastasis), particularly because rapidly proliferating lesions are very amenable to treatment. Palliative chemotherapy is indicated when the curative potential is very low and the primary goals are to decrease the patient's tumor burden and prolong life expectancy.

Current Knowledge

Recent neuropsychological research has indicated that chemotherapy can also adversely impact cognitive functioning, both short-term and delayed. In particular, neuropsychological research studies have provided evidence discussing the impact of chemotherapy on attention, memory, and concentration (Armstrong, Gyato, Awadalla, Lustig, & Tochner, 2004). Consequently, many of these chemotherapy-induced cognitive impairments can significantly impair patients' daily activities, such as working, being involved in a committed relationship, and attending to personal responsibilities. Research has suggested that many of these impairments are temporary but some may be more long-term, or even permanent. The cognitive effects are not uniform, and the severity appears to reflect a higher concentration and/or larger dose of chemotherapy.

Most of the different types of chemotherapy drugs damage or interfere with the replication of DNA and/or RNA, and are used to treat several malignancies, particularly brain tumors, lymphomas, and leukemias. Some of the chemotherapies include: alkylating agents (such as cisplatin, carboplatin, cyclophosphamide, and temozolomide) to treat brain tumors, lymphomas, and leukemias; nitrosoureas (e.g., carmustine and lomustine) to treat brain tumors and lymphomas; antimetabolites to treat leukemias (such as methotrexate); anthracycline and related drugs (e.g., doxorubicin), which have toxic effects on the heart; topoisomerase inhibitors (such as topotecan, irinotecan, and etoposide); mitotic inhibitors (e.g., vinblastine and vincristine), which can cause peripheral nerve damage; and corticosteroid hormones, which can be used to kill or slow the growth of cancer cells. There are other chemotherapies that are excluded from these categories, such as L-asparaginase, -hydroxyurea, and -thalidomide. The specific manner in which chemotherapy achieves the intended effect is contingent upon the particular drug(s) employed. However, cytotoxicity usually occurs when the cell attempts to divide and before repair occurs (Chabner & Longo, 2004).

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Treatment regimens may be given daily, weekly, or monthly and are based upon a drug's efficacy or toxicity. Chemotherapy is usually administered before (neo-adjuvant) surgery or post (adjuvant) surgery. Neo-adjuvant chemotherapy is intended to decrease the primary tumor's size. This potentially mitigates the harmful effects of surgery or radiotherapy and enhances the efficacy of chemotherapy.

Adjuvant chemotherapy is employed when there is scant evidence of residual disease, but there is an increased

risk of cancer recurrence. Adjuvant chemotherapy may also reduce the probability of tumor resistance to drug therapy in the event of disease recurrence (Chabner & Longo, 2004). Furthermore, adjuvant chemotherapy is effective at destroying residual cancer cells that have spread to distal parts of the body (i.e., metastasis), particularly since rapidly proliferating lesions are very amenable to treatment. Palliative chemotherapy is indicated when the curative potential is very low and the primary goals are to decrease the patient's tumor burden and prolong life expectancy.

Cross References

► Systemic Therapy

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Chief Sensory Nucleus of V

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Synonyms

Principal sensory nucleus of the trigeminal nerve; Principal sensory nucleus of V

Definition

Nucleus responsible for proprioceptive feedback from the muscles of facial expression, stereognosis or fine tactual discrimination, and vibratory sensations from the face. Located in the dorsolateral pons just medial to the middle cerebellar peduncle and inferior to the superior cerebellar peduncle, it is the functional equivalent of the nuclei cuneatus and gracilis in the medulla, which mediate similar input from the trunk and extremities. It gives rise to trigeminothalamic fibers, which terminate in the ventral posterior medial nucleus of the thalamus.

Current Knowledge

Because of its size and density, it is rare for brainstem lesions to be isolated to a single nucleus or pathway. Theoretically, lesions which involve this nucleus might most readily be distinguished on a routine neurological exam by changes (asymmetries) in two-point discrimination on the ipsilateral face. In practice, however, such lesions are likely to involve other brainstem nuclei and pathways, including the adjacent motor nucleus of V, spinal trigeminal tract and/or nucleus, spinal thalamic tracts, lateral portions of the medial lemniscus, and middle cerebellar peduncles resulting in ipsilateral muscle weakness of the jaw muscles, ipsilateral changes in pain and temperature in the face and diminished or abolished corneal reflex, contralateral loss of pain and temperature in the extremities, diminished or loss of proprioception, stereognosis, and vibration in the contralateral extremities (leg > arm), and ipsilateral cerebellar signs.

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Child Behavior Checklist

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Synonyms

ASEBA; CBCL

Description

The Achenbach System of Empirically Based Assessment (ASEBA) comprises a family of forms for rating

behavioral/emotional problems and adaptive characteristics. For ages 1½ to 90+ years, developmentally appropriate forms are designed to be completed by collaterals who know the person who is being assessed. These forms include versions of the Child Behavior Checklist (CBCL), completed by parent figures for 1½- to 5-year-olds and for 6- to 18-year-olds; the Caregiver-Teacher Report Form (C-TRF) for ages 1½–5, completed by daycare providers and preschool teachers; the Teacher's Report Form (TRF) for ages 6–18, completed by teachers and other school personnel; the Adult Behavior Checklist (ABCL) for ages 18–59, completed by spouses, partners, family members, friends, therapists, and other collaterals; and the Older Adult Behavior Checklist (OABCL) for ages 60 and older, completed by caregivers as well as by collaterals.

The ASEBA also includes parallel forms completed by the people being assessed, including the Youth Self-Report (YSR) for ages 11–18, the Adult Self-Report (ASR) for ages 18–59, and the Older Adult Self-Report (OASR) for ages 60 and older. The collateral and self-report forms assess functioning in everyday contexts over periods of 2–6 months.

In addition to the collateral and self-report forms, other ASEBA forms are designed for rating behavior observed in specific situations. These forms include the Direct Observation Form (DOF), which is completed by observers who rate two or more 10-min samples of children's behavior observed in classrooms and other group settings; the Semistructured Clinical Interview for Children and Adolescents (SCICA), which provides an interview protocol and a rating form completed by the interviewer who administers the SCICA to 6- to 18-year-olds and the Test Observation Form (TOF), which test examiners use to rate the behavior observed during the administration of individual ability and achievement tests to 2- to 18-year-olds. Table 1 summarizes the ASEBA forms, ages covered, who completes the forms, and references to manuals for each form.

Normed Profiles

Scores obtained from all ASEBA forms are displayed on profiles in relation to norms that are based on distributions of scale scores obtained by large samples of peers. For the collateral and self-report forms for ages 1½ to 90+ years, norms are based on a US national probability sample of people who had not received mental health or substance abuse services in the preceding 12 months. For the CBCL/6–18, TRF, and YSR, norms are provided for many cultures in addition to the USA, as detailed later (Achenbach & Rescorla, 2007a, b). Multicultural norms for the CBCL/1½–5 and C-TRF were released in 2010. For the DOF, SCICA, and TOF, norms are based on ratings of children observed in the contexts for which these instruments are designed.

Each profile displays an individual's scale scores in terms of standard scores (*T* scores) and percentiles based on the normative sample of that individual's peers, as rated by a particular type of informant (e.g., parent, teacher, self). The profiles also display demarcations between the normal range, borderline clinical range, and clinical range on each scale. Figure 1 illustrates a profile

Child Behavior Checklist. Table 1 ASEBA assessment instruments

Instrument	Ages	Completed by	Reference
CBCL/1½–5	1½–5	Parent figures	Achenbach and Rescorla (2000)
C-TRF	1½–5	Daycare providers, preschool teachers	Achenbach and Rescorla (2000)
CBCL/6–18	6–18	Parent figures	Achenbach and Rescorla (2001, 2007a)
TRF	6–18	Teachers	Achenbach and Rescorla (2001, 2007a)
YSR	11–18	Youths	Achenbach and Rescorla (2001, 2007a)
TOF	2–18	Psychological examiner	McConaughy and Achenbach (2004)
DOF	6–11	Observer	McConaughy and Achenbach (2009)
SCICA	6–18	Interviewer	McConaughy and Achenbach (2001)
ASR	18–59	Adults	Achenbach and Rescorla (2003)
ABCL	18–59	Collaterals	Achenbach and Rescorla (2003)
OASR	≥60	Older adults	Achenbach, Newhouse, and Rescorla (2004)
OABCL	≥60	Collaterals	Achenbach, Newhouse, and Rescorla (2004)

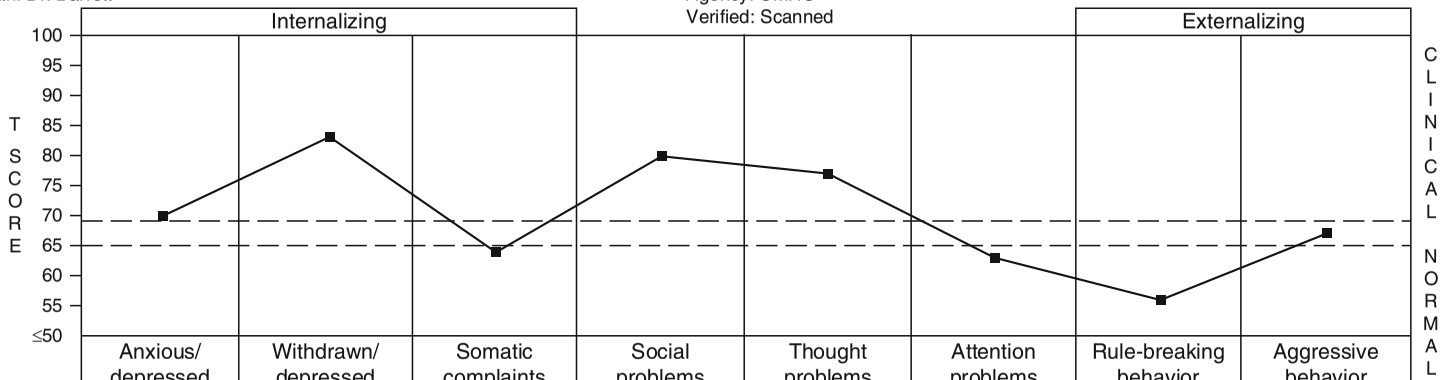
YSR/11–18 - Syndrome scale scores for Boys (2001 version)

ID: 2301251405–003
Name: Wayne webster
Clinician: Dr. Barrett

Gender: Male
Age: 15

Date filled: 01/08/2001
Birth date: 03/03/1986
Agency: CMHC
Verified: Scanned

Informant: Self
Relationship: Self



	Anxious/ depressed	Withdrawn/ depressed	Somatic complaints	Social problems	Thought problems	Attention problems	Rule-breaking behavior	Aggressive behavior
Total score	12	12	6	14	14	9	5	14
T score	70-C	83-C	64	80-C	77-C	63	56	67-B
Percentile	>97	>97	92	>97	>97	90	73	96
	1 14.Cries	2 5.EnjoysLittle	2 47.Nightm ares	2 11.Dependent	2 9.MindOff	0 1.ActsYoung	1 2.Alcohol	2 3.Argues
	0 29.Fears	2 42.PreferAlone	1 51.Dizzy	2 12.Lonely	0 18.HarmSelf	0 4.FailsToFinish	0 26.NoGuilt	1 16.Mean
	0 30.FearSchool	2 65.Won'tTalk	1 54.Tired	1 25.NotGetAlong	1 40.HearsThings	2 8.Concentrate	0 28.BreaksRules	0 19.DemAtten
	0 31.FearDoBad	2 69.Secretive	0 56a.Aches	0 27.Jealous	2 46.Twitch	0 10.SitStill	0 39.BadFriends	1 20.DestroyOwn
	0 32.Perfect	0 75.Shy	1 56b.Headaches	2 34.OutToGet	0 58.PicksSkin	2 13.Confused	0 43.LieCheat	0 21.DestroyOther
	2 33.Unloved	0 102.LacksEnergy	0 56c.Nausea	2 36.GetsHurt	2 66.RepeatsActs	1 17.Daydream	0 63.PreferOlder	1 22.DisobeyHome
	2 35.Worthless	2 103.Sad	0 56d.EyeProb	1 38.Teased	0 70.SeesThings	1 41.Impulsive	0 67.RunAway	1 23.DisobeySchl
	2 45.Nervous	2 111.Withdrawn	1 56e.SkinProb	2 48.NotLiked	2 76.SleepsLess	1 61.PoorSchool	1 72.SetsFires	1 37.Fights
	0 50.Fearful		0 56f.Stomach	1 62.Clumsy	0 83.StoresUp	2 78.Inattentive	0 81.StealsHome	0 57.Attacks
	0 52.Guilty		0 56g.Vomit	1 64.PreferYoung	1 84.StrangeBehav		0 82.StealsOther	0 68.Screams
	1 71.SelfConsc			0 79.SpeechProb	2 85.StrangeIdeas		1 90.Swears	1 86.Stubborn
	2 91.ThinkSuic				2 100.SleepProb		1 96.ThinksSex	2 87.MoodChang
	2 112.Worries						0 99.Tobacco	2 89.Suspicious
							0 101.Truant	0 94.Teases
							1 105.UsesDrugs	2 95.Temper
								0 97.Threaten
								0 104.Loud

B = Borderline clinical range; C = Clinical range

Broken lines = Borderline clinical range

Child Behavior Checklist. Figure 1 Syndrome profile scored from the Youth Self-Report completed by 15-year-old Wayne Webster (From Achenbach & Rescorla, 2001, p. 33)

of syndrome scales scored from the YSR completed by 15-year-old Wayne Webster (not his real name).

Scales on Which ASEBA Instruments Are Scored

ASEBA problem items are scored on scales for syndromes derived empirically via exploratory factor analyses (EFAs) and confirmatory factor analyses (CFAs). These empirically derived syndromes reflect patterns of problems found to co-occur in ratings by each kind of informant.

In addition to the syndrome scales, each form is scored on DSM-oriented scales constructed by having experts from many cultures select ASEBA problem items that are very consistent with particular diagnostic categories of the American Psychiatric Association's (1994) *Diagnostic and Statistical Manual-Fourth Edition* (DSM-IV). Like the syndrome scales, the DSM-oriented scales are displayed on profiles in terms of *T* scores, percentiles, and normal, borderline clinical, and clinical ranges. Most forms are also scored on scales comprising critical items that are of particular concern to clinicians.

The collateral and self-report forms are additionally scored on scales for favorable characteristics, such as competence, adaptive functioning, and personal strengths. The particular items and scales are geared to the developmental level of people being assessed and to the informants' knowledge of people being assessed. For example, parents of 6- to 18-year-olds provide data regarding their children's involvement in sports, nonsports activities, organizations, jobs and chores, friendships, and relationships with parents, siblings, and peers. Teachers provide data on children's academic performance and adaptive characteristics at school. For adults, data are requested regarding friendships, relations with spouse or partner, children, job, and enrolment in educational programs. Only the items relevant to the adult being assessed are scored. For example, adults who lack a spouse or partner, children, job, or enrolment in educational programs are not scored on those items. Adult forms also have normed scales for substance use.

Cross-Informant Comparisons

Meta-analyses have revealed that correlations between parent, teacher, and self-reports of children's problems are typically only low to moderate (Achenbach, McConaughy, & Howell, 1987). Consequently, professionals who work with children recognize the need to obtain reports from multiple informants. Meta-analyses of correlations between collateral and self-reports of adult psychopathology have also revealed only modest correlations that argue for using multi-informant data to assess adults (Achenbach, Krukowski, Dumenci, & Ivanova, 2005).

Because each informant may provide valid and useful information that differs from what other informants provide, data from multiple informants should be compared. Software for scoring ASEBA forms facilitates cross-informant comparisons by printing scores obtained from parallel forms on parallel profiles. In addition, it prints side-by-side comparisons of ratings by up to eight informants on all problem items that have counterparts on forms completed by different informants. It also prints *Q* correlations that measure the degree of agreement between each pair of informants and compares them with *Q* correlations between pairs of informants in large reference samples.

An especially useful kind of comparison between informants' reports is illustrated in Fig. 2. This is a comparison between syndromes scored from the YSR completed by Wayne Webster, CBCLs completed by Wayne's parents, and TRFs completed by three of Wayne's teachers. For each syndrome, such as the Anxious/Depressed syndrome shown in the upper left-hand corner, the bars reflect the magnitude of standard scores (*T* scores) obtained from ratings by each kind of informant. Because the *T* scores are based on ratings by each kind of informant for a normative sample of children, the height of the bar indicates the level of the problems reported by a particular kind of informant compared to problems reported by that kind of informant for a normative sample of children. For example, the leftmost bars indicate that the Anxious/Depressed syndrome scores obtained from CBCL ratings by Wayne's parents are above the top broken line compared to parents' CBCL ratings of a normative sample of adolescent boys. As scores above the top broken line are in the clinical range, the CBCL bars indicate that Wayne's parents reported more problems of this syndrome than were reported by parents of 97% of boys in the normative sample.

Multicultural Norms

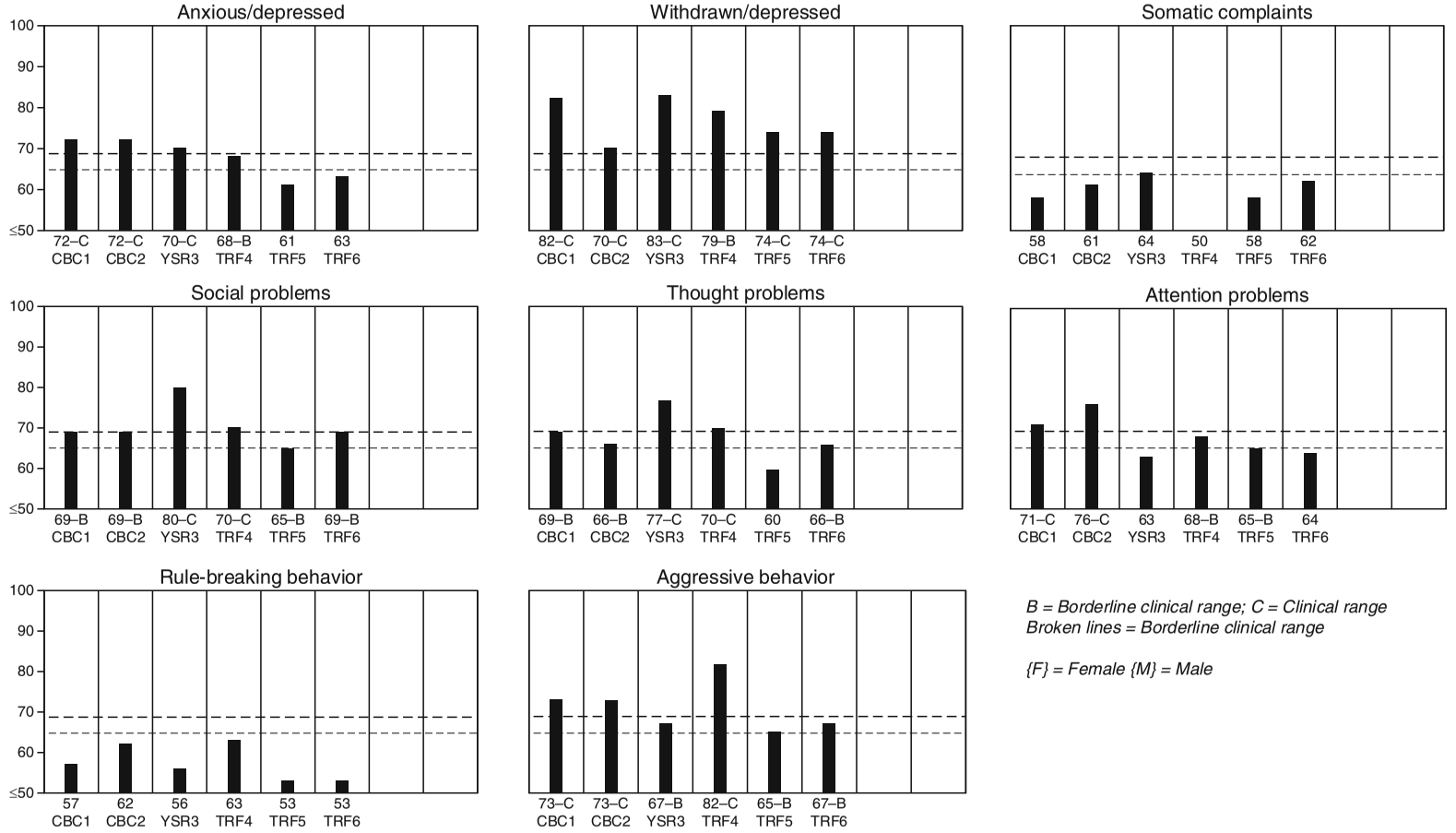
Norms obtained in one society may not be generalizable to other societies. To determine the degree of

Cross-informant comparison - CBCL/TRF/YSR Syndrome scale T Scores (2001 version)

ID: 2301251405 Name: Wayne webster Gender: Male Birth date: 03/03/1986 Comparison date: 04/13/2001

Form	Eval ID	Age	Informant name	Relationship	Date
CBC1	001	15	Alice N. Webster	Biological mother	04/04/2001
CBC2	002	15	Ralph F. Webster	Biological father	04/05/2001
YSR3	003	15	Self	Self	04/08/2001
TRF4	004	15	George jackson	Classroom teacher {M}	04/10/2001

Form	Eval ID	Age	Informant name	Relationship	Date
TRF5	005	15	Carmen Hernandez	Classroom teacher {F}	04/11/2001
TRF6	006	15	Charles Dwyer	Classroom teacher {M}	04/12/2001



B = Borderline clinical range; C = Clinical range
Broken lines = Borderline clinical range

{F} = Female {M} = Male

Child Behavior Checklist. Figure 2 Cross-informant comparisons of syndrome scores for Wayne Webster (From Achenbach & Rescorla, 2001, p. 39)

generalizability across societies, the same assessment instruments must be administered to large representative samples of people in different societies. This has been done with ASEBA instruments in many societies. CFAs of CBCL, TRF, and YSR data from many societies support the generalizability of syndromes that were initially derived from US samples. Comparisons of scale scores show that the distributions of CBCL, TRF, and YSR scores in many societies approximate those obtained in the USA. However, some societies have substantially lower or higher mean scores. To take account of societal differences in scale scores, separate sets of norms have been constructed for the societies obtaining relatively low scores, societies obtaining intermediate scores, and societies obtaining relatively high scores. Because parent, teacher, and self-ratings often yield different scores, the multicultural CBCL, TRF, and YSR norms were constructed separately. For some societies, problem scores obtained from one kind of informant are relatively low, while scores obtained from another kind of informant are intermediate or high. For example, CBCL and TRF problem scores obtained from Japanese parents and teachers are in the low range, whereas YSR scores obtained from self-ratings by Japanese youths are in the intermediate range.

To enable practitioners and researchers to compare CBCL, TRF, and YSR scores with culturally appropriate norms, the scoring software provides options for displaying problem scale scores in relation to norms for low-scoring, intermediate-scoring, and high-scoring societies. For example, CBCL and TRF scores for a Japanese youth would typically be displayed in relation to norms for low-scoring societies. However, the youth's YSR scores would be displayed in relation to norms for intermediate-scoring societies. If the Japanese youth lived in the USA and attended an American school, the TRF scores would be displayed in relation to US norms for teachers' ratings. If the youth's parents were well acculturated to the USA, the CBCL scores could be displayed in relation to US norms and also in relation to Japanese norms to see whether the scores were clinically deviant according to either set of norms.

Historical Background

The ASEBA stems from Achenbach's (1966) factor-analytic derivation of syndromes of child and adolescent psychopathology. Since then, over 4 decades of research and practical experience have produced ASEBA instruments for ages 1½ to 90+ years. Achenbach (2009) documents the historical development of ASEBA research, instruments, theory, and applications, as well as directions in which the ASEBA is now moving. Translations are available in 85 languages. Over 6,500 publications by some 9,000 authors report use of the ASEBA in 80 cultural groups and societies (Bérubé & Achenbach, 2010). ASEBA instruments are available in paper and Internet-based electronic versions in many countries around the world for practical assessment in clinical, educational, forensic, and other services, as well as for research on countless topics, such as genetics, medical conditions, outcome evaluations, epidemiology, development, diagnosis, and multicultural comparisons. Because the ASEBA's conceptual framework is open ended and generative, it continues to advance in multiple directions (Achenbach, 2009).

Psychometric Data

Table 2 summarizes psychometric data for all ASEBA instruments in terms of mean alphas, test-retest reliability, and the percentage of variance in ASEBA scale scores accounted for by clinical referral status, after partialing out demographic effects. Many additional psychometric findings – including goodness of fit obtained from CFAs in diverse samples – are reported in ASEBA manuals and in refereed publications listed by Bérubé and Achenbach (2010).

Clinical Uses

ASEBA instruments have numerous clinical uses. Bérubé and Achenbach (2010) list publications reporting use of the ASEBA in relation to over 150 medical conditions. Some 600 publications report use of the ASEBA for evaluating treatments and outcomes for many kinds of psychopathology and other problems.

ASEBA instruments can be used at many stages of clinical processes, including screening to identify needs for help, documentation of problems and adaptive functioning for use in clinical referrals, and intake assessment on which to base treatment decisions. During the course of treatment, ASEBA instruments are useful for determining whether goals are being met. Following the treatment, ASEBA instruments can be readministered to evaluate outcomes and subsequent functioning. At any point, ASEBA instruments can be used to assess behavioral/emotional concomitants of neuropsychological and medical disorders. The availability of similar ASEBA instruments for children and adults facilitates family

Child Behavior Checklist. Table 2 Summary of ASEBA psychometric data

Instrument	Alpha[a]		Reliability[b]		Validity[c]	
	Narrow	Broad	Narrow	Broad	Narrow	Broad
CBCL/1½–5	0.76	0.92	0.82	0.89	11	17
C-TRF	0.80	0.94	0.78	0.85	13	20
CBCL/6–18	0.83	0.94	0.88	0.92	24	32
TRF	0.86	0.94	0.84	0.90	15	20
YSR	0.78	0.92	0.78	0.85	10	14
TOF	0.82	0.90	0.76	0.84	9	14
DOF	0.68	0.79	0.51	0.72	7	20
SCICA	0.70	0.84	0.75	0.80	9	16
ASR	0.78	0.93	0.84	0.91	10	14
ABCL	0.80	0.94	0.84	0.88	6	8
OASR	0.80	0.96	0.86	0.95	13	20
OABCL	0.83	0.97	0.94	0.95	19	29

Narrow, mean for syndrome and DSM-oriented scales; broad, mean for internalizing, externalizing, and total problems. Data are from manuals listed in the Further Reading.

[a]Cronbach's coefficient *alpha* for the internal consistency of scales.

[b]Pearson *rs* for test-retest reliability over 8- to 16-day intervals. SCICA *rs* are between ratings by different interviewers who interviewed children over intervals averaging 12 days.

[c]Percentage of variance accounted for by clinical referral status (referred vs. nonreferred) in multiple regressions of referral status on ASEBA scale scores with demographic variables partialed out.

assessment, as well as close coordination between interventions for parents and their children.

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Childhood Autism

► Autistic Disorder

Childhood Autism Rating Scales

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Synonyms

CARS

Description

The childhood autism rating scale (CARS) is a 15 item measure intended to assist in distinguishing children with autism spectrum disorder (ASD) from children with other types of delays. It is an observational scale in which each item is rated from 1 (within normal limits) to 4 (severely abnormal) and ratings include consideration of "peculiarity, frequency, and duration" of the behavior rated (Schopler, Reichler, & Renner, 1988). It yields a total score ranging from 15 to 60. Scores of 30–36.5 suggest mild to moderate Autism and 37–60 suggest severe Autism. However, when used with adolescents and adults, the cut-off has been decreased to 28 (Schopler et al., 1988).

The CARS was initially developed using a sample of 1,606 children, approximately three quarters of whom were male. Sixty seven percent of the sample was white, 30% African American, and 3% was of other racial descent. Within the male and female samples, the age distribution was similar with 56% age 5 years or younger, 32% between ages 6 and 10 years, and 11% age over 10 years. Seventy one percent of the sample had an IQ (as determined by one of various measures) below 70, 17% with an IQ between 70 and 84, and 13% with an IQ above 84 (Schopler et al., 1988).

Historical Background

The CARS was developed at the University of North Carolina at Chapel Hill and was used to evaluate children referred to the TEACCH program. The CARS has been in use since 1971 (Reichler & Schopler, 1971) at which point it was called the childhood psychosis rating scale (CPRS) and the CARS was published as an appendix to the 1980 article by Schopler, Reichler, DeVellis, and Daly. It was originally intended for use by trained diagnosticians making ratings based on observation; however, the measure has been expanded for use by others (e.g., physicians, speech–language pathologists, special educators, and audiologists) as well as to include information from other sources such as parent report and classroom observations. It is important to note that the CARS was developed prior to the DSM-IV concept of Autism as a spectrum of disorders (Magyar & Pandolfi, 2008).

Psychometric Data

The CARS Manual (1988) describes high internal consistency reliability (alpha = 0.94) and an average interrater reliability of 0.71 for two independent raters across 280 cases. Test–retest reliability for 91 cases rated 1 year apart by two separate raters was 0.88. The sensitivity of the CARS was 100% for a sample of 54 children with autistic disorder in a study by Rellini et al. (2003). However, there is concern that the CARS may overdiagnose children with cognitive impairments as having autism with lower IQ scores correlating with higher CARS scores (Perry, Condillac, Freeman, Dunn-Geier, & Belair, 2005). In addition, sensitivity in the Rellini et al. study was not as great for distinguishing children with Asperger's disorder and pervasive developmental disorder-not otherwise specified (PDD-NOS) from children with attention-deficit/hyperactivity disorder (ADHD) and language delay. Despite the fact that the CARS was initially published during the time of the DSM-III, the CARS has shown good agreement with clinical diagnosis with the DSM-IV/ICD-10 criteria (Perry, Condillac, Freeman, Dunn-Geier, & Belair, 2005).

Several studies have looked at the relations between the CARS and other measures intended to assist in the diagnostic process. In one study, the CARS and the Autism Diagnostic Interview-Revised (ADI-R) were found to correlate 91.8% for cases in which a child received a diagnosis of Autism and 44.4% for those in which an Autism diagnosis was not given (Pilowsky, Yirmiya, Shulman, & Dover, 1998). More recently, Saemundsen, Magnusson, Smari, and Sigurdardottir (2003) found a correlation of 0.81 between the CARS (having a total score greater than 30) and the ADI-R (meeting cut-offs in all three domains) in a study that included 54 children. Several factor analyses of the 15 items comprising the CARS have been conducted. Magyar and Pandolfi (2007) found that four components: (1) a social component, (2) a negative emotionality construct, (3) a

sensory and stereotypy construct, and (4) an "activity level and consistency of intellectual response" component, accounted for 57% of the variance in ratings.

Clinical Uses

The CARS can be used as part of multimodal approach to the diagnosis of ASD that "includes observation of the child, caregiver interview, assessment of developmental levels, detailed developmental history, and screening for associated disorders such as Fragile X" (Magyar & Pandolfi, 2007, p. 1787).

Cross References

- ▶ Autism Diagnostic Interview-Revised
- ▶ Asperger's Disorder
- ▶ Attention-Deficit/Hyperactivity Disorder
- ▶ Autism Diagnostic Observation Schedule
- ▶ Autistic Disorder
- ▶ Pervasive Developmental Disorder Not Otherwise Specified
- ▶ TEACCH

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Childhood Epileptic Encephalopathy with Diffuse Slow Spike-and-waves

▶ Lennox–Gastaut Syndrome

Childhood or Adolescent Brain Injury

▶ Pediatric Traumatic Brain Injury

Children's Category Test

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Synonyms

CCT

Description

The Children's Category Test (CCT) is an abbreviated version of the original Halstead Category Test (HCT; Reitan & Wolfson, 1992). The CCT is an individually administered instrument designed to measure nonverbal learning and memory, concept formation, and problem-solving abilities. The CCT consists of two levels. Level 1 is given to children aged 5–8 and consists of five subtests and 80 items. Level 2 is given to children aged 9–16 and consists of six subtests and 83 items. The child's task is to identify the single conceptual rule underlying the items in

each subtest. The last subtest on both levels requires the child to remember and reapply the conceptual rules from previous subtests.

The CCT was normed on a stratified representative sample of 920 children in 12 age groups ranging from 5 years to 16 years, 11 months. Administration requires approximately 15–20 min. The raw score is the total number of errors (CCT Total), which is converted into an age-normed T-score (M = 50, SD = 10). The CCT is easy to administer and score.

Historical Background

The CCT was developed in an effort to provide an efficient and well-normed children's version of the HCT, which is well documented to be sensitive to cerebral impairment (Reiten & Wolfson, 1992), and the previous children's versions of the Category Test (Reiten & Wolfson, 1993). Concerns about lengthy administration times and expensive, bulky equipment led to the development of various short forms of these tests (e.g., Short Category Test Booklet Format), with the most comprehensive of these efforts resulting in the CCT (Boll, 1993). The CCT and California Verbal Learning Test-Children's Version (CVLT-C) were standardized and normed on the same population.

Psychometric Data

Median internal consistency reliability for the CCT Total score is 0.88 for Level 1 and 0.86 for Level 2, with average standard errors of measurement of 3.46 and 3.74, respectively (Boll, 1993). The separate age-level values and averaged coefficients indicate that the CCT possesses a high degree of internal consistency across ages. The manual reports a variety of studies demonstrating that the CCT consistently and significantly correlates with other measures of achievement or cognitive ability, such as the WISC-R and CVLT-C.

Clinical Uses

The CCT is a widely used test of the ability to solve problems by developing and modifying strategies of responding to various visual designs and patterns (Nesbit-Greene & Donders, 2002). The CCT does not require demonstration of acquired skills, ability, or knowledge, and eliminates potential confounding variables because it can be used with children with motor deficits or speech/language difficulties (Boll, 1993). The CCT can provide insights regarding a child's cognitive abilities and learning strategies in terms of difficulties in memory or shifting between conceptual ideas (MacNeil Horton, 1996). However, a poor score on the CCT does not necessarily indicate a neurologically based disorder; rather it indicates a disruption in mental processing (Boll, 1993).

Studies of the CCT's sensitivity to brain dysfunction indicate that the measure is not consistently sensitive to structural brain damage or neurodevelopment disorders in children (Bello, Allen, & Mayfield, 2008; Donders, 1996). Results from studies examining the sensitivity of the CCT to various forms of brain dysfunction suggest that the CCT assesses multiple dimensions of problem solving, rather than a general construct of abstraction (Nesbit-Greene & Donders, 2002), which has led some to caution against relying on the total error score as the sole index of brain dysfunction (Allen, Knatz, & Mayfield, 2006; Bello et al., 2008). The use of the CCT with brain-injured children may be hampered by the fact that the overall T-score is based on six different subtests that differ in their sensitivity to the severity of the injury (Nesbit-Greene & Donders, 2002). Another problem with using the CCT with brain-injured children is that it is untimed and children are provided with corrective feedback throughout the test administration, and these features may compensate for any deficits in processing speed or executive function that the child may be experiencing (Donders & Nesbit-Greene, 2004). Overall, studies investigating the use of the CCT with brain-injured children suggest that caution is needed in interpreting the results, and that it should be supplemented with psychometric data from additional neuropsychological tests (Donders & Nesbit-Greene, 2004; Moore, Donders, & Thompson, 2004).

Cross References

- ▶ Concept Learning
- ▶ Delis-Kaplan Executive Function System
- ▶ Halstead-Reitan Neuropsychological Test Battery
- ▶ Memory (Including Memory Impairment)
- ▶ Nonverbal Learning Disabilities
- ▶ Problem Solving

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Children's Memory Scale

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Synonyms

CMS

Description

The Children's Memory Scale (CMS), published in 1997, provides a comprehensive assessment of learning and memory in children and adolescents of ages 5 through 16 years. The CMS is individually administered and designed to be used as part of a standard psychological or neuropsychological evaluation. It assesses declarative learning and memory functions across three domains: Auditory/Verbal, Visual/Nonverbal, and Attention/Concentration (working memory). Each domain contains two core subtests and one supplemental subtest. The subtests comprising the Attention/Concentration domain provide measures of attention and working memory. Each subtest in the Auditory/Verbal and Visual/Nonverbal domains provide measures of both immediate and delayed (30 min) recall. Each Auditory/Verbal subtest also provides a measure of recognition recall.

After administration of the core subtests (Table 1), the examiner can derive eight index scores: Attention/Concentration, Verbal Immediate, Verbal Delayed, Delayed Recognition, Visual Immediate, Visual Delayed, Learning, and General Memory (Fig. 1). The learning Index is derived using subtest scores from the Auditory/Verbal (Word Pairs) and the Visual/Nonverbal (Dot Locations) domains. The General Memory Index is a measure of global memory functioning and is generated using both the immediate and delayed memory indexes from the Auditory/Verbal and Visual/Nonverbal domains.

The immediate portion of the core battery takes approximately 30–40 min to administer, with an additional 10–20 min required for administration of the delayed recognition sections. Two record forms are provided for students of 5–8 and 9–16 years. Scoring tables in the manual or computer software allow for conversion of raw scores to scaled scores (mean = 10; SD = 3) at the individual subtest level and standard scores (mean = 100; SD = 15) at the index level. Table 1 provides a brief description of the core subtests.

Historical Background

Development of the CMS began in 1985 to provide clinicians with a comprehensive, well-standardized, individually administered instrument that would assess the important processes involved in learning and memory within the pediatric population. Prior to this, traditional psychological evaluation of children with neurological and neurodevelopmental disorders included tests of intelligence, achievement, and behavior/emotional functioning with little if any attention paid to the child's ability to learn and remember new information. This was the case despite the fact that most referrals were in some way related to the student's inability to learn and remember school-related content. As a result, the CMS was developed with five goals in mind:

1. The development of an instrument that was consistent with current theoretical models of learning and memory.
2. The development of an instrument that was sensitive to developmental changes over time.
3. To evaluate the relationship between memory and intelligence and provide the clinician with a mechanism to meaningfully evaluate discrepancies between IQ and learning/memory performance.

Children's Memory Scale. Table 1 Description of CMS core index and subtest components

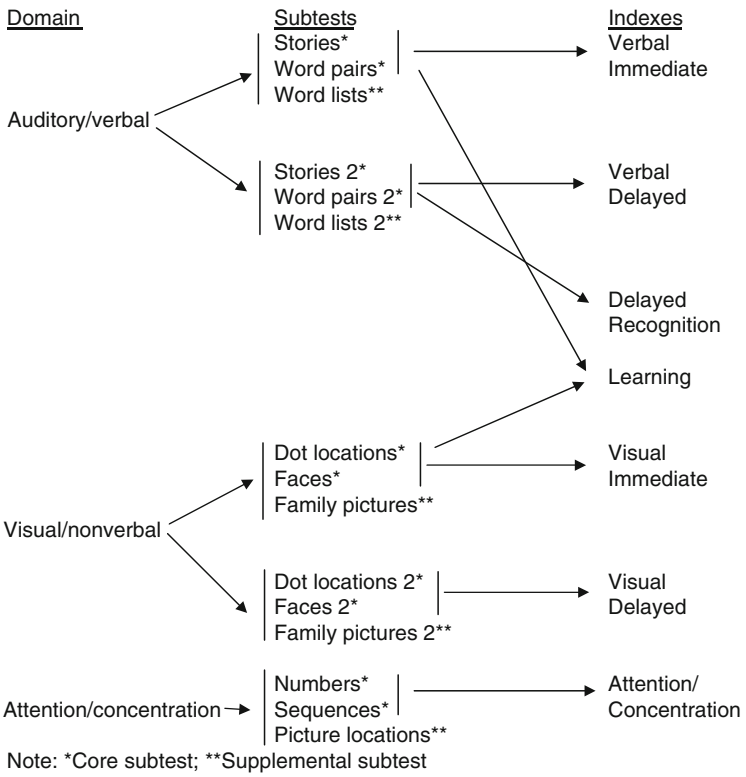
Core Index	Core Subtest	Subtest Description
Verbal Memory Indexes (Immediate, Delayed, and Recognition)	Stories	Two stories (age-dependent 5–8-, 9–12-, and 13–16-year olds) are read. Immediately after presentation of each story, the child is asked to repeat as much of the story as can be remembered. In the delayed portion, the child retells the stories and then answers questions about the stories (recognition recall).
	Word Pairs	A list of 10 or 14 (age-dependent 5–8-year olds; 9–16-year olds) related and unrelated word pairs are read; thereafter the stem is read and the child recalls the associate. Three learning trials are administered followed by a free recall. In the delayed portion, the child is asked to recall the word pairs spontaneously followed by a recognition section.
Visual/Non-Verbal Memory Indexes (Immediate and Delayed)	Dot Locations	The child is shown an array of dots (blue) located within a rectangle in a stimulus book. This page is removed and the client is asked to replicate the spatial location of the dots by placing chips on a 3 × 4 or 4 × 4 rectangular grid (depending on age 6 dots for 5–8-year olds; 8 for dots 9–16-year olds). Three learning trials are administered followed by presentation of a distractor array (red dots) after which an immediate recall trial is presented. In the delayed portion, the child is asked to reproduce the original blue dot array.
	Faces	The child is presented with a series of 12 (5–8-year olds) or 16 (9–16-year olds) pictured human faces one at a time. In the immediate and delayed recall sections, the child is asked to identify the stimulus faces from a different set of foils (36 or 48 colored photos).
Attention/Concentration Index	Numbers	A digit span forwards and backwards task (similar to the WISC-III subtest).
	Sequences	The client is asked to mentally sequence or manipulate information as quickly as possible. The 12 items include such tasks as reciting numbers, days of the week and months in forward and reverse order, and counting by 2s, 4s, and 6s. Scoring is based upon accuracy and speed.

4. The inclusion of a diversified selection of clinically and educationally relevant tasks that would allow clinicians to indentify and characterize learning and memory disorders in children and help them to design remedial and compensatory programs based upon the child's performance.
5. The development of an instrument that could be successfully administered within a standardized testing situation and also be child friendly.

As such, the CMS focused upon the assessment of declarative memory with no attempt made to formally evaluate procedural memory, which involves skill learning and classical conditioning. Further, the CMS was unable to provide measures of long-term memory beyond 30 min due to the time restriction of a traditional assessment and the logistical problems inherent in the reevaluation of the standardization sample over a longer time interval.

Psychometric Data

The CMS was standardized on a representative US sample of 1,000 children. The sample was stratified according to age (10 age groups ranging in age from 5 to 16 years; 100 per age group), sex (equal number of males and females in each age group), race/ethnicity (White, African American, Hispanic, and other), geographic region (northeast, north central, south, west), and parent education level (five categories ranging from <8th grade to university degree). Further, the CMS is the only memory assessment instrument to provide a linking sample co-normed with an individually administered intelligence scale (WISC-III and WPPSI-R). This sample was comprised of 300 children (ages 5–16; 50% male 50% female) and provided the examiner with an empirically grounded basis for predicting the level of memory performance from IQ and determining when a child's memory performance deviated significantly from IQ



Children's Memory Scale. Figure 1 Structure of the children's memory scale

expectancy. With the release of the WISC-IV in 2004, a smaller correlation study was conducted involving 126 children of ages 6–16 years (Drozdick, Holdnack, Rolfhus, & Weiss, 2005). The authors provide tables which allow for ability-memory discrepancy analysis using the predicted actual or simple difference methods and base rates.

The CMS manual provides both split-half and test-retest reliability coefficients for the index scores and subtests. For the indexes, average split-half reliability estimates ranged from .76 to .91. Average split-half reliability estimates for the subtests ranged from .71 to .91. Test-retest reliability (mean re-test interval 59.6 days) was assessed across three age bands using a sample of 125 students. Reliability estimates ranged from .29 to .89 for the indexes. Due to the nature of memory assessment and psychometric considerations (restriction of range), decision consistency test-retest coefficients were also calculated. These ranged from .61 to .93 for the indexes and from .71 to .93 for the core subtests. Results indicated a general practice effect of up to one standard deviation, similar to what was obtained on the WISC-III. Inter-rater reliability was also assessed on subtests requiring subjective scoring (e.g., stories) and was found to be high.

With respect to validity, the results of confirmatory factor analysis yielded a three-factor solution consisting of Auditory/Verbal memory, Visual/Nonverbal memory, and Attention/Concentration. The manual also provides concurrent validity studies with different measures of intelligence and achievement, which consistently show a moderate positive correlation. The CMS correlations with measures of executive functioning and language were low to moderate. Finally, with respect to other measures of memory, moderate to high correlation were obtained across the indexes of the CMS and Wechsler Memory Scale–III, and low to moderate correlations were obtained across the indexes of the CMS and the Wide Range Assessment of Learning and Memory. More recently, performance on both the CMS and WRAML-2 was reported as low to moderate in the latter test's Examiner's Manual (Sheslow & Adams, 2003).

Clinical Uses

As previously stated, the CMS was designed to be used as part of a standard psychological or neuropsychological

evaluation in order to provide a comprehensive assessment of learning and memory in children and adolescents of ages 5–16 years. The clinical sensitivity and usefulness of the CMS was demonstrated by the inclusion of case studies and clinical validity studies in the test manual. These included individual studies comparing the performance of children with temporal lobe epilepsy, traumatic brain injury, brain tumors, learning disabilities, ADHD, and specific language impairment to that of normal controls. Additional studies have been published examining learning and memory performance in children with dyslexia and/or ADHD (Kibby & Cohen, 2008), and specific language impairment (Riccio, Cash, & Cohen, 2007). The relationship between continuous performance testing and performance on the CMS has been investigated in a clinic sample (Riccio, Garland, & Cohen, 2007). Studies have also been reported on learning and memory performance of children with complex partial epilepsy of temporal origin, using an experimental version of the CMS (Cohen, 1992; Cohen, Holmes, Campbell, Smith, & Flanigan, 1990).

Cross References

- ▶ Memory
- ▶ Wechsler Memory Scale
- ▶ Wide Range Assessment of Memory and Learning

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Chi-square

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Definition

Chi-square is a nonparametric statistic that evaluates the likelihood that two observed distributions are different. The first step to calculating a chi-square is to construct a contingency table in which observations are plotted on the basis of the values of the relevant variables. For example, a contingency table might be constructed where one axis is gender and the other axis is diagnosis. The chi-square would describe whether female and male patients have approximately the same distribution of diagnoses.

Cross References

- ▶ Correlation Coefficients

References and Readings

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Chlordiazepoxide

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Generic Name

Chlordiazepoxide

Brand Name

Librium, Librax

Class

Anxiolytic

Proposed Mechanism(s) of Action

Chlordiazepoxide binds to the GABA-A ligand-gated chloride channel complex and, thereby, enhances the inhibitory effects of GABA.

Indication

Anxiety disorders, symptoms of anxiety, preoperative apprehension and anxiety, and withdrawal from acute alcoholism.

Side Effects

Serious

Respiratory depression, kidney dysfunction, and liver disorders (rare).

Common

Sedation, dizziness, depression, forgetfulness, confusion, and hyperexcitability.

References and Readings

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

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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

Chlorpromazine

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Generic Name

Chlorpromazine

Brand Name

Thorazine

Class

Conventional antipsychotic

Proposed Mechanism(s) of Action

Blocks dopamine 2 receptors; dopamine D2, histamine H1, and cholinergic M1 blockade in the vomiting center (area postrema)

Indication

Schizophrenia, nausea, vomiting, restlessness before surgery, manic episodes of bipolar disorder, tetanus

Off Label Use

Bipolar disorder

Side Effects

Serious

Neuroleptic malignant syndrome, jaundice, agranulocytosis, seizures

Common

Neuroleptic-induced deficit syndrome, akathisia, extrapyramidal symptoms, priapism, amenorrhea

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

Cholesterol

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Synonyms

Fats; Lipids

Definition

Cholesterol is a naturally occurring lipid found in the bloodstream and in cells throughout the body. It is an important component of cell membranes and some hormones. However, hypercholesterolemia, or high blood concentrations of cholesterol, is a major risk factor for heart disease and stroke.

Current Knowledge

Cholesterol does not typically exist on its own in the blood; it is transported to and from cells by lipoprotein molecules. Low-density lipoprotein (LDL) is the major carrier of cholesterol. In the presence of high circulating concentrations, LDL-cholesterol gradually builds up in and on the walls of the coronary, cerebrovascular, or peripheral arteries. Together with coagulation factors and platelets, cholesterol deposits on the vessel walls can form thick hard "plaques" which, over time, can occlude the blood vessels in a process called "atherosclerosis." Therefore, a high level of LDL-cholesterol (usually greater than 160 mg/dL) indicates an increased risk of heart disease and, to a lesser extent, stroke. For this reason, LDL-cholesterol is often called "bad cholesterol." About one-third of the circulating cholesterol is transported by high-density lipoprotein (HDL), which more commonly carries the cholesterol *away* from the arteries, and toward the liver where it is metabolized and eliminated. It has been suggested that HDL removes excess cholesterol from plaques, and consequently HDL-cholesterol is known as "good cholesterol" because a high HDL-cholesterol level seems to protect against coronary heart disease. Typically, the liver meets all of the body's requirements for cholesterol by manufacturing it, which means that dietary cholesterol is not needed for nutritional purposes. In foods, saturated fatty acids, and to a lesser extent, trans fats, are the main sources of elevated blood cholesterol levels. Measures to reduce elevated cholesterol levels include reductions in dietary cholesterol intake, cholesterol-lowering medications, increases in physical activity levels (which increase HDL-cholesterol), and cessation of smoking.

Cross References

▶ Atherosclerosis
▶ Cerebrovascular Disease
▶ Coronary Disease
▶ Ischemic Stroke
▶ Myocardial Infarction
▶ Peripheral Vascular Disease
▶ Stent
▶ Thrombosis

References and Readings

Fletcher, B., Berra, K., Ades, P., Braun, L. T., Burke, L. E., Durstine, J. L. et al. (2002). Managing abnormal blood lipids: A collaborative approach. *Circulation, 112*, 3184–3209.
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Cholinergic System

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Synonyms

Acetylcholinergic system

Definition

The cholinergic system is composed of organized nerve cells that use the neurotransmitter acetylcholine in the transduction of action potentials. These nerve cells are activated by or contain and release acetylcholine during the propagation of a nerve impulse. The cholinergic system has been associated with a number of cognitive functions, including memory, selective attention, and emotional processing.

Current Knowledge

Acetylcholine Synthesis and Metabolism

The synthesis of acetylcholine, the neurotransmitter used by nerve cells in the cholinergic system, requires choline, a natural amine found in the lipid bilayer of the cell membrane, and acetyl coenzyme A (acetyl-CoA), a thioester used in metabolic reactions and an acceptor and donor of acetyl groups. These molecules are catalyzed by choline acetyltransferase, an enzyme found only in acetylcholinergic cells, in order to produce acetylcholine.

In the metabolism of acetylcholine, acetylcholinesterase degrades the molecule to produce choline and acetic acid. The choline molecules generated through this reaction are transported, via a high-affinity choline uptake protein, back to the nerve terminal. They are then used to synthesize new acetylcholine molecules.

Cholinergic Receptor Types and Subtypes

Nicotinic: The nicotinic acetylcholine receptor type forms ligand-gated ion channels in the membranes of neurons receptive to acetylcholine. As an ionotropic receptor, the nicotinic receptor is directly linked to an ion channel. When acetylcholine binds to the nicotinic receptor, it generates the opening of the ion pore, causing a rapid influx of cations. There are four nicotinic receptor subtypes (N1, N2, N3, and N4), as well as α, β, δ, and γ subunits for each subtype.

Muscarinic: The muscarinic acetylcholine receptor type forms G protein-coupled channels in the membranes of neurons receptive to acetylcholine. When acetylcholine binds to one of these metabotropic channels, it generates a cascade of information transduction within the cell via intracellular proteins. There are five muscarinic receptor subtypes (M1, M2, M3, M4, and M5). All five receptor subtypes are present in the central nervous system; however, M1, M2, and M4, are more predominant.

Cholinergic Projections

The cholinergic system is formed by a broadly projecting circuitry, as well as local circuitry. Long-projection cholinergic neurons originating in the nuclei of the basal forebrain project widely throughout the brain. For example, the nucleus basalis of Meynert and the diagonal band of Broca, both structures within the basal forebrain, transmit acetylcholine to the cerebral cortex. In addition, cholinergic projections exist between the septal nucleus and the hippocampus. Local cholinergic circuitry within the striatum has important interactions with nigrostriatal dopamine neurons and striatal GABAergic neurons involved in extrapyramidal movement.

Cognitive Role of the Cholinergic System

The cholinergic system has been implicated in a number of cognitive abilities, including attention, memory, and emotional processing. Both human and animal studies indicate that cholinergic input originating in the basal forebrain mediates sustained attentional performance (see Sarter, Givens, & Bruno, 2001 for review). In addition, the cholinergic system also acts as a modulator of level of processing (e.g., primitive responses such as reflexes vs. limbic system responses vs. evaluative and discriminative executive functioning processes). Broadly, activation of the cholinergic system supports attentional processing of threat-related stimuli (Berntston, Sarter, & Cacioppo, 1998), while specific projections to the medial prefrontal cortex influence anxious responses to contextual stimuli (Hart, Sarter, & Bernston, 1999). By influencing arousal and attention, acetylcholine also impacts

working memory and the attentional processes required for error detection (see Sarter, Gehring, & Kozak, 2006 for review). Emotional processing is also intricately related to the cholinergic system; additionally, cholinergic inputs to the frontoparietal cortex may influence how attention is directed toward emotional information (Bentley, Vuilleumier, Thiel, Driver, & Dolan, 2003). As would be expected from a system influencing such a variety of cognitive functions, decreased cholinergic tone, associated with Alzheimer's disease, results in impaired cognitive performance broadly extending to memory, attention, and executive functioning (Terry & Buccafusco, 2003).

Pharmacological Agents Acting on the Cholinergic System

Pharmacological agents impact the cholinergic system in a number of different ways. At the receptor level, nicotinic acetylcholine agonists stimulate cholinergic activity largely in peripheral acetylcholine neurons. However, nicotine, a nicotinic acetylcholine agonist acts at nicotinic receptors in the central nervous system. Nicotinic acetylcholine antagonists acting on cells in the central nervous system are rarely used in clinical practice.

Pilocarpine is an example of a muscarinic acetylcholine agonist that acts on the parasympathetic nervous system. While pilocarpine is not used clinically to treat central nervous system disorders, it has a range of side effects, including excessive sweating and salivation, which are related to its action on muscarinic receptors in the parasympathetic nervous system. Muscarinic acetylcholine antagonists, such as scopolamine and atropine, block muscarinic receptors. Their anticholinergic effects on the parasympathetic nervous system result in side effects such as dry mouth, constipation, and tachycardia.

Acetylcholinesterase inhibitors, such as tacrine and donepezil, block the breakdown of acetylcholine in the synaptic cleft. The result is sustained levels of acetylcholine in the synapse that are capable of transmitting chemical information to other cells. Acetylcholinesterase inhibitors are commonly used in the treatment of Alzheimer's disease, as well as Lewy Body dementia.

Presently, there is a developing focus on the cholinergic system in the treatment of schizophrenia. Xanomeline, an M1/M4 muscarinic acetylcholine agonist that was initially assessed as a possible treatment for Alzheimer's disease, has shown efficacy in treating psychotic symptoms (Bodick et al., 1997). In addition, N-desmethylclozapine (norclozapine, NDMC), a metabolite of the antipsychotic clozapine, is a partial M1 agonist. This agent is currently being assessed as a treatment for schizophrenia (Lameh et al., 2007).

Future Directions

Future research focused on the interaction between the cholinergic system and other neurotransmitters (e.g., norepinephrine, dopamine, and serotonin), and neuromodulators (e.g., substance P) will be important in understanding the complexities that exist within the brain and body regarding the communication of chemical signals. Further development of biomarkers (e.g., behavioral, electrophysiological, and neuroimaging) associated with cholinergic decline will allow for improved identification of disorders associated with the cholinergic system. A continued focus on the role of the cholinergic system in Alzheimer's disease, as well as the development of pharmaceutical interventions targeting the cholinergic system in the treatment of this disease (e.g., alpha-7 subunit nicotinic agonists), remains critical, particularly with the continued rise in the elderly population. Finally, research on the role of the cholinergic system in a number of psychological disorders, including stress, affective and panic disorders, and schizophrenia, will continue to highlight the broad implications on mental health that this system has.

Cross References

- ▶ Alzheimer's Disease
- ▶ Cholinesterase Inhibitors
- ▶ Memory
- ▶ Memory Impairment
- ▶ Mild Cognitive Impairment

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Cholinesterase Inhibitors

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Synonyms

Acetylcholinesterase inhibitors; AchEIs; Anticholinesterase inhibitors; CHEIs

Definition

Cholinesterase inhibitors are a class of medications used in the treatment of Alzheimer's disease (AD) that targets the cholinergic neurotransmitter system. The treatment is based on the observation of a cholinergic deficiency in AD. Cholinesterase inhibitors block the activity of acetylcholinesterase, the primary enzyme that breaks down acetylcholine, and allows the neurotransmitter substance to remain in the synaptic cleft longer in order to stimulate postsynaptic receptors. These drugs do *not* prevent the further degeneration of cholinergic neurons. They are modestly effective in the short-term improvement of attention, concentration, memory, and some behavioural symptoms, but are not thought to alter the progression of the disease.

Cross References

► Alzheimer's Dementia
► Alzheimer's Disease
► Anticholinesterase Inhibitors

References and Readings

Orgogozo, J.-M. (2003). Treatment of Alzheimer's disease with cholinesterase inhibitors. An update on currently used drugs. In K. Iqbal & B. Winblad (Eds.), *Alzheimer's disease and related disorders: Research advances* (pp. 663–675). Bucharest, Romania: Ana Asian International Academy of Aging.

Chorea

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Synonyms

Dance-like

Definition

Chorea is characterized by brief, irregular muscle contractions that are not repetitive or rhythmic, but appear to flow from one muscle to the next. They may appear as dance-like movements of the limbs, trunk, or head. Typical movements include facial grimacing, shoulder adduction, and finger extension and contractions. They can be associated with snakelike writhing movements of the hands or feet known as athetosis.

Current Knowledge

Chorea is a feature of Huntington's disease, and may be present with rheumatic fever. It can be seen as a side effect of the medication levodopa or the dopamine agonists and may result from metabolic disorders, endocrine disorders, and vascular incidents.

Cross References

► Huntington's Disease

References and Readings

Marshall, F. J. (2004). Clinical features and treatment of Huntington's disease. In R. L. Watts, & W. C. Koller (Eds.), *Movement disorders* (2nd ed., pp. 589–603). New York: McGraw-Hill.

Christensen, Anne-Lise (1926–)

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Landmark Clinical, Scientific, and Professional Contributions

- In 1966, Anne-Lise Christensen reviewed a newly published English translation of Aleksandr Luria's (1966) *Higher Cortical Functions in Man* and instantly realized its value in the clinical bedside examination of cognitive function in neurological patients. Luria provided a comprehensive theory of the brain organization, from which cognitive tasks could be developed to measure various cortical functions. However, Luria's scholarly presentation required some adaptation before it could be practically applied by clinicians. Christensen began to translate Luria's methods into her native Danish as well as adapt them for use in the clinic. Responding to Luria's invitation, Christensen visited him in Moscow and presented her Danish translation. Luria labeled it a "vulgarization" of his method, but added he had always wanted someone to do this. He encouraged Christensen to do an English translation. Five years later, Christensen presented the first English draft to Luria on her second visit to Moscow. Luria made edits and provided a paper for Christensen to translate and include in the final draft. Christensen published *Luria's Neuropsychological Investigation (LNI)* in 1975, including a textbook, manual, and stimulus cards.
- The LNI introduced a qualitative approach to neuropsychological examination, which contrasted with the predominant quantitative batteries used in the USA. The LNI is one of the few theory-based assessment approaches in neuropsychology and has been lauded for its flexible administration, brevity, and qualitative focus (Kolb & Wishaw, 1990). It has also been criticized for its lack of norms and insensitivity to mild impairment (Lezak, 1983). Though Christensen declined to collaborate, Charles Golden and colleagues (Golden, Purisch, & Hammeke, 1979) in the USA attempted to standardize the administration of the LNI and establish a normative base for interpretation. This unfortunately violated the original qualitative focus of the LNI and generated considerable controversy and criticism. Nonetheless, Christensen's original qualitatively oriented LNI continues to be used in Europe, and somewhat less frequently in the USA

Education and Training

- 1954–1955 – Visiting research fellow at Radcliffe University
- 1956 – Master of Arts in Psychology, University of Copenhagen, Denmark
- 1957 – Doctor of Philosophy, University of Copenhagen, Denmark

Christensen began attending the University of Copenhagen in 1945, shortly after the liberation of Denmark from the Nazi occupation. She writes about the "overwhelming experience of freedom and peace . . . [when] the borders were opened, and we were full of hope and expectations for the future" (Christensen, 2002, p. 119). Her studies were interrupted for 6 years by marriage and motherhood, but she returned to the university in 1952, dividing her time between literature and psychology. Christensen was accepted at Radcliffe University in 1954, where she was exposed to the work of Talcott Parsons, Gordon Allport, Gardner Lindzey, and George Mandler. She cites, Jerome S. Bruner, however, as her greatest influence. In his work, Christensen was exposed to "New Look Psychology" which took the methods of the psychophysics laboratory and applied them to everyday perceptual experience.

Christensen returned to Denmark in 1955 and completed her doctoral studies at the University of Copenhagen in 1957. Bruner continued to influence Christensen's career for more than a decade later when he insisted she introduced herself to Luria at a 1969 conference in London. Impressed with her attempts at using his method in Denmark, Luria invited Christensen to visit him in Moscow, but a sudden heart attack prevented his participation in the conference. Nonetheless, an official invitation arrived in Denmark, and Christensen made her visit to the Bourdenko Neurosurgical University Institute to work with Luria in September 1970 (Christensen, 2002). This began the collaboration between the two, which resulted in the introduction of the LNI to the West.

Major Appointments

- University Hospital of Aarhus (Psychiatry and Neurosurgery Departments), Jutland, Denmark, 1959–1968
- University Hospital of Aarhus (Head of Clinical Psychological Department), Jutland, Denmark, 1969–1981

- University of Copenhagen (Research Neuropsychologist), Denmark, 1981–1985
- Center for Rehabilitation of Brain Injury (Founder and Director), University of Copenhagen, Denmark, 1985–1998
- University of Copenhagen (Professor of Neuropsychological Rehabilitation), Denmark, 1985–1998

Major Honors and Awards

- 1987 – Lady of the Dannebrog Order (bestowed by the Queen of Denmark)
- 1994 – Philosophia Doctor Honoris Causa, University of Lund, Sweden

Short Biography

A deep and lasting penchant for collaboration is evident both in Anne-Lise Christensen's personal and professional lives. At 19, she married Niels Egmont Christensen, a former classmate. Their careers progressed in tandem. While Anne-Lise was at Radcliffe, Niels was at Harvard. They were at the University of Aarhus together beginning in 1959, and Niels became the Chair of the Philosophy Department just a year before Anne-Lise became the Chair of Clinical Psychology. Over the course of her career, Christensen has had many distinguished collaborators, colleagues, and mentors. Besides Bruner and Luria, she cites the noted neuropsychologists Elkhonon Goldberg (whom she met in Moscow while training with Luria), Edith Kaplan, and Muriel Lezak, and neuroscientists Donald Stein and Stanley Finger (Christensen, 2002). Her collaborations have always been international in scope, stretching across Europe and the Americas. Her collaboration with her husband Niels came to a sad ending with his suicide in 1980 after battling bipolar disorder. Yet, even after retiring, her international collaborations continued, with Christensen recently serving on the Board of Consultants for the SARAH Network Hospitals in Brazil, a complex of facilities, she believes bridge "the chasm between humanistic and empirical scientific approaches" to rehabilitation (Christensen, 2002, p. 134).

Cross References

- ▶ Kaplan, Edith (1924–2009)
- ▶ Lezak, Muriel
- ▶ Luria, Alexander Romanovich (1902–1977)
- ▶ Luria Nebraska Neuropsychological Battery
- ▶ Qualitative Neuropsychological Assessment

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Chromosome

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Definition

Chromosomes are threadlike structures made of proteins and nucleic acids. They are found in the nucleus of eukaryotic cells and carry genetic information along their length in the form of genes. In the early 1900s, Walter Sutton and Theodor Boveri argued that the understanding of chromosomes was consistent with Mendelian genetics, and the result of their thinking is called the chromosome theory of heredity. A part of that theory is that Mendelian genes have specific locations (or loci) on chromosomes – a topic under heavy research since that time.

Chromosomes differ in overall length and the length of their parts, and they have distinctive banding patterns that allow them to be recognized. Humans have 23 pairs or 46 chromosomes. Of those 23 pairs, one pair is called the sex chromosomes, and the remaining 22 pairs are called autosomes.

Cross References

► Deoxyribonucleic Acid (DNA)
► Gene

Chronic Bronchitis

► Chronic Obstructive Pulmonary Disease

Chronic Familial Vascular Encephalopathy

► Cadasil

Chronic Fatigue Immune Deficiency Syndrome (CFIDS)

► Chronic Fatigue Syndrome

Chronic Fatigue Syndrome

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Synonyms

Chronic Fatigue Immune Deficiency Syndrome (CFIDS); Medically unexplained symptoms (MUS); Myalgic encephalitis (ME); Post-infectious fatigue syndrome (PFIS). Nomenclature used by patients or healthcare providers is generally based on their etiologic perspective

Short Description or Definition

Chronic fatigue syndrome is a complex illness defined by unexplained disabling fatigue and a combination of nonspecific accompanying symptoms that can have sudden or gradual onset. The diagnosis of the condition was initially defined by the Centers for Disease Control (CDC) study group (Holmes et al., 1988); they named the condition Chronic Fatigue Syndrome (CFS). In subsequent years, case definitions for CFS were created in Australia, Great Britain (Sharpe et al., 1991; Wessely, 1995), and Canada (Carruthers et al., 2003), and a pediatric case definition for ME/CFS was published in 2006 (Jason et al., 2006). Currently, the most common case definition used in the United States for the clinical diagnosis of CFS is a version of the original 1988 CDC case definition revised in 1994 by an International CFS Study Group (Fukuda, Straus, Hickie, Sharpe, Dobbins, & Komaroff, 1994).

The diagnosis of CFS cannot be made if the following are present and could account for the presence of persistent fatigue: (1) permanent medical exclusions include organ failure, chronic infections, rheumatic and chronic inflammatory diseases, major neurologic diseases requiring systemic treatment, major endocrine diseases, and primary sleep disorders; (2) temporary medical exclusions include treatable conditions that require evaluation over time to determine the extent to which they contribute to the fatiguing illness such as conditions discovered at onset or initial evaluation (e.g., effects of medications, sleep deprivation, untreated hypothyroidism, untreated or unstable diabetes mellitus, active infection), conditions that resolve (e.g., pregnancy), cardiac conditions, and morbid obesity specified as BMI $>$ 45; 3) permanent psychiatric exclusions include lifetime diagnoses of bipolar affective disorders, schizophrenia of any subtype, delusional disorders of any subtype, dementias of any subtype, organic brain disorders, and alcohol or substance abuse within 2 years before onset of the fatiguing illness, any past or current diagnosis of major depressive disorder with psychotic or melancholic features, anorexia nervosa, or bulimia.

If none of the exclusions apply, using the 1994 case definition, CFS is diagnosed based on the following criteria: (1) fatigue is of new or definite onset, not due to exertion, not relieved by rest, and must result in substantial reductions in previous levels of educational, occupational, social, or personal activities, (2) fatigue is of at least 6 months duration, and (3) concurrent with at least four of the following eight symptoms that could not have predated the onset of fatigue: impairment in short-term memory or concentration severe enough to result in substantial reductions in previous levels of educational, occupational, social, or personal activities, painful lymph nodes in front or back of the neck or under the arms, sore throat, muscle pain, pain in more than one joint without accompanying redness or swelling, headaches of a new type, unrefreshing sleep, postexertional malaise lasting

more than 24 h. These symptoms are nonspecific and variable in both nature and severity over time. They were selected on the basis of consensus clinical opinion and were not identified empirically.

Categorization

Only a small percentage of patients complaining of fatigue will be categorized as having CFS. Most patients either have prolonged fatigue, defined as self-reported, persistent fatigue of 1 month or longer, or chronic fatigue defined as self-reported persistent or relapsing fatigue of 6 or more consecutive months (Fukuda et al., 1994). Other conditions of unexplained etiology with similar symptom profiles are often comorbid with CFS and can include Fibromyalgia Syndrome (FMS), Temporo-Mandibular Syndrome (TMS), Irritable Bowel Syndrome (IBS), Multiple Chemical Sensitivity (MCS), Gulf War Syndrome (GWS), Major Depressive Disorder (MDD), and anxiety disorders.

Epidemiology

In the United States, CFS occurs in up to about 0.5% of the general population. It is most commonly found in middle-aged women and is most common in Latinos, followed by African Americans, and Whites. The illness affects women (predominantly between the ages of 40 and 59) more often than men (Reyes et al., 2003). In general, the expression of the syndrome is not gender-specific (Buchwald, Pearlman, Kith, & Schmaling, 1994). Chronic Fatigue Syndrome can also occur in children and adolescents. While gender distribution is similar to that of adults, prevalence rates are significantly lower.

Natural History, Prognostic Factors, Outcomes

Disorders with similar symptom profiles have been described for at least two centuries and have been known under a variety of names including neurasthenia, Akureyri disease, Epstein Barr Syndrome, and chronic mononucleosis. Although many hypotheses exist about the causes for CFS, the etiology of the condition is still unclear. Some believe that CFS is a latent form of depression and anxiety disorder, while others view the syndrome as a sleep disorder, attribute it to endocrine dysfunction or abnormalities within the central nervous system (CNS). Abnormalities identified in individuals with CFS include Hypothalamic–Pituitary–Adrenal Axis dysfunction, cortisol dysregulation, small white matter lesions in the frontal regions of the brain, orthostatic and cognitive dysfunction. Most abnormalities are found in CFS patients who do not suffer from comorbid psychiatric disorder, most commonly depression. Treatments, especially cognitive behavioral and graded exercise treatments, enhance the prognosis for improvement. If untreated, complete recovery from CFS is rare.

Neuropsychology and Psychology of Chronic Fatigue Syndrome

CFS patients typically complain of difficulties with concentration, memory, and thinking, yet neuropsychological testing does not generally confirm the reported cognitive dysfunction. Available data suggest that the main cognitive deficit in individuals with CFS is slowed information processing, which can affect memory as well as executive function. Depression is a very common comorbid condition (Tiersky, Johnson, Lange, Natelson, & DeLuca, 1997). Neuroimaging data increasingly provide evidence for decreased cerebral blood flow and functional activation of brain areas suggesting increased cognitive effort (Lange, Wang, Deluca, & Natelson, 1998; Lange et al., 2005).

Evaluation

A neuropsychological testing battery for individuals with CFS should include measures of overall current and premorbid cognitive function (i.e., Wechsler Test of Adult Reading, Wechsler Adult Intelligence Scale III/IV), simple and complex attention as well as information processing and working memory (i.e., Continuous Performance Test, Gordon, Trails, Paced Auditory Serial Addition Test), executive function (i.e., subtests of Delis–Kaplan Executive Function System including verbal fluency, towers test; Wisconsin Card Sort Test), memory (i.e., Wechsler Memory Scale III/IV, California Verbal Learning Test II, Rey Osterrieth Complex Figure Test), language function (i.e., Boston Naming Test), visual–perceptual function (i.e., Judgment of Line Orientation, Hooper), and motor function (i.e., grip strength, finger tapping, pegboard). It is also recommended to test the level of motivation and effort expanded during neuropsychological testing as well as emotional functioning to improve interpretability of test results.

When an individual is diagnosed with CFS, it may be desirable to evaluate the intensity and severity of symptoms associated with CFS using self-report

questionnaires. To assess fatigue intensity, several measures have been used including the Chalder Fatigue Scale and the Krupp Fatigue Scale; both have acceptable psychometric properties. The Chalder Fatigue Scale is a 14-item instrument with a 4-choice format and separates mental and physical fatigue (Chalder et al., 1993). The Krupp Fatigue Severity Scale includes nine items rated on 7-point scales and is sensitive to different aspects and gradations of fatigue severity. Most items in the Krupp scale are related to behavioral consequences of fatigue (Krupp, LaRocca, Muir-Nash, & Steinberg, 1989). The Checklist Individual Strength (CIS) is a 20-item inventory with four subscales commonly used to measure fatigue severity, concentration, reduced motivation, and physical activity. The CIS focuses on fatigue over the preceding 2 weeks (Vercoulen, Swanink, Fennis, Galama, Van der Meer, & Bleijenberg, 1994). Another commonly used instrument to measure fatigue severity is the Multidimensional Fatigue Inventory, also a 20-item questionnaire providing scores for severity of general fatigue, physical fatigue, mental fatigue, as well as reduced motivation and activity (Smets, Garssen, Bonke, & De Haes, 1995).

Another symptom that might warrant additional investigation is pain. Five of the eight CFS-defining symptoms reflect pain (headaches of a new type, pattern, or severity, muscle pain, and multi-joint pain without swelling or redness, sore throat, tender cervical/axillary lymph nodes). In many cases, pain may be the primary determinant of disability for some individuals with CFS. The McGill Pain Questionnaire (MPQ) is a well-validated questionnaire that can be used to further characterize pain or follow the course of pain in CFS (Melzack, 1975). The MPQ includes four components: (1) a human figure drawing on which patients are asked to mark the location of their pain; (2) a series of adjectives divided into groups from which patients identify their experience by circling word descriptors; (3) questions about prior pain experience, pain location, and information on the use of pain medication; and (4) a present pain intensity index.

Treatment

Effective treatment needs to be tailored to each individual diagnosed with CFS and often consists of a combination of behavioral, pharmacological, and physical interventions. Behavior modification or cognitive restructuring are two cognitive behavioral therapy (CBT) approaches used to treat CFS. Using this modality, reductions in the effect of fatigue on functional ability and quality of life have been shown in CFS (Kroenke, Taylor-Vaisey, Dietrich, & Oxman, 2000). Medication management of fatigue is in its infancy; presently, there is no FDA approved drug for fatigue. Over the counter medications offer a first line of therapeutic possibilities. Stronger medications, including stimulants and antidepressants, require a physician's prescription. In general, medications used to treat depression, anxiety, and pain are very often used for the pharmacologic treatment of CFS. It is important to recognize that fatigue is not solely due to disease processes, but can occur as an indirect consequence of decreased physical activity and conditioning. Graded aerobic exercise training is a safe and effective treatment for CFS and has been shown to improve quality of life. The primary goal of exercise is to avoid the spiral of deconditioning that is common in most fatiguing diseases.

Cross References

- ▶ Epstein Barr Syndrome
- ▶ Gulf War Syndrome
- ▶ Neurasthenia
- ▶ Unexplained Illness

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Chronic Multisymptom Illnesses

► Unexplained Illness

Chronic Obstructive Pulmonary Disease

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Synonyms

Chronic bronchitis; COPD; Emphysema

Short Description or Definition

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) Workshop defines COPD as "a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases." The GOLD committee was organized by the World Health Organization (WHO) and the US National Heart Lung and Blood Institute (NHLBI). Additional information about definitions, diagnosis, treatment, and research can be found at www.goldcopd.com.

Categorization

COPD has traditionally been defined by severity of airflow limitation. Indeed, the GOLD COPD classification system relies on measures of airflow limitation to stratify COPD severity (i.e., Forced Expiratory Volume in 1 Second (FEV_1) and Forced Expiratory Volume in 1 Second/Forced Vital Capacity(FEV_1/FVC ratio)). This staging system is currently the primary determinant of treatment guidelines. Heterogenity in the pathophysiology underlying COPD has been increasingly recognized over the past decade including chronic bronchitis, peripheral airways disease, and emphysema (Friedlander, Lynch, Dyar, & Bowler, 2007). At times, asthma has also been included in grouping of patients with COPD.

Epidemiology

COPD is the fourth leading cause of death in United States. Worldwide mortality from COPD is predicted to increase owing to the increased tobacco consumption in third world countries, and COPD mortality among women is increasing faster than mortality among men (Anthonisen & Manfred, 2004). The third National Health and Nutrition Examination Survey, conducted between 1988 and 1994, demonstrated that between 5% and 8% of the US population had COPD as defined by physiological parameters (NHANES III). The worldwide prevalence of COPD in 1990 was estimated by the WHO/World Global Burden of Disease Study to be 9.34 per 1,000 in men and 7.33 per 1,000 in women. In the US, COPD death rates are low among people under the age of 45, but increase with age.

Natural History, Prognostic Factors, Outcomes

Risk factors for COPD include both environmental and host-related variables. Cigarette smoke has been identified as the most important external risk factor, followed by pipe and cigar smoke, occupational dusts and chemicals, history of severe childhood respiratory infections, HIV infection, outdoor pollution, and IV drug use. Host-related factors include airways hyper-responsiveness, genetic factors, severe hereditary alpha1-antitrypsin deficiency, low birth weight, and maternal cigarette smoking during gestation (Anthonisen & Manfred, 2004).

Neuropsychology and Psychology of COPD

Empirical studies have documented neuropsychological (i.e., cognitive) deficits in patients with chronic airway obstruction and COPD. Two of the largest studies conducted in the 1980s were the Nocturnal Oxygen Therapy Trial (NOTT) and the Intermittent Positive Pressure Breathing Trial (Prigatano & Levin, 1988). The pattern and extent of cognitive dysfunction reported in COPD vary across patients and appear to be associated with disease severity. In COPD patients with moderate to severe hypoxemia, deficits have been identified in simple motor movement and overall strength, perceptual-motor integration, abstract reasoning, attention to auditory stimuli, learning and memory, and language skills. In patients with mild hypoxemia, impairments in higher cerebral functioning include abstract reasoning, auditory and visual attention, verbal and nonverbal learning and recall, and reasoning and motor skills (Kozora et al., 2008). There is some evidence that cognitive impairment has an independent impact on patients' daily functioning, medical regimen adherence, and quality of life, although results have been mixed (McSweeny & Labuhn, 1996).

Psychological changes and emotional distress have also been noted in COPD patients. To date, depression and anxiety are the most commonly observed psychological problems in COPD (Hynninen, Breitve, Wiborg, Pallesen, & Nordhus, 2005) with estimates of the prevalence of depression ranging from approximately 25% to 50%. Some of the discrepancies in prevalence estimates across studies may be related to differences in the method used to assess depression. In addition to emotional distress, multiple studies have demonstrated poor quality of life in patients with COPD, which has been associated with restrictions in daily activity, increased disease severity, and impaired health status (Hopkins & Bigler, 2001; McSweeny & Labuhn, 1996).

Evaluation

Neuropsychological evaluation for cognitive problems associated with COPD typically involves measurement of attention, learning and memory, reasoning and executive functioning skills, visuomotor speed, and visuoperceptual function. Psychological evaluation typically involves measures of depression, anxiety, and health-related quality of life.

Treatment

Research to date suggests that a variety of therapeuticapproaches utilized in COPD patients (including oxygen therapy, comprehensive rehabilitation programs, and surgical techniques) improve psychological and cognitive functioning. Long-term (greater than 6 months) use of oxygen therapy improves cognitive performance in COPD, probably due to direct effects of improved oxygen delivery to the central nervous system (Prigatano & Levin, 1988). Early studies reported improvement in visual memory, verbal memory, and motor speed among the COPD subjects following 6 months of continuous oxygen therapy. Large multisite studies have also demonstrated benefits of oxygen for cognitive function in COPD. For example, in the Nocturnal Oxygen Therapy Trial, COPD patients receiving continuous oxygen therapy for 12 months experienced greater improvements in cognitive performance than did patients receiving only nocturnal oxygen therapy.

There is also evidence suggesting that comprehensive multidisciplinary rehabilitation programs can improve cognitive functioning and psychological status in emphysema patients. Comprehensive rehabilitation programs for treatment of COPD are well established and typically include assessment, education, instruction on respiration, psychosocial support, and exercise training with the goal of restoring patients to the highest level of independent function (Make, 2004). As noted in a recent review article (Kozora et al., 2008), Emery and colleagues first reported in 1991 improved complex attention in COPD patients following a 30-day exercise rehabilitation program that included instructional/educational components, psychosocial counseling, and stress reduction. In a

subsequent study published in 1998, they reported improvement in verbal fluency and reduction in symptoms of anxiety and depression in a group participating in exercise, stress reduction, and education programs when compared to a control group participating in stress reduction and education treatment only. This finding highlighted the utility of the exercise component toward improved cognitive and psychological functions. In 2002, Kozora and colleagues also reported improvement in visual attention and semantic fluency, among COPD patients following a 3-week comprehensive rehabilitation program when compared to untreated COPD and healthy control subjects similar in age, education, and gender. This program included exercise, educational, instructional, and psychosocial components. In addition to cognitive changes, significant improvement in depressive symptoms was reported. Together, these studies indicate improved cognitive performance and psychological status following comprehensive rehabilitation with an exercise component. Many studies have also reported increased quality of life following comprehensive rehabilitation (Make, 2004).

Finally, studies have suggested some improvement in cognition for moderate to severe COPD patients following lung volume reduction surgery (LVRS). In an ancillary study of the National Emphysema Treatment Trial (NETT), Kozora and colleagues (Kozora et al., 2008) examined neuropsychological and psychological functioning of patients receiving LVRS when compared to patients receiving only medical therapy (MT). The LVRS group showed significant improvement compared to the MT group at 6 months on a measure of psychomotor speed, delayed recall for verbal information, and trends toward improved sequential thinking, and psychomotor speed and naming to confrontation. LVRS patients also experienced significant reduction in depression at 6 months, as reflected in total Beck Depression Inventory score. There was no direct evidence that improved cognition in LVRS was related to improved physical capacity (workload and 6 min walk) or pulmonary function.

Cross References

► Anoxia

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Chronic Organic Brain Syndrome

► Organic Brain Syndrome

Chronic Progressive Multiple Sclerosis

► Primary Progressive Multiple Sclerosis

Chronic Widespread Pain Disorder

► Fibromyalgia

Cingulate Cortex

► Cingulate Gyrus

Cingulate Gyrus

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Synonyms

Cingulate cortex; Subcallosal; Subgenual

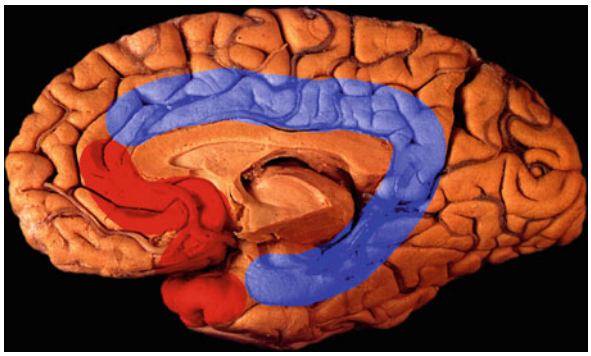
Structure

The cingulate gyrus, first named by Burdach in 1822, was combined with the anterior olfactory region and hippocampus by Broca to form the ring of olfactory processing he termed the *grand lobe limbique*. Anatomic studies revealed extensive connections between the anterior thalamus, known to be associated with the hippocampus and hypothalamus, and the cingulate. In 1937, Papez combined these anatomic results with the clinical reports of emotional disturbances following lesions to the cingulate and other limbic structures to propose a mechanism of emotion based on a limbic circuit. For Papez, the integration of internal feelings and emotional responsiveness with the functions of the lateral cerebral cortex occurred in the cingulate. The limbic circuit of Papez however did not find anatomical evidence to support the closing connection of the cingulate to the hippocampus until 1975, when Shipley and Sørensen documented that the presubiculum, which receives a dense cingulate outflow, projects heavily to layer III of the entorhinal cortex – the origin of the perforant pathway into hippocampal pyramidal cells.

Understanding the function of the cingulate in the integration of internal feelings and emotional responsiveness with movement and thought begins with an appreciation of its cytoarchitecture. Brodmann's original cytoarchitectonic separation of anterior and posterior cingulate cortex into areas 24 and 23 based upon the presence of agranular cortex in area 24 contrasted with the granular cortex of area 23 has been refined by more accurate studies of the progressive cytoarchitectonic elaboration in the ventral to dorsal direction. Two main centers of isocortical development can be traced phylogenetically and through cytoarchitectonic progression. These two developmental trends, termed *paralimbic belts*, are transitional cortical zones from less-differentiated allocortex to more-differentiated isocortex with two functional centers: the more rostral olfactory piriform paleocortex unites the orbitofrontal, insular, and temporopolar regions, while the more caudal archicortex of the hippocampus provides the nidus for developmental spread through parahippocampal and entorhinal regions into the posterior cingulate. Both paralimbic belts reflect a different emphasis within the cingulate (Fig. 1). The orbitofrontal-centered belt processes the internal *affective* state of the organism. The more recent hippocampal-centered belt is the externally directed *evaluative* arm of the limbic system.

Both work in concert, enabling the selection of environmental stimuli based on the internal relevance those stimuli have for the organism. Although both areas 24 and 32 are part of the hippocampal belt, the more rostral connections of area 24 contrast with the more caudal sensory connections of area 23, distinguishing the anterior executive from the posterior evaluative cingulate. Appreciating that the major reciprocal connections between the orbitofrontal trends are with the anterior cingulate, while the hippocampal trends are with the posterior cingulate, will assist the understanding of cingulate function.

Three anterior effector regions and a posterior processing region also emerge through a review of the efferent and afferent connections of the cingulate. The three anterior regions include a *visceral effector region* inferior to the genu of the corpus callosum encompassing area 25, the anterior subcallosal portions of 24a–b, and 32; a *cognitive effector region*, which includes most of the supra-callosal area 24, and areas 24a'–b' and 32'; and a *skeletomotor effector region* within the depths of the cingulate sulcus that includes areas 24c'/23c on the ventral bank, with 24c'g and 6c on the dorsal bank. These three cingulate effector regions integrate ascending input concerning the internal milieu of the organism with visceral motor systems, cognitive-attentional networks, and skeletomotor

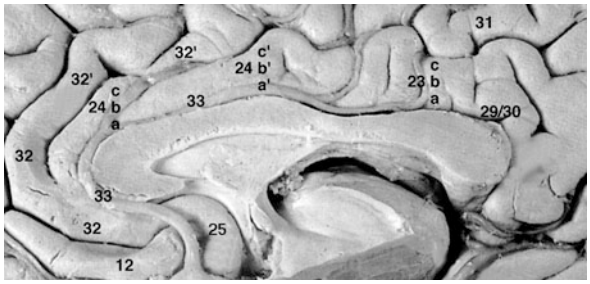


Cingulate Gyrus. Figure 1 The Orbiotfrontal (*red*) and hippocampal (*blue*) paralimbic trends

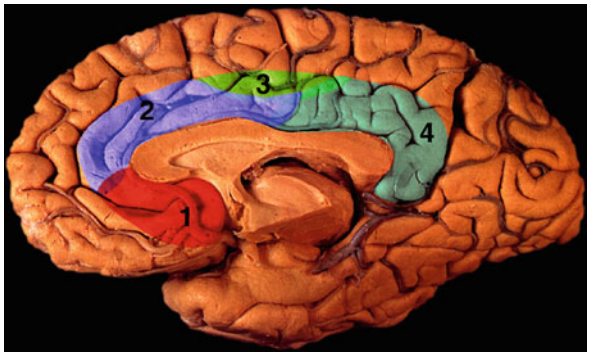
centers to produce the affective motivation necessary for the organism's engagement in the environment. The posterior *sensory processing region* (areas 23a–b and 29/30) assists in the memory and processing of environmental stimuli targeted as relevant to the organism based on their motivational valence. The following sections will explore the connections of these four cingulate regions (Figs. 2 and 3) and their function as observed from electrical stimulation, imaging, and clinical data.

Function

Visceral effector region. Major reciprocal connections with the visceral effector region are with the basal and accessory basal amygdala, medial orbitofrontal cortex areas 11, 12, and 13, anterior superior temporal pole area 38, and the anterior ventral claustrum. Major nonreciprocal projections from the visceral effector region target the parasympathetic nucleus of the solitary tract, the sympathetic thoracic intermediolateral column, the dorsal motor nucleus of the vagus, and the nucleus accumbens/olfactory tubercle of the ventral striatum.



Cingulate Gyrus. Figure 2 Cytoarchitectonic divisions of the cingulate and adjacent areas



Cingulate Gyrus. Figure 3 The four functional divisions of the cingulate. The visceral effector region (1), the cognitive effector region (2), the skeletomotor effector region (3), and the posterior cingulate (4)

Brain areas that have reciprocal connections with the visceral effector region, except the claustrum that supplies auditory input, also influence visceral function when stimulated. This results from their amygdalar connections that convey the visceral state of the organism to paralimbic areas. The orbitofrontal cortex mediates empathic, civil, and socially appropriate behavior. Rostral auditory association cortex in the superior temporal area also provides auditory information to the visceral effector region. No visual information has direct access to the subcallosal area.

The dorsolateral prefrontal lobe, functioning as an "executive processor," provides nonreciprocal input from areas 9 and 46 to the subcallosal anterior cingulate region. Executive functions permit an organized behavioral response to solve a complex problem. This includes the activation of remote memories, self-direction, and independence from environmental contingencies, shifting and maintaining behavioral sets appropriately, generating motor programs, and using verbal skills to guide behavior. Dorsolateral prefrontal efferents into the subcallosal cingulate provide feedback inhibition on the basic drives of hunger, aggression, and reproductive urges. Table 1 for a summary of these anatomic connections.

Subcallosal cingulate processing modulates visceral output from brain-stem sympathetic and parasympathetic centers. Access to the basal ganglia, via the ventral striatum, allows processing of the internal milieu of the organism to influence the skeletal motor system as well. This visceral motor network encompasses the bulk of the orbitofrontal-centered paralimbic belt dedicated to assessing the emotional valence of objects based upon internal motivating drives. Patients with lesions in this area are often

Cingulate Gyrus. Table 1 Major reciprocal connections and nonreciprocal targets for the visceral effector region of the cingulate cortex

Reciprocal connections
Basal and accessory basal amygdala
Medial orbitofrontal areas 11, 12, and 13
Superior temporal pole area 38
Anterior ventral claustrum
Nonreciprocal targets
Parasympathetic nucleus of solitary tract
Sympathetic intermediolateral column
Dorsal motor nucleus of the vagus
Nucleus accumbens and olfactory tubercle

disinhibited with irritability, lability, tactlessness, and fatuous euphoria. Patients act upon visceral drives without regard to social decorum.

Cognitive effector region. Major reciprocal connections with the cognitive effector region are with the basal amygdala, prefrontal areas 8, 9, 10, and 46, caudal orbitofrontal area 12, inferior temporal pole area 38, rostral insula, anterior parahippocampal areas 35 and 36, and the anterior medial claustrum. Areas reciprocally connected to the cognitive effector region share a general similarity with the subcallosal anterior region underscoring their common membership in the orbitofrontal paralimbic belt. The cognitive effector region is more developed in its cytoarchitecture, than the subcallosal cingulate, and thus has stronger connections with more phylogenetically recent neocortex of dorsolateral prefrontal areas 8, 9, 10, and 46 devoted to executive function. The amygdala provides internal affective input to the supracallosal anterior cingulate. The distribution of amygdala efferents delineates the dorsal boundary of the cingulate as a functional system. Auditory input arises from the anterior medial claustrum as well as a minor link with the auditory association area of the superior temporal gyrus. The rostral insula and anterior parahippocampal areas provide additional reciprocal connections with the cognitive effector region not associated with the subcallosal region. Rostral insular cortex is a transitional paralimbic region that integrates visceral alimentary input with olfactory and gustatory afferents. Connections with the anterior parahippocampal areas 35 and 36 allow the supracallosal cingulate to influence multimodal sensory afferents entering the hippocampus.

Major nonreciprocal projections of the supracallosal anterior cingulate include the auditory association cortex of anterior superior temporal area 22, allowing the cognitive effector region to influence language and the access of semantic stores. The posterior parietal area 7a and the dorsomedial head of the caudate are also targets. Parietal area 7a is the sensory component in the extrapersonal attentional network linked with the dorsolateral prefrontal "executive system." The head of the caudate is also a target of this executive prefrontal cortex. Cingulate input to the caudate assists in the initiation of vocalization behavior as well as executive function. Emotional vocalizations occurring during stimulation in monkeys requires intact cingulate efferents to the periaqueductal gray that produce similar behaviors when stimulated. The most caudal amygdalar projections to 24c, extending into anterior 24c', innervate a face representation region that may have direct connections with the facial nucleus in the pons. Efferents to the dorsomedial pons provide cingulate influence on the reticular activating system and its control over arousal. Table 2 for a summary of these anatomic connections.

Skeletomotor effector region. Major reciprocal connections with the skeletomotor effector region are with the primary and supplementary/premotor motor cortex areas 4 and 6, prefrontal areas 8, 9, and 46, parietal areas 1, 2, 3a, 5, and 7b, and caudal insula. Major nonreciprocal projections from the skeletomotor area target the lateral putamen, spinal cord, ventromedial parvocellular division of the red nucleus, and the ventrolateral pontine gray matter.

Primary motor cortex has very limited input. The medial supplementary, lateral premotor, and cingulate skeletal motor regions are the only forebrain inputs to the primary motor cortex. The skeletomotor region in the banks of the cingulate sulcus conveys limbic influence to the medial supplementary, lateral premotor, and primary motor cortex. Frontal eye fields in area 8 also share reciprocal connections with the skeletal motor effector region. Areas 9 and 46 of the dorsolateral prefrontal cortex contribute executive input to the limbic motor system. Thus, executive and limbic systems gain access to primary motor area 4 indirectly. The cingulate skeletal motor region receives the greatest outflow from executive prefrontal cortex than all other motor cortices underscoring its influence over goal-directed behavior. Sensory-motor parietal areas 1, 2, 3a, and 5 also have reciprocal connections with the skeletal motor center within the banks of the cingulate sulcus. The rostral parietal area 7b has a strong relationship with the premotor cortex, while the granular cortex of the caudal

Cingulate Gyrus. Table 2 Major reciprocal connections and nonreciprocal targets for the cognitive effector region of the cingulate cortex

Reciprocal connections
Basal amygdale
Prefrontal areas 8, 9, 10, and 46
Caudal orbitofrontal cortex area 12
Inferior temporal pole area 38
Rostral insula
Anterior parahippocampal areas 35 and 36
Anterior medial claustrum
Nonreciprocal targets
Anterior superior temporal area 22
Parietal area 7a
Dorsomedial head and body of caudate
Periaqueductal gray
Dorsomedial pontine gray matter

insula is a somatosensory limbic region. More corticospinal neurons are found in the cingulate than are found in the supplementary motor cortex, and the cingulate has about 40% of the amount found in primary motor cortex. Table 3 for a summary of these anatomic connections.

Sensory processing region. Major reciprocal connections with the sensory processing region are with caudal parietal area 7a, frontal eye fields area 8, posterior perirhinal area 35, presubiculum, posterior parahippocampal area 36, prefrontal area 46, and the ventral caudal claustrum. Major nonreciprocal projections from the sensory processing region target the dorsal caudate, posterior superior temporal gyrus area 22, and orbitofrontal area 11. These regions are shown in Table 4.

Cingulate Gyrus. Table 3 Major reciprocal connections and nonreciprocal targets for the skeletomotor effector region of the cingulate cortex

Reciprocal connections
Primary motor area 4
Supplementary motor area 6
Prefrontal areas 8, 9, and 46
Parietal areas 1, 2, 3a, 5, and 7b
Caudal insula
Nonreciprocal targets
Lateral putamen
Spinal cord
Red nucleus
Ventrolateral pontine gray matter

Cingulate Gyrus. Table 4 Major reciprocal connections and nonreciprocal targets for the sensory processing region of the cingulate cortex

Reciprocal connections
Caudal parietal area 7
Frontal eye fields area 8
Prefrontal area 46
Posterior parahippocampal areas 35 and 36
Presubiculum
Ventral caudal claustrum
Nonreciprocal targets
Dorsal caudate
Posterior superior temporal area 22
Orbitofrontal area 11

The posterior granular sensory cortices are distinguished from the anterior agranular executive cortices. The posterior cingulate, with its prominent granular layer IV, is dedicated to visuospatial and memory processing. Major reciprocal connections are with the dorsal visual system of the inferior parietal lobe dedicated to spatial processing and with the frontal eye fields in area 8. Reciprocal connections with lateral prefrontal area 46 allow an interaction between executive and sensory/mnemonic processing, which may mediate perceptual working memory tasks. Posterior parahippocampal and perirhinal areas 36 and 35, as well as the presubiculum, are reciprocally connected to the sensory processing region of the posterior cingulate. These connections modulate the multimodal efferents entering the entorhinal layer III cells that form the perforant pathway into the hippocampus. Feedback from these areas to the cingulate provides highly processed sensory information, and the ventral visual system involved in feature analysis can influence the posterior cingulate through these connections. Although in cats dorsal caudal claustrum is related to visual processing, while the ventral caudal claustrum receives auditory input, it is possible that in primates visual information may reach the posterior cingulate via the ventral caudal claustrum.

The nonreciprocal targets of the posterior sensory processing region include the dorsal caudate which also receives input from area 7a of the caudal inferior parietal lobe. This shared connection between the dorsal head of the caudate and the dorsal visual system of area 7a supports the role of the posterior cingulate in visual attention. Output to posterior superior temporal area 22 will influence auditory association cortex. Limited efferents to the rostral portion of area 11 provide the only overlap with the orbitofrontal-centered belt.

Electrical stimulation of the cingulate. Electrical stimulation studies of the cingulate in humans and animals are difficult to interpret because differing techniques have been used in these investigations. With varying intensity, time course, and location of stimulation, it is not surprising that a spectrum of results is noted. Despite technical variations, stimulation of the anterior cingulate in humans regularly produces visceral motor and affective changes, speech alterations, and automatic motor behaviors (Meyer, McElhaney, Martin, & McGraw, 1973). In contrast to inhibitory responses elicited by the stimulation of primary motor cortex, which cannot be controlled, respiratory arrest from cingulate stimulation can be overcome volitionally (Penfield & Jasper, 1954). Automatic behaviors noted include unilateral and bilateral movements and repetitive "tic like" movements of the hands,

lips, or tongue. These movements can also be consciously suppressed; implicating the cingulate as an "unconscious" effector supports its role in the pathophysiology of obsessive–compulsive disorder (behaviors that respond well to cingulotomy). Fear, pleasure, agitation, euphoria, and a sense of well being – affective phenomena also common after limbic stimulation – have been reported (Meyer, McElhaney, Martin, & McGraw, 1973). Involuntary vocalizations and speech arrests are less common in humans than in animals with stimulation of areas 32, 24, and the rostral part of 25 (Vogt & Barbas, 1988).

Functional activation of the cingulate. In functional activation studies, the cognitive effector region of the anterior cingulate is activated when sustained attention to *novel* tasks is required. Tasks spanning motor, language, memory, and visuospatial paradigms all produce supracallosal anterior cingulate activation. When memory encoding is combined with a motor task demanding sustained divided attention (Fletcher et al., 1995), only the anterior cingulate is activated due to the sustained vigilance demanded by dividing effort between the two tasks. When motivation to master a task is no longer required, and accurate performance of a task becomes routine, the anterior cingulate returns to a baseline activity level (Raichle et al., 1994). The acquisition of novel cognitive strategies requires the "dynamic vigilance" of the supracallosal cingulate, but with practice the motivation required to entrain new cognitive networks to a novel task is no longer necessary. A distinction between motivation and attention is important. A task is still attended to and completed correctly after the motivating influence of the supracallosal cingulate has initiated the acquisition of an efficient cognitive routine. Through the activation of the anterior supracallosal cingulate limbic motivation directs the selection of the best cognitive strategy among many competing contingencies. Thus, activation studies using varied tasks consistently activate the cognitive effector region in normals motivated to succeed in whatever task is given them. The contribution to an extrapersonal attentional network – involving direct links between the anterior cingulate, dorsolateral executive frontal area, and the inferior parietal lobule – provided by the cognitive effector region is the *motivation* to engage in a cognitive challenge.

Functional imaging has also confirmed the role of the skeletomotor effector region in the preparation of motor output and motor learning. When a motor task is only imagined, the cingulate cortex inferior and anterior to the supplementary motor area (dorsal bank of the cingulate sulcus) shows significant activation. During the acquisition of procedural learning in a rotory pursuit task, the cingulate skeletomotor region is also activated (Grafton, Woods, & Tyszka, 1994).

Illness

Structural and functional abnormalities. Seizures originating in the anterior cingulate can alter visceral activity, produce involuntary skeletomotor output, result in disturbed attention, and cause interictal behavior abnormalities. The severity and specific abnormality will depend upon the location of the seizure focus and ensuing damage that affects interictal brain function. A diverse assortment of atonic, absence, speech arrest, autonomic, and complex partial seizures with secondary generalization have been described. Inaccessibility of the medial hemisphere to surface electrode recording is the greatest obstacle to the elucidation of cingulate seizures.

In a study involving 36 cases (Mazars, 1970), depth electrodes revealed near-instantaneous bilateral spread to the frontal poles when the focus was in the anterior cingulate; posterior foci spread to the contralateral cingulate within seconds, followed by involvement of the convexities with generalized tonic–clonic seizures. Emotional stress often precipitated the seizures. Psychoses and episodic rage were common interictal behavioral abnormalities that responded to the removal of the anterior cingulate and occasionally the frontal pole as well. Consciousness may be altered and automatisms can be voluntarily inhibited or integrated with ongoing movements (Geier et al., 1977).

An 11-year-old girl who initially had atonic seizures at age 30 months was reported to develop complex partial seizures with blinking, lip smacking, automatisms, and humming (Levin & Duchowny, 1991). An obsessive–compulsive disorder developed over a 5-year period, and by age 8, she became preoccupied with Satan and her personal hygiene. Seizure focus, recorded from depth electrodes, was in the right anterior cingulate. The patient's behavioral abnormalities responded well to a 4-cm ablation of the right anterior cingulate.

Another case of a right anterior cingulate focus with accompanying behavioral abnormalities has been described (Devinsky, Morrell, & Vogt, 1995). One year after mild head trauma, a 42-year-old male developed, over a 15-year period, sociopathic behavior and complex partial seizures unresponsive to medical treatment. Seizures were usually nocturnal and frequent (10–20 per night), with stereotypic motor output: facial contortions, tongue thrusting, a strangulated yell, flexion of the neck and trunk, *bilateral* extremity extension, and thrashing

with *preserved* consciousness. Occasionally, generalization to tonic–clonic seizures developed with loss of consciousness.

Irritability, disinhibition, and sexual deviancy were behavioral complications in a police officer who was dismissed from the force because of brutality and the use of confiscated drugs. Surface and sphenoidal electroencephalogram showed rhythmic bifrontal theta. Magnetic resonance imaging (MRI) and [^{18}F]-fluorodeoxyglucose positron emission tomography (FDG-PET) were essentially normal, but depth electrodes revealed a right cingulate focus which spreads to the ipsilateral orbitofrontal area and contralateral anterior cingulate in 300 ms. Resection of the right cingulate and anterior corpus callosum relieved 90% of the spells with only brief axial flexion being the residual seizures. The behavioral abnormalities were reported to improve with the patient married and employed as a fast-food restaurant manager.

Both stimulation and seizure activity can discharge the functional centers of the cingulate to produce a visceral effect, a cognitive or behavioral change, and a speech or motor output. Appreciating the functional centers within the cingulate assists the interpretation of the signs and symptoms exhibited when it discharges. The interictal behavioral abnormalities of anterior cingulate epilepsy reflects the dysfunction of limbic networks which, if affecting infracallosal and orbitofrontal cortex, will result in visceral motor disturbances and disinhibition with socially inappropriate behavior. Obsessive–compulsive features may occur with dysfunction of the cognitive component of the supracallosal cingulate. This abnormal "dynamic vigilance" exerted by the cognitive effector region in obsessive–compulsive disorders can occur from a well-circumscribed seizure focus in this region (Levin & Duchowny, 1991) and is relieved by surgical ablation of this region or its outflow (Tow & Whitty, 1953).

Focal lesions and syndromes. Well-circumscribed lesions in humans are rarely confined to one region of the cingulate. With an anterior lesion, both the cognitive, skeletomotor and visceral effector regions are often affected. Bilateral lesions result in an akinetic mute state (► *Akinetic Mutism*). Patients are profoundly apathetic. Rarely moving, and incontinent, they eat and drink only when fed, and if speech occurs it is limited to monosyllabic responses to questioning. Patients appear awake with eyes tracking objects. Displaying no emotions, even when in pain, patients show complete indifference to their circumstance. Transient akinetic mutism with similar features occurs with unilateral lesions. The akinetic mute state can also result from bilateral paramedian diencephalic and midbrain lesions, possibly affecting the ascending reticular core. Failure of response inhibition on go-no-go tests is the major neuropsychological deficit in the patient with an anterior medial frontal damage. The loss of spontaneous motor activity results when the lesion involves the supplementary motor area and the skeletomotor effector region. When these two motor regions are spared, motor activity will be normal but the patient will demonstrate profound indifference, docility, and the loss of motivation to engage in a task. They can be led by the examiner to engage in a task but will fail to self-generate sustained directed attention. They lack cognitive motivation.

The role of the anterior cingulate as a cognitive effector is appreciated within the realm of language. Language, a cognitive function, is distinguished from the motor function of speech. Transcortical motor aphasia (TCMA) is the usual result of left anterior medial or anterior dorsolateral prefrontal lesions. The classic syndrome of TCMA is initial mutism that resolves in days to weeks, yielding a syndrome featuring delayed initiation of brief utterances without impaired articulation, excellent repetition, inappropriate word selection, agrammatism, and poor comprehension of complex syntax. Activation of dorsolateral prefrontal cortices enabling language and speech arises from two sources: the anterior cingulate and the supplementary motor area (with the cingulate skeletomotor effector region). When the executive prefrontal cortex (areas 9, 10, and 46) is disrupted, cognitive language deficits are prominent (TCMA, type I); when motor neurons in area 4, devoted to the speech apparatus, are disconnected from their activation, speech hesitancy and impoverished output ensues (TCMA, type II). These two functional realms are separable and can be disconnected anywhere along two pathways. Direct damage to the supplementary motor area or its efferent pathway to the motor cortex traveling in the anterior superior paraventricular white matter will produce TCMA type II. Direct damage to the anterior cingulate, its outflow to areas 9, 10, and 46, or to the caudate – via the subcallosal fasciculus, just inferior to the frontal horn of the lateral ventricle – will disrupt frontal-subcortical circuits involved in motivation and executive cognitive functions. The initial muteness has been described by a patient after recovering from an anterior cingulate/supplementary motor infarction as a loss of the "will" to reply to her examiners, because she had "nothing to say," her "mind was empty," and "nothing mattered" (► *Akinetic Mutism*).

The cingulum bundle has also been the site of surgical lesions (cingulumotomy, or cingulotomy when cingulate cortex is also removed) to treat psychiatric and pain disorders. The cingulum contains the efferents and afferents

of the cingulate to the hippocampus, basal forebrain, amygdala, and all cortical areas, as well as fibers of passage between hippocampus and prefrontal cortex, and from the median raphé to the dorsal hippocampus. Surgical ablation of the anterior portion (sparing fibers relevant to memory function) is most successful when treating aggression, extreme anxiety, obsessive–compulsive behaviors, and severe pain. Psychotic symptoms show only a temporary response. The three anterior cingulate regions, by virtue of the distinct functional systems they access, are the conduits through which limbic motivation can activate feeling, thought, and movement.

Lesions of the posterior cingulate disrupt memory function in animals and humans. The closing link in the circuit of Papez, from the anterior thalamic efferents traveling through the posterior cingulum to areas 32 and 29/30, is the cingulate projection sent to the presubiculum. Anterior cingulotomy will not disrupt this memory circuit but rarely pathologic lesions will extend into, and beyond, the posterior cingulate. If the lesion extends inferior to the splenium of the corpus callosum, it may also disrupt the fornix, thus disconnecting the efferents from the hippocampus to the diencephalon. If the lesion extends posteriorly, it may damage the supracommissural portion of the hippocampus – the gyrus fasciolaris and the fasciola cinerea. A large left-sided lesion that extended beyond the posterior cingulate into the fornix and supracommissural hippocampus after the surgical repair of an arteriovenous malformation resulted in a persistent amnesia (Cramon & Schuri, 1992). Disruption of septo-hippocampal pathways in the cingulum and fornix were thought by the authors to play a significant role in the patient's clinical deficit, but other important components of Papez's circuit had clearly been damaged. A rare lesion restricted to the left posterior cingulate, the cingulum, and the splenium of the corpus callosum (but possibly sparing the fornix) resulted in a severe amnesia after the repair of an arteriovenous malformation (Valenstein et al., 1987).

The analysis of rare circumscribed lesion in humans cannot determine if the posterior cingulate cortex, rather than the cingulum or neighboring members of Papez's circuit, result in amnesia when lesioned due to the location of fiber pathways to the hippocampus that are buried in the posterior cingulate. Excitotoxic lesions in animals that destroy neurons but spare fibers of passage can clarify this issue. Based upon posterior cingulate cortical lesions, using the selective cytotoxin quisqualic acid (Sutherland & Hoesing, 1993), results in animal studies reveal that area 29 neurons are necessary for the acquisition and retention of spatial and nonspatial memory. Furthermore, the posterior cingulate acts in concert with the anterior thalamus and the hippocampus during encoding and may also be important in the storage of long-term information.

Synthesizing cytoarchitectonic refinements, nonhuman primate tracer studies, clinical-behavioral correlation data, and functional neuroimaging results have been refined – but have not significantly added to – the basic description of cingulate function offered by Papez:

> It is thus evident that the afferent pathways from the receptor organs split at the thalamic level into three routes, each conducting a stream of impulses of special importance. One route conducts impulses through the dorsal thalamus and the internal capsule to the corpus striatum. This route represents 'the stream of movement.' The second conducts impulses from the thalamus through the internal capsule to the lateral cerebral cortex. This route represents 'the stream of thought.' The third conducts a set of concomitant impulses through the ventral thalamus to the hypothalamus and by way of the mamillary body and the anterior thalamic nuclei to the gyrus cinguli, in the medial wall of the cerebral hemisphere. This route represents 'the stream of feeling.' In this way, the sensory excitations which reach the lateral cortex through the internal capsule receive their emotional coloring from the concurrent processes of hypothalamic origin which irradiate them from the gyrus cinguli. (Papez, 1937)

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Cingulum

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Definition

The cingulum is a collection of white matter fibers projecting from the cingulate gyrus to the entorhinal cortex in the brain, allowing for communication between components of the limbic system. The cingulum connects the medial temporal lobe with the posterior cingulate gyrus. Diffusion tensor imaging studies have reported cingulum bundle disruption in mild cognitive impairment and Alzheimer's disease. In addition, some research indicates that in Alzheimer's disease, the posterior cingulate cortex hypofunction is due to the indirect effect of the degeneration of cingulum fibers secondary to medial temporal lobe atrophy.

Cross References

► Cingulate Gyrus

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CIQ

► Community Integration Questionnaire

Circadian Clock

► Circadian Rhythms

Circadian Rhythms

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Synonyms

Biological cycles; Biorhythms; Circadian clock

Short Description or Definition

The pervasive characteristics of living organisms are rhythmic biological and behavioral changes, which are expressed at varying levels of organization ranging from basic cellular to the highly complex level. These rhythms

are generally referred to as circadian rhythms and they organize a variety of behaviors, including sleep, which is characterized by a 90-min Rapid Eye Movement (REM) cycle and the wakefulness–sleep cycle, which is organized around an approximate 24-h cycle (Carlson, 1999).

Categorization

The core body temperature rhythm and the sleep–wake cycle are among the most extensively studied of the human biorhythms (Waterhouse & DeCoursey, 2004b) with the 24-h circadian rhythms represented most prominently in the research literature. This is because these rhythms impact daily life (e.g., work, school, medical–psychological status) and are of relatively brief duration, so they are amenable to study (Clark, 2005). There is also a developmental progression in how rhythmicity is expressed from birth to old age (Waterhouse & DeCoursey, 2004b). There are a number of biological rhythms characterized by different time periods (Table 1).

Mechanisms

Circadian rhythms are modulated by both internal clocks (e.g., pacemakers) and external triggers that can entrain biorhythms, acting as zeitgebers (e.g., changes in illumination). The superchiasmic nucleus (SCN) of the hypothalamus has been identified as the principal biological clock (Carlson, 1999) based on lesion studies and day–night changes in activity levels. The mechanisms mediating communication and synchronization between neurons appear to be chemical in nature. Both the SCN and the pineal gland exert an influence on seasonal rhythms with SCN-induced melatonin secretion involved in synchronizing circadian rhythms (Carlson, 1999).

Circadian Rhythms. Table 1 Biological rhythms

Biological rhythms	Time period	Activity
Infradian	Period of less than 24 h	Eating behaviors
Circadian	Period of about 24 h	Sleep–wake
Ultradian	Period of about 28 days	Human female menstrual cycle
Circannual	Yearly	Migratory birds

Developmental factors may also mediate some of the variations in circadian rhythms. For example, the aging process is often accompanied by changes in circadian rhythms that may affect sleep and place older adults at a higher risk for sleep disturbance (Lee-Chiong, 2005).

History and Impact

Endogenous biological clocks were demonstrated over 200 years ago (Kolb & Wishaw, 2005) and entrainment was identified as the most important property for determining the phase relationship of a circadian clock (Johnson, Elliot & Foster, 2004). Circadian rhythms may confer an adaptive value on an organism, since the organism can anticipate environmental changes (Clark, 2005), and physiological functions synchronized with the time of day are associated with enhanced efficiency (Quigg, 2006). Disruption in circadian rhythms can adversely impact individuals involved in shift and night work, who suffer jet lag or whose schedules are temporally irregular. The impact is more profound when task demands involve vigilance and reaction time (Costa, 1999) and are, therefore, more vulnerable to the effects of fatigue. Attentional regulation over both incoming information and outgoing responses may also be vulnerable to time of day effects. Disturbances in sleep–wake cycles and biological rhythms have been associated with affective disorders including depression.

Evaluation

There are large differences in circadian cycles between younger and older adults. Clinicians should assess the quantitative and qualitative nature of cognitive decline in the elderly with attention to circadian influences (with the elderly tending to be morning oriented) (Hasher, Goldstein & May, 2005). In evaluating clients for possible disruptions in circadian rhythms, clinicians would be well advised to determine if clients engage in shift work, have experienced jet lag or changes in schedule, or exhibit fatigue, sleep disorders, excessive daytime drowsiness, or decrements in job-related performance (Costa, 1999).

Treatment

While the circadian clock of healthy humans is entrained to a period of about 24 h, the precise timing varies on an

intraindividual and interindividual basis. At a certain point, these deviations may be considered clinical abnormalities (i.e., seasonal affective disorder). In fact, seasonal affective disorder has been treated using phototherapy, which involves the use of light boxes, whereby a patient is exposed to light each winter morning or evening and the circadian rhythm becomes entrained in ways that induce circadian phase shifts (Kolb & Wishaw, 2005). Individuals who make substantial changes to their sleep–wake cycles during night work or after trans-meridian travel could be helped using a chronobiological treatment. The treatment would involve exposure to appropriate circadian synchronizers and basing therapy on phase response curves for light and melatonin (Waterhouse & DeCoursey, 2004a). By making accommodations in the timing of work-related activities (Hasher et al., 2005), performance efficiency can be improved and fatigue decreased, thus enhancing health and safety (Costa, 1999). And in treating patients with bipolar disorder, psychosocial therapies should identify areas of vulnerability including unhealthy circadian rhythms (Newman, 2006).

Cross References

► Fatigue
► Sleep Disturbance

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Circle of Friends

► Circles of Support

Circle of Willis

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Definition

The circle of Willis is the anatomical name given to the formation of arteries at the base of the brain that contribute the overwhelming majority of blood supply to the brain.

Current Knowledge

The circle of Willis is formed by the connections between the predominantly horizontal branches that derive from the middle cerebral arteries anteriorly and from the basilar artery posteriorly. The right and left middle cerebral arteries each give off an anterior cerebral artery (forming the anterolateral borders of the circle of Willis), which goes forward to supply blood to the frontal lobe. These anterior cerebral arteries are connected to each other by the anterior communicating artery, which forms the front of the circle. Posteriorly, the basilar artery bifurcates into the right and left posterior cerebral arteries, which supply the occipital and posterior temporal lobes and the cerebellum, forming the posterior

border of the circle. Each posterior cerebral artery is connected to the middle cerebral artery on its corresponding side by a posterior communicating artery, forming the posterolateral borders of the circle. There are several clinical implications of the circle pattern of these arteries. Perhaps most importantly, because of the interconnectedness of the arteries that result from this circle format, if one of the main arteries is occluded, the distal smaller arteries that it supplies can potentially receive blood from the other arteries that make up the circle, a phenomenon known as collateral circulation. This helps to prevent cerebral ischemia and stroke. The circle of Willis also is a common site for cerebral aneurysms, with the greatest numbers involving the anterior communicating artery, posterior communicating arteries, and middle cerebral arteries.

Cross References

- ▶ Anterior Cerebral Artery
- ▶ Anterior Communicating Artery
- ▶ Basilar Artery
- ▶ Internal Carotid Artery
- ▶ Middle Cerebral Artery
- ▶ Posterior Cerebral Artery
- ▶ Posterior Communicating Artery
- ▶ Vertebrobasilar System

Circles of Support

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Synonyms

Circle of friends

Definition

A circle of support is a group of people that forms a community around a specific individual (focus person) with significant disabilities to assist him or her to achieve personal goals. It is one of many tools addressing life planning from a functional or strategic assessment approach known as person-centered planning. Person-centered planning replaces more traditional assessment approaches associated with the medical model of services. Circles of support originated in Canada and have experienced widespread use in North America. Circles view people as individuals and assist them to attain self-determination focusing upon empowerment and not dependence of the individual. It is capacity oriented and identifies strengths, preferences, likes, and dislikes of the individual. The circle will also identify support needs in order to achieve a particular goal.

The focus person leads the process and decides who will participate in the circle and the direction which the planning will take. Typically, a facilitator is selected from within the circle to help energize the group. The first circle is the circle of intimacy and includes the people most intimate in the focus person's life. The second circle, the circle of friendship, includes good friends and close relatives. The third circle, the circle of participation includes the people and organizations the focus person is involved with. The fourth circle is the circle of exchange and includes those that are paid to be in the focus person's life. Members are not paid to be involved in a circle of support, but are involved in the focus person's life in some capacity. Members use their skills, knowledge, and networks to help the focus person accomplish goals. The circle develops and monitors the plan, making sure that it is current with the wishes of the focus person. It is as ongoing process that is dynamic.

Cross References

- ▶ Inclusion
- ▶ Medical Model

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Circumduction

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Synonyms

Spastic gait

Definition

Circumduction describes the movement of the leg of a person with hemiplegia, hemiparesis or paraplegia, paraparesis. Lesions of the pyramidal tract cause more weakness in the flexors of the leg than the extensors (hip and knee flexors and ankle dorsiflexors). Because of this weakness, the foot cannot be raised from the floor, and the leg cannot be advanced in a straight line forward, as it would do normally. The leg moves away from the trunk, then it is advanced toward it during walking, in a circular pattern. The medial side of the shoe scrapes the floor, causing excessive wear in that area.

Cross References

- ▶ Hemiparesis
- ▶ Hemiplegia
- ▶ Pyramidal Tract

References and Readings

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Circumlocution

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Definition

The use of an unnecessarily large number of words to express an idea. Evasion in speech. Circumlocutions are often used by persons with aphasia when having difficulty recalling or retrieving a word. In place of the target word, a description of the word is used. Circumlocutions, or "*substitutions of object description (e.g., snow/soft, white/cold) and instrumental function (e.g., watch/knowing the hour) can be observed in aphasic output*" (Benson & Ardila, 1996; p. 53). They occur frequently with a posterior (sensory) aphasia.

Circumlocutions can represent a positive symptom of anomia in which, upon failure to retrieve a word, the subject talks around the word by defining it, describing a referent, or even making sound effects. Pointing to his wrist, a patient might say, "I wear it right here, and I tell time with it; mine goes tick, tick." *The use of circumlocutions "is indicative of intact semantic activation and a general capacity to retrieve lexical forms" (Davis, p. 109).*

Cross References

- ▶ Anomia
- ▶ Aphasia

References and Readings

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Circumstantiality

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Definition

Circumstantiality is circuitous thinking and speech that digresses from the essential point. It differs from tangentiality in which the individual ultimately fails to address the main idea. In circumsatantiality, the main point is never lost but may be "clouded" and its appearance

delayed by excess and repeated material. Circumstantial thinking is a characteristic of thought disorders.

Cross References

► Tangentiality

Circumventricular Organ

► Subfornical Organ

Citalopram

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Generic Name

Citalopram

Brand Name

Celexa

Class

Selective Serotonin Reuptake Inhibitor

Proposed Mechanism(s) of Action

Citalopram blocks the presynaptic serotonin reuptake pump and desensitizes serotonin receptors, theoretically increases serotonin neurotransmission, and is a mild antihistamine.

Indication

Depression.

Off Label Use

Premenstrual dysphoric disorder, obsessive compulsive disorder, panic disorder, generalized anxiety disorder, posttraumatic stress disorder, and social anxiety disorder.

Side Effects

Serious

Seizures, mania, and suicidal ideation (all rare).

Common

Sexual dysfunction, gastrointestinal upset, insomnia, sedation, tremor, headache, dizziness, sweating, bruising and very rare bleeding, rare hyponatremia, and a potential for SIADH (syndrome of inappropriate antidiuretic hormone secretion).

References and Readings

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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

Civil Action

► Litigation

Civil Law

► Personal Injury

Civil Litigation

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Definition

Civil law is a division of the law that deals primarily with disputes between individuals and/or organizations, in which some form of compensation may be awarded to the victim. Typically, civil law involves filing of a lawsuit by a private party, called "the plaintiff." Civil litigation commonly involves hearing related to disputes regarding torts, contracts, probate of wills, trusts, property, administrative law, commercial law as well as a plethora of other matters related to private parties and organizations. Civil litigation primarily aims to correct an injustice, uphold an agreement, or settle a dispute. If compensation is awarded to the victim, then the person/organization responsible for the injustice is responsible for covering the compensation. In civil litigation, the burden of proof is usually placed on the plaintiff, though exceptions do exist. Punishment related to civil litigation is typically limited to reimbursement for loses incurred by the plaintiff as a result of actions/inactions committed by the defendant. Incarceration is not a punishment rendered via civil litigation. Civil litigation and criminal litigation are not mutually exclusive entities. For example, an individual involved in a criminal case may seek compensation in civil court. For neuropsychologists, civil litigation typically involves determination of causation, damages, and disability in disputes in which neuropsychological functioning is of relevance (e.g., personal injury, medical malpractice).

References and Readings

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CJD

▶ Creutzfeldt-Jakob Disease

Classical Model of Aphasia

▶ Wernicke–Lichtheim Model of Aphasia

Classical Test Theory

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Definition

Classical test theory is the body of concepts and methods that have formed the basis for psychological assessment. Classical test theory posits that observed scores are the additive function of true scores and error terms. True scores are the ideal value of a construct in a particular person or situation. The error term is the effect of factors extraneous to the construct of interest. Error terms are assumed to be independent of (or uncorrelated with) the true scores. Analysis of the reliability of a score can be accomplished by manipulating factors thought to be influencing the error term. For example, in order to examine the effect of factors related to time or instance of measurement, a test might be administered to the same individuals on two different occasions. The relation between the two observed scores, determined by calculating a correlation coefficient or by performing an analysis of variance, helps to estimate the magnitude of the error term and the proportion of the observed score that is likely to be true score.

Cross References

▶ Item Response Theory
▶ Reliability
▶ Sources of Error

References and Readings

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Clear and Convincing Evidence

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Definition

The burden of proof is the obligation to shift the assumed conclusion away from an oppositional opinion to one's own position: it may only be fulfilled by evidence. Under the Latin maxim, necessitas probandi incumbit ei qui agit, the general rule is that "the necessity of proof lies with he who complains." The burden of proof, therefore, usually lies with the party making the claim. The exception to this rule is when a prima facie case has been made. He who does not carry the burden of proof carries the benefit of assumption, meaning he needs no evidence to support his claim. Fulfilling the burden of proof effectively captures the benefit of assumption, passing the burden of proof off to another party. Clear and convincing evidence is a burden of proof required of a plaintiff for him to win the lawsuit. This standard is higher than mere preponderance of the evidence. Proof of fraud, for example, usually requires clear and convincing evidence. Clear and convincing evidence is the higher level of burden of persuasion and is most often employed in civil litigation. To prove something by "clear and convincing evidence," the party with the burden of proof must convince the trier of fact that it is substantially more likely than not that the thing is in fact true. This is a lesser requirement than "proof beyond a reasonable doubt," which requires that the trier of fact be close to certain of the truth of the matter asserted, but a stricter requirement than proof by "preponderance of the evidence," which merely requires that the matter asserted seem more likely true than not.

Cross References

▶ Beyond Reasonable Doubt
▶ Burden of Proof
▶ Preponderance of Evidence

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.

Client Generated Index (CGI)

▶ Patient Generated Index

Clinical Dementia Rating

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Synonyms

CDR

Description

The Clinical Dementia Rating (CDR; Hughes, Berg, Danziger, Coben, & Martin, 1982) is a semi-structured, clinician-rated interview widely used to stage the progression of dementia using information provided by the patient and an informant. A global CDR score is generated to stage the severity of dementia. It is based on ratings of the patient's functioning in six domains commonly affected in Alzheimer's disease (AD): memory, orientation, judgment and problem solving, community affairs, home and hobbies, and personal care. The CDR rates only

impairments due to cognitive deficits rather than to physical disability. Based solely on clinical information obtained from the patient and informant, and without reference to psychometric performance, a box score describing the level of impairment is generated for each domain. The box score ranges from 0 to 3, representing "none" to "severe" impairment. Using a scoring algorithm, one of five possible stages is then derived from the individual box scores as follows: CDR = 0 (no dementia); CDR = 0.5 (questionable dementia); CDR = 1 (mild dementia); CDR = 2 (moderate dementia); CDR = 3 (severe dementia). Although not part of the original protocol, CDR = 4 (profound) and CDR = 5 (terminal) can also be used to classify the later stages of dementia. An alternative method that generates a total score (range 0–18) from the sum of boxes (CDR-SB) has also been used for quantification purposes in longitudinal studies (e.g., Cortes et al., 2008). The CDR takes about 90 min to administer.

The CDR has been used in clinical practice and multicenter clinical trials, as well as in cross-cultural dementia studies around the world. The CDR protocol is available in over 60 languages and dialects, including English, French, Spanish, Italian, Portuguese, Dutch, Czech, Bulgarian, Russian, Tagalog, Afrikaans, Greek, German, Hebrew, Indian dialects, Chinese dialects, Japanese, and Korean. These translations can be downloaded free of cost on the CDR Web site (http://alzheimer.wustl.edu/cdr/default.htm).

An online training video on the use of the CDR is available free for individual users, and takes about 8–9 h to complete. Detailed scoring algorithms, including "tie-break" rules, and an online scoring worksheet are available on the CDR Web site.

Historical Background

The CDR was originally developed at the Washington University School of Medicine in 1979 to evaluate the progression of AD (Hughes et al., 1982). The original protocol has evolved somewhat over the years, with box descriptors updated to sharpen the distinction between severity levels within each domain, and new scoring rules added to resolve scoring ambiguity (Morris, 1993). Alternative scoring methods have also been suggested to improve scoring accuracy (Gelb & St. Laurent, 1993). An extension of this scale to include CDR = 4 (profound) and CDR = 5 (terminal) to classify the later stages of dementia (Dooneief, Marder, Tang, & Stern, 1996) among nursing home elderly has been proposed. As well, another method that uses a total score generated from the sum of boxes (CDR-SB) has gained popularity to quantify progression of AD in longitudinal clinical trials of experimental therapies (e.g., Petersen et al., 2005).

Psychometric Data

Interrater Reliability

Most of the psychometric studies on the CDR have focused on interrater reliability in multicenter clinical trials. These studies have concluded that experience using the CDR increases reliability estimates, although adequately trained inexperienced raters may also demonstrate a high level of agreement (kappa = 0.83 or higher; Schafer et al., 2004; Tractenberg, Schafer, & Morris, 2001).

The CDR also shows good reliability among raters of various qualifications. There were no major differences in reliability among physicians, nurses, PhDs, social workers, psychometrists, or research assistant raters (85% for non-MDs and 82% for MDs; Oremus, Perrault, Demers, & Wolfson, 2000). Kappas between physician raters range from 0.75 to 0.94 for the six individual domain scores and CDR-SB score. Kappas between nurses, or between nurses and physicians, range from 0.66 to 0.77 (Oremus et al., 2000).

Construct Validity

Evidence for construct validity of the CDR appears solid. In the original study, the CDR had strong correlations with the Blessed Dementia Scale (BDS; r = 0.74) and the Pfeiffer Short Portable Mental Status Questionnaire (SPMSQ; r = 0.84) among individuals with CDR ratings between no dementia and very mild dementia (Hughes et al., 1982). Correlations with various cognitive measures ranged from small to large in community-dwelling samples (Folstein's Mini-Mental State Examination = 0.33; BDS = 0.74; SPMSQ = 0.84). Similar correlations were found with neuropsychological measures such as the CERAD Boston Naming Test, list learning, and verbal fluency (Oremus et al., 2000).

In terms of neuropathology, CDR = 0.5 (a proxy for mild cognitive impairment; MCI) has been associated with multiple pathological signs including those related to AD, Lewy body dementia, or vascular dementia, as well as nonspecific pathology (Saito & Murayama, 2007). Further, increased microglia activation, an inflammatory biomarker of AD, was seen with advancing CDR

stages (Xiang, Haroutunian, Ho, Purohit, & Pasinetti, 2006). A negative association was also found between the CDR-SB score and glucose metabolism in the right posterior cingulate gyrus (Perneczky, Hartmann, Grimmer, Drzezga, & Kurz, 2007).

Predictive Validity

There is evidence that individuals with higher CDR-SB scores are more likely to develop dementia in the future (Lynch et al., 2006). In addition, CDR scores predict survival in individuals with suspected dementia. Using survival as outcome, the median survival was 1 year for CDR = 5, 2 years for CDR = 4, 2.5 years for CDR = 3, 3 years for CDR = 2, and 3.5 years for CDR = 1 (Dooneief et al., 1996). Use of the CDR as a screening tool for dementia revealed a sensitivity of 92% and specificity of 94% for mild dementia in a community sample of adults older than age 75 (Juva et al., 1995).

Clinical Uses

The original focus of the CDR was to assess community-dwelling older adults, since its anchor points probe for examples of one's engagement with the home and community. However, it has also been adapted for use in chronic long-term care facilities (Marin et al., 2001). The global CDR is widely used primarily for the staging of AD. It has also been used to stage other dementing disorders such as Parkinson's disease and frontotemporal dementia. In recent years, CDR = 0.5 has been used to characterize MCI; however, there is evidence that a large proportion (29.7%) of individuals at CDR = 0.5 also meet ICD-10 criteria for mild dementia (Lynch et al., 2006). In terms of longitudinal studies of AD progression, both the global CDR and the CDR-SB appear to be useful for tracking cognitive changes over a 2–3 year period (e.g., Cortes et al., 2008; Meguro et al., 2004).

Of the two scoring methods, the CDR-SB is more commonly used in clinical drug trials because it was found to be sensitive to changes within 12 months following baseline measurement in donepezil drug trials, whereas the global CDR was not (e.g., Petersen et al., 2005). There is also evidence that the CDR-SB is more useful than the global CDR in distinguishing mild cognitive deficits from dementia (Lynch et al., 2006).

In addition to western populations, use of the CDR has also been accepted as an appropriate comprehensive measure for studies of dementia patients in Asian populations (Lim, Chin, Lam, Lim, & Sahadevan, 2005; Senanarong, Chen, & Orgogozo, 2006), although empirical validation of various translations is needed.

The CDR presents several advantages over psychometric tests. First, it is an assessment option for patients who are illiterate and/or have limited English language proficiency. Moreover, it can also be used in the presence of aphasia, a condition common among patients with dementing disorders. Lastly, the administration of the CDR does not require a standardized set of instructions, but is dependent upon a set of guidelines. As such, the CDR is easily adapted for cross-cultural use (e.g., Lim et al., 2005). A disadvantage of the CDR is that it is somewhat lengthy to administer.

Cross References

- ▶ Alzheimer's Disease
- ▶ CERAD
- ▶ Dementia

References and Readings

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Clinical Depression

► Major Depression

Clinical Extender

► Psychometrician

Clinical Importance

► Clinical Significance

Clinical Interview

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Synonyms

Diagnostic interview; Intake; Intake interview; Unstructured clinical interview

Definition

A skillfully conducted clinical interview is the cornerstone of psychological assessment. This interaction, typically a face-to-face meeting that lasts between 30 min and 2 h, generates a tremendous amount of data for the clinician via both observation and direct questioning. Information obtained through observation during the clinical interview is considered qualitative or descriptive, and can include impressions about cognition, attention, orientation, language, sensorimotor deficits, affect, insight, attitude toward assessment, acculturation, hygiene, interpersonal relations, and coping mechanisms, among other variables. In addition, the verbal exchange between patient and clinician yields information about current life circumstances, including the reason for referral and history of the presenting problem, as well as an account of developmental/medical/family history, educational and occupational achievement, legal problems, sociocultural/religious considerations, substance abuse, and other relevant psychiatric issues. Together with test data and collateral information, interview material is an invaluable tool for clinical hypothesis formulation and testing, as well as treatment planning.

Current Knowledge

There are three types of clinical interview, reflecting the degree to which the content and questions are scripted: structured, semi-structured, and unstructured. A structured interview (e.g., the Structured Clinical Interview for DSM-IV-Clinical Version [SCID-I]; First, Gibbon, Spitzer, & Williams, 2001), like any standardized assessment tool, gathers specific data that allows clinicians to make comparisons between client and normative group function. Criticisms of structured clinical interviews include a frustration with lengthy questionnaires and the missed

opportunity for meaningful dialogue (Maruish, 2008). An unstructured clinical interview, on the other hand, is principally reliant on clinical skill for direction. An optimal unstructured clinical interview involves moving from broad content areas to more specific ones, from open-ended to more directive questions seeking yes/no responses. While the goals of clinical interviewing remain much the same regardless of format, critics have argued that bias is more easily introduced into unstructured clinical interviews than standardized approaches. A hybrid, the semi-structured clinical interview, offers many of the benefits of its structured and unstructured counterparts, with breadth and depth chief among them. The semi-structured format ensures that all areas of potential clinical concern are assessed, while affording the clinician the flexibility to dictate the degree of attention each content area receives. In addition, the semi-structured clinical interview can be altered to accommodate disabilities, it can be abbreviated to meet the needs of the client (e.g., fatigue) or the setting (e.g., bedside assessment), and it can be amended to include additional lines of inquiry.

As well as yielding information regarding patient history and current functioning, a clinical interview offers opportunities to build rapport and foster a working alliance. In addition to promoting satisfaction with the assessment process, the development of rapport may promote compliance from a reluctant patient and provide a foundation for follow-up discussions and interventions that may be indicated. Also, the dialogue during a clinical interview allows a clinician to provide important patient education. The initial conversation may explicitly address confidentiality, insurance/fee setting, the nature and purpose of the examination, the intended use of assessment data, and a summary of follow-up plans including feedback sessions (Lezak, Howieson, & Lorig, 2004).

Despite routine use in clinical practice, there is considerable debate about the reliability of clinical interviews. Many believe the variability in questioning undermines the utility of the tool itself. Some research has suggested that unstructured clinical interviews often fail to detect psychiatric and comorbid conditions (Zimmerman & Mattia, 1999). Other researchers find "empirical rigor" in skillful clinical interviewing (Shedler, 2002, p. 429). This argument notwithstanding, the clinical interview remains a staple assessment tool, as fallible or effectual as the clinician.

Cross References

- ▶ Behavioral Assessment
- ▶ Mental Status Examination
- ▶ Referral Question
- ▶ Self-Report Measures
- ▶ Structured Clinical Interview for DSM-IV (SCID-I)
- ▶ Structured Interview of Reported Symptoms (SIRS)

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Clinical Neuropsychology

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Definition

Clinical neuropsychology is a specialty within psychology that applies the science of brain-behavior relations to the assessment, diagnosis, treatment, and rehabilitation of patients across the life span with neurological, medical, neurodevelopmental, psychiatric, or other cognitive and learning disorders (Barth et al., 2003). The American Psychological Association (APA) defines a clinical neuropsychologist as "a professional psychologist who applies principles of assessment and intervention based upon the scientific study of human behavior as it relates to normal and abnormal functioning of the nervous system" (APA, 1989, pp. 22).

Clinical neuropsychology is recognized as a specialty by APA, the American Board of Professional Psychology, and the Canadian Psychological Association. Though there is no agreed upon date for the emergence of the field, clinical neuropsychology began to be recognized as a distinct professional discipline following a 1948 symposium at the APA annual meeting appropriately entitled "Neuropsychology." In this symposium, Hans-Lukas Teuber described procedures he and Morris Bender developed to study the behavioral effects of penetrating missile wounds to the brain (Benton, 1987).

Clinical neuropsychology had many forerunners. In the 1880s, Sir Francis Galton opened a laboratory in London where for a few pennies, people could take tests of visual acuity, reaction time, and various psychophysical abilities. A cousin of Charles Darwin, Galton's ideas about eugenics have made him a historically controversial figure. Nonetheless, factor analytic statistical methods grew out of his work and became critical for the development and validation of mental ability tests. His work was put to practical use in early twentieth-century France when psychologists Alfred Binet and Théodore Simon developed intellectual tests to identify and place children in need of special education. World War I also stimulated interest in his work in the USA, as the military sought tests that could efficiently identify the strengths and weaknesses of large numbers of military recruits. As many as one million soldiers underwent mental ability testing during World War I (Hartman, 1991).

Just after World War I, the German American psychologist Shepherd Ivory Franz (1919) published detailed descriptions of tests of tactile sensation, motor coordination, praxis, language, attention, memory, visuospatial perception, reasoning, and intelligence. By 1924, hand dynamometers, finger tapping keys, motor steadiness tests, form perception boards, and tests of color vision, vibration sense, attention, and memory were available (Hartman, 1991). Though these tests were quickly adopted by researchers (see *Neuropsychology, Science of*), clinical application came a few decades later. Franz advocated for their clinical use in a series of lectures to the Government Hospital for the Insane, starting in 1910 (Hartman, 1991) and in 1904 was instrumental in establishing a psychological laboratory at Mclean Hospital in Boston. This was the first such laboratory in a hospital setting in the USA

In 1935, the experimental psychologist Ward Halstead opened a laboratory at the University of Chicago for the psychological study of neurology and neurosurgery patients. Adopting techniques used to test animals in ablation studies, Halstead designed a series of tests intended to measure what he termed "biological intelligence." The influence of Galton's statistical methods is seen in Halstead's factor analysis of his test battery. Halstead proposed a four-factor theory of biological intelligence in his 1947 book *Brain and Intelligence.* Halstead's book was severely criticized by contemporary scholars, and ultimately had little influence on theories of intelligence (Hartman, 1991). Halstead's test battery, however, did prove highly influential. Ralph Reitan, a student of Halstead, established a laboratory at the University of Indiana Medical Center in 1950. Over the course of several decades, Reitan expanded upon Halstead's initial battery, adding procedures for detecting aphasia and sensory perceptual impairments and for comparing performances of the two sides of the body. Reitan developed adaptations of the original adult battery for children and adolescents, collected normative data, and used discriminant function analysis to validate the ability of the various batteries to discriminate brain damaged from neurologically healthy individuals. The Halstead–Reitan Neuropsychological Test Battery set a standard of excellence for test development in neuropsychology and for a time was the most widely used approach in clinical neuropsychological assessment.

Reitan's students were also prolific (Reed, 1985). They include Hallgrim Kløve who studied with Reitan in the 1950s and then established a laboratory at the University of Wisconsin. Another student, Homer Reed, directed the Neuropsychology Laboratory at the New England Medical Center in Boston where he concentrated on use of the battery with pediatric patients. Phillip Rennick, who did fellowship training with Reitan, went on to establish a laboratory at the former (now defunct) Lafayette Clinic in Detroit, and developed a repeatable neuropsychological battery for situations in which serial testing is needed.

Just as World War I provided an impetus for the development of psychological ability tests, World War II stimulated the development of neuropsychological assessment and treatment methods because of the large number of veterans who returned having survived penetrating missile wounds to the brain. Reitan's early work involved the examination of brain-injured World War II veterans. This population also provided the impetus for Hans-Lukas Teuber's work in the USA and Aleksandr Romanovich Luria's work in Russia.

Teuber immigrated from Germany and served as a noncommissioned naval officer before establishing the Psychophysiological Laboratory with the neurologist Morris Bender at New York University (Weinstein, 1985). Besides being the focal point for numerous seminal studies, Teuber's laboratory was the incubator for many

neuropsychologists who went on to make important contributions including Joseph Altman, Lila Ghent, Rita Rudel, Josephine Semmes, and Sidney Weinstein. Meanwhile, working with Russian World War II veterans at the Burdenko Neurosurgical Institute in Moscow, Luria developed a richly qualitative approach to neuropsychological assessment that was in stark contrast to the quantitative and normatively based test batteries that were in use in the USA Luria also was an exponent of neuropsychological approaches to rehabilitation and to use of pharmacologic agents to enhance recovery of function (Gualtieri, 1988).

Besides having cognitive disorders, many injured World War II veterans were aphasic, sparking neuropsychological interest in language and the brain. Kurt Goldstein's (► *Goldstein, Kurt*) book *Language and Language Disorders*, published in 1948, combined his theories about abstracting ability with neurology's classic anatomico-clinical syndrome approach to aphasia. The numbers of aphasic veterans available for study along with Goldstein's influential book attracted experimental neuropsychologists and psycholinguists to the study of language disorders (Goodglass, 1985). Aphasia research centers began to appear in the USA, among them being a laboratory established by Arthur Benton.

In 1948, the year of Teuber's seminal APA presentation, Arthur Benton accepted an appointment as Professor of Psychology at the University of Iowa, and by 1950 established a neuropsychological testing unit at the University of Iowa Hospitals (Hamsher, 1985). There, Benton and his students and colleagues conducted normative studies, examining the effects of age, gender, and education. They also developed a variety of tests for use in studying what had previously been vaguely defined clinical disorders such as the Gerstmann syndrome (► *Gerstmann Syndrome*). Over the next 2 decades, Benton's laboratory was home to numerous neuropsychology pioneers including Max Fogel, Donald Shankweiler, Kerry deS. Hamsher, Nils Varney, Scott Lindgren, Otfried Spreen, and Harvey Levin (Hamsher, 1985). Benton's laboratory conducted important studies of aphasia using control group designs, psychological test construction methods, statistical analysis, and psycholinguistic theory.

The behavioral neurologist Norman Geschwind, along with his colleagues Davis Howes and Harold Goodglass, established an aphasiology center at the Boston Veterans Administration Hospital, continuing the more rigorous neuropsychological approach to language disorders. The Boston VA became a major center for neuropsychological training and research with Dr. Edith Kaplan famously serving there as "mother" to a generation of neuropsychology practitioners and researchers. Dr. Kaplan and her associates in Boston developed the process-approach to neuropsychological assessment, recognizing that tests are complex and multifactorial and that patients can take different paths to the same test score. According to this approach, only by systematically analyzing the process(es) by which patients arrive at their responses, often by parsing a test into fine-grained components, can a neuropsychologist truly understand what aspect of brain functioning is compromised.

Activity was not confined to North America. Clinical neuropsychology got its South American start when C. Mendilaharsu and S. Acevedo de Mendilaharsu established the first South American neuropsychological laboratory in 1958 at the Neurological Institute in Montevideo, Uruguay (Ardila, 1990). Despite sometimes challenging economic conditions, South American neuropsychologists conducted investigations of constructional ability, dementia, and language. The field spread to Mexico, Peru, Columbia, Chile, Argentina, Brazil, Honduras, Nicaragua, and elsewhere.

Clinical neuropsychology as a profession has become increasingly organized since its inception. The International Neuropsychological Society (INS), the National Academy of Neuropsychology (NAN), and the APA Division of Clinical Neuropsychology (Division 40) are the most well-known professional neuropsychological organizations within the USA. Founded in 1967, INS has a membership that exceeds 3,500. NAN, formed in 1974, includes more than 3,000 members. Division 40 of the American Psychological Association, incorporated in 1980, is the most recent of these professional organizations, with a membership of over 4,000 neuropsychologists. Although smaller in membership, neuropsychologists have formed similar professional organizations throughout Europe, Asia, South America, Australia, and Africa (see, for example, Jodzio, 1998; Nihashi, 1998; Preilowski, 1997).

Formal courses and organized programs of instruction in clinical neuropsychology began to appear in the 1960s, with one of the first offered in the Biological Psychology Doctoral Program at the University of Oklahoma Health Sciences Center (Parsons, 1991). Quality and content of instruction varied from program to program as they multiplied in the 1970s. In 1979, Dr. Manfred Meier initiated an effort to establish standards of training and competence for neuropsychologists in the USA. This effort culminated first in the establishment of the American Board of Clinical Neuropsychology (ABCN) in 1981. A year later, the American Board of Professional Neuropsychology (ABN) was established

with a similar goal of establishing standards of professional expertise in neuropsychology. These two certification bodies continue to function autonomously, though ABCN affiliated with the American Board of Professional Psychology (ABPP) in 1984, joining other boards that conduct peer review of the credentials, knowledge, and practice of clinical psychologists seeking subspecialty certification in the USA.

In 1987, APA Division 40 published guidelines for doctoral, internship, and postdoctoral training in neuropsychology and a year later formally adopted the definition of a clinical neuropsychologist quoted above. Included in this definition (but not quoted) were explicit training, supervision, licensing, and peer review requirements that must be met by neuropsychologists. APA also recognized attainment of the ABCN/ABPP diploma as "the clearest evidence of competence as a Clinical Neuropsychologist" (APA Division 40, 1989). As of 2010, over 500 practicing clinicians have attained ABPP/ABCN diplomate status in neuropsychology and over 350 neuropsychologists had attained ABN certification.

Though ABCN and ABN initially differed in their examination procedures, currently both boards require a peer review of credentials, a multiple-choice written exam, and a 1 h ethics oral exam. The two boards differ, however, in other aspects of their oral examination. In particular, ABCN, but not ABN, conducts an oral "fact-finding" examination in which candidates solicit information about a neuropsychological patient in order to arrive at a diagnostic formulation and clinical recommendations. Nonetheless, the two boards have grown more similar since their inception, and have discussed merger, but these efforts have been unsuccessful. One milestone likely to occur in the USA in the twenty-first century is the achievement of widespread ABCN or ABN diplomacy among those identifying neuropsychology as their primary area of specialization. This milestone would signal the professional and organizational maturity of clinical neuropsychology.

Cross References

- ▶ American Board of Clinical Neuropsychology (ABCN)
- ▶ American Board of Professional Neuropsychology
- ▶ American Psychological Association (APA), Division 40
- ▶ Benton, Arthur (1909–2006)
- ▶ Forensic Neuropsychologist
- ▶ Forensic Neuropsychology
- ▶ Geschwind, Norman (1926–1984)
- ▶ Halstead, Ward (1908–1968)
- ▶ International Neuropsychological Society
- ▶ Luria, Alexander Romanovich (1902–1977)
- ▶ National Academy of Neuropsychology
- ▶ Neuropsychiatry
- ▶ Neuropsychology, Science of
- ▶ Reitan, Ralph (1922–)
- ▶ Teuber, Hans-Lukas (1916–1977)

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Clinical Practice Guidelines

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Synonyms

Practice guidelines

Definition

According to the Institute of Medicine's 1990 report, *Clinical Practice Guidelines: Directions for a New Program*, clinical practice guidelines are "systematically developed statements to assist practitioner and patient decisions about appropriate health care for specific clinical circumstances."

Historical Background

Recommendations for appropriate care have been found in ancient writings (IOM, 1992). Modern guidelines have been developed by professional organizations for over 50 years. It was only in the 1990s, however, that the systematic, evidence-based guidelines began to appear with any regularity. In November 1989, the Agency for Health Care Policy and Research (AHCPR) was created with, among other responsibilities, a mandate to develop, disseminate, and evaluate clinical practice guidelines. The AHCPR then enlisted the Institute of Medicine (IOM) for advice. The result was the 1990 report generated by the IOM, called Clinical Practice Guidelines: Directions for a New Program, that aimed to encourage standardization and consistency in the development of guidelines.

Current Knowledge

The five major purposes of clinical practice guidelines are to: (1) aid in clinical decision-making by patients and health care providers, (2) educate individuals or groups, (3) assess and ensure quality of care, (4) guide the allocation of health care resources, and (5) reduce the risk of liability for negligent care (IOM, 1992).

In addition to patients and their families, health care providers, and health care institutions, others such as payers, health care benefit providers, and public policy makers have a stake in clinical practice guidelines. Legislators, regulators, and health care purchasers take an interest in studying them to assist them in making decisions that control health care costs. It is assumed that if the most appropriate care is administered, better health outcomes will be achieved and health care costs will be lowered. Although some guidelines could likely achieve such hopes for decreased health care costs, other guidelines may not.

The Agency for Healthcare Research and Quality (AHRQ), formerly known as the AHCPR, created a central public resource for evidenced-based clinical practice guidelines called the National Guideline Clearinghouse (NGC) that can be accessed at http://www.guideline.gov/

For a clinical practice guideline to be included on the NGC, the following criteria must be met: (1) The guideline must contain systematically developed recommendations, strategies, or information that assist in the decision making of appropriate health care in specific clinical situations by healthcare providers and patients; (2) the guideline must be produced by a medical specialty association, professional societies, public or private organizations, government agencies, or health care organizations or plans; (3) a documentation should be produced that verifies that a systematic literature search and review of scientific literature was performed during the guideline development; (4) the guidelines should have been reviewed or produced within 5 years and should be available in print or electronic format in the English language (National Guideline Clearinghouse, 2009). These criteria ensure minimum quality of guidelines submitted to the NGC.

Future Directions

An ongoing challenge is the implementation of clinical practice guidelines and ensuring their application by health care providers. Factors such as information overload, habitual practice patterns, fears of malpractice, and a lack of economic incentives create barriers to guideline application and each must be addressed on an individual and systemic level.

Cross References

► AACN Practice Guidelines

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Clinical Relevance

► Clinical Significance

Clinical Significance

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Synonyms

Clinical importance; Clinical relevance

Definition

Clinical significance is a perceived, valued, and functionally relevant discrepancy in symptoms/abilities that reflects an important change in functioning. This can involve either an improvement (usually as a result of treatment or intervention) or a decline (typically due progression of illness or disorder) as measured by symptoms or impairment level. The relevance and value of this to difference may be determined by the client, mental health professional, and/or the client's significant other(s).

Clinical significance also refers to a static condition of import – for example, a functionally relevant discrepancy between cognitive abilities in different domains (e.g., language vs. visual-perceptual abilities). The term can also refer to the level of distress or impairment related to psychological symptoms as criteria for a DSM-IV TR disorder diagnosis.

While clinical significance may be supported by statistically significant differences on quantitative measures of functioning, statistical significance cannot be equated with clinical significance.

Cross References

► Functional Assessment
► Premorbid Functioning
► Quality of Life
► Reliable Change Index
► Response to Intervention
► Statistical Significance

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Clinical Target Volume

► Involved Field Radiotherapy

Clock Drawing

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Description

Clock Drawing Test (CDT) is a widely used and popular neuropsychological test. Rubin, Barr, and Burton (2005) reported that the CDT appears in the top 40 tests most commonly used by neuropsychologists. The CDT is often considered to be a visuoconstructional test. Modern versions of the CDT usually contain at least two parts – clock drawing to command and clock drawing to copy. In the command condition, patients are presented with a blank sheet of paper and are asked to "draw the face of a clock showing the numbers and the two hands set for ten after eleven." In the copy condition, a pre-drawn model of a clock with numbers and hands set for 10 after 11 is presented, and the patient is asked to copy the model. Clock drawing with hands set for ten after eleven is an innovation introduced by Edith Kaplan (Kaplan, 1988, 1990).

As described below, other versions of the CDT ask the patient to set the hands for other times. Also, some versions of the CDT include a clock subtest in which patients are presented with pre-drawn clock faces with and without numbers and/or hands and are asked to either draw clock hands to designate specific times or read the specified time (Borod, Goodglass, & Kaplan, 1980; Leach, Kaplan, Reweilak, Richards, & Proulx, 2000; Tuokko, Hadjustavropoulos, Miller, & Beattie, 1992).

Historical Background

Since the late 1980s, a profusion of research using the CDT as an assessment tool for dementia has emerged. However, some of the more interesting historical roots of the CDT came from research with aphasic patients. Henry Head, in his magnum opus *Aphasia and Kindred Disorders of Speech* (1926), assessed aphasic patients using a wide number of drawing tests including "the clock test" (p. 214) to assess deficits in understanding and executing complex propositional speech and deficits associated with symbol formation. In describing propositional speech and symbolic formation deficits associated with aphasia, Head (1926) wrote "any act of mental expression, which demands symbolic formulation, tends to be defective and the higher its propositional value the greater the difficulty it will present" (p. 212). Head (1926) provided many examples of aphasic patients who demonstrated striking impairment in executing the propositional command to set the clock hands for a specified time. For example, Head described an aphasic patient who was able to set clock hands "correctly at 3:40," but not when he was told to place the hands at "20 minutes to 4." For this latter test condition ("20 minutes to 4"), Head commented that his patient appeared "doubtful of the meaning of the words 20 minutes to." Other researchers have used the CDT to assess deficits in symbolic formation in neurologic patients (Mayer-Gross, 1935; McFie & Zangwill, 1960; Van Horst, 1934).

Classically trained neurologists often associate the CDT as a means to assess *constructional apraxia*. Kleist (1912, cited in Benton & Tranel, 1993) described constructional apraxia as deficits in formative activities necessary to assemble parts into a meaningful whole. For Kleist, the essential defect in constructional apraxia was the ineffective translation of visuoperceptual information into an effective motor act. In this sense, the concepts of Kleist are consistent with the constructs Head (1926) used to understand impaired clock drawings produced by aphasic patients. Interestingly, Kleist tended to associate constructional apraxia with lesions in the left posterior cortex. This is in stark contrast to the anecdotal point of view that tends to automatically associate defective clock drawing (or any other impaired figure copying test for that matter) with a right parietal lesion. While it is certainly true that patients with right parietal lesions often produce very spatially impaired clock drawings, it is naive to automatically associate impaired clock drawing with either a lesion in any single brain region, or as a measure of any single cognitive operation. As described below, defective clock drawings to command and copy can be associated with a wide array of neurologic lesions and underlying cognitive disorders.

Many time settings have been used in the CDT. Kaplan (1988, 1990) recommends using "ten after eleven." First, clock setting for ten after eleven requires the patient to disambiguate a complex propositional command. Second, since the numbers "10" and "11" are on the clock,

patients need to resist the temptation to be pulled to the numbers "10" and "11." Thus, clock setting to ten after eleven tends to elicit a variety of stimulus-bound errors. In a survey of neuropsychologists and neurologists described by Freedman, Leach, Kaplan, Shulman, and Delis (1994), other commonly used clock settings include "20 after 8" and "3 o'clock."

Psychometric Data

Some scoring guidelines for the CDT can be found as part of the Boston Diagnostic Aphasia Examination supplementary language tests (BDAE; Goodglass & Kaplan, 1972, 1983; Goodglass, Kaplan, & Baresi, 2001). In the original BDAE (with the *rakishly purple cover* [see Holland's forward to the third edition of the BDAE, 2001]), the CDT test was one of several tests believed to be sensitive to parietal lobe injury. As described in the BDAE corpus, clock drawing is one of six figures where patients are asked to draw to command and copy (i.e., a clock with hands set for 10 after 11, a daisy, an elephant, a red cross, a three-dimensional cube, and a house). For the clock drawing portion of the test, a three-point scoring system was described awarding a point for an approximately circular clock face, symmetry of number placement, and correctness of numbers. No scoring for the representation of the clock hands was suggested. Normative data for the entire figure drawing test (range 0–13 for the separate command and copy test conditions) is provided. Separate normative data for the CDT is not included. Additional normative information is provided by Borod, Goodglass, and Kaplan (1980). In this report, norms are also provided for two additional clock assessment procedures – clock setting with numbers and clock setting without numbers. In both tests, the patient is presented with pre-drawn clock faces with and without numbers and are asked to draw the hands to read 1:00, 3:00, 9:15, and 7:30. Performance is assessed using a 12-point scoring system. Borod and colleagues (1980) described both age and educational effects for this clock assessment procedure. Other researchers have commented on the effects of education on clock drawing test performance (Marcopulos, McLean, & Giuliano, 1997).

The Kaplan-Baycrest Neuropsychological Survey (KBNS; Leach et al., 2000) contains a comprehensive clock test consisting of five parts: (1) clock drawing to command where patients are asked to draw the face of clock put in all the numbers and set the hands for 10 after 11, (2) a clock drawing to copy condition where patients are presented with a blank page and asked to draw a clock with numbers and set the hands for ten after eleven, (3) a pre-drawn clock face where patients are asked to put in all the numbers and set the hands for 20 after 8, (4) a clock reading test with hands, but without numbers where patients are asked to identify a specified time, and (5) a clock reading test with numbers and hands where patients are, again, asked for the specified time. For clock drawing to command and copy, behavior related to the drawing of the clock face, the numbers, and hands, and the representation of the clock hands originating from the center of the clock are each scored using a 13-point scoring system. The pre-drawn clock face condition is scored using an 11-point scoring system. Thus, for this portion of the test scores have a 0–37 point range. Each clock reading subtest contains six test stimuli. Performance on the three clock drawing test conditions are combined with a score measuring the copy of a complex figure for a combined age-corrected scale score. The two clock reading subtests are scored separately (range 0–6). For these two tests, age-related percentile cut scores are provided.

Freedman, Leach, Kaplan, Shulman, and Delis (1994) described a very comprehensive clock scoring system using a variety of clock drawing conditions and clock settings. Normative data was collected and grouped by decade from age 20 to 80+. Separate scales were developed to assess the drawing of the clock face, the drawing of the numbers, the presence and drawing of the clock hands, and the degree to which the clock hands emanated from the center of the clock face. Base rates for a wide range of clock drawing behavior are provided. These data show that certain errors occur more frequently with age. For example, for clock setting using "ten after eleven," the representation of the clock hands tends to be differentially affected by age.

As noted above, a wide number of clock drawing procedures have been reported (Lezak, Howison, & Loring, 2004); however, most researchers follow Kaplan's (1988, 1990) suggestion and ask patients to set the hands for "ten after eleven" (Freedman et al., 1994). It is important to understand that many CDT scoring procedures are essentially atheoretical, that is, the administration and scoring procedures were devised with an eye toward sensitivity to brain damage or neurological insult rather than to assess for deficits involving specific cognitive constructs. Recent research, particularly in using the CDT as part of a dementia evaluation, suggests that the command and copy conditions are related to different underlying cognitive mechanisms (Cosentino, Jefferson, Chute, Kaplan, & Libon, 2004; Libon, Swenson,

Barnoski, & Sands, 1993; Libon, Malamut, Swenson, & Cloud, 1996; Rouleau, Salmon, Butters, Kennedy, & McGuire, 1992). Equally important, different results are obtained depending on test instruction (see Cosentino et al., 2004 for a review). These considerations are critically important when the task at hand is to differentiate between say, dementia subtypes.

Clinical Uses

Focal lesions – Clock drawing has not been extensively studied in non-dementia, focal lesioned patients. Nonetheless, specific patterns of deficits can be associated with specific neurologic lesions. Freedman et al. (1994) and Kaplan (1988, 1990) provide some instructive exemplars. For example, patients with left posterior brain lesion resulting in a Wernicke's aphasia often present with language comprehension deficits. While these patients may demonstrate general understanding of the clock drawing instructions, numbers may be omitted entirely with hatch marks used as substitutes (Freedman et al., 1994). Patients with a left anterior lesion presenting with a Broca's aphasia often have difficulty in understanding functor words such as "to" and "after." These patients, therefore, may be apt to draw the clock hands pointing to the numbers "10" and "11." Further assessment is required to see if this kind of error is caused by a language-related deficit or represents an executive deficit. Patients with left hemisphere lesions might initiate their drawing on the left side of the clock, that is, on the side contralateral to their intact right hemisphere. Thus, numbers may be written correctly but in a counterclockwise direction (Freedman et al., 1994).

Interesting dissociations can be found in clock drawings to command versus copy in focal lesion patients. Kaplan (1990) provides several instructive examples. An analysis of clock drawings produced by a patient with a right parietal lesion demonstrates differential impairment in the *copy* versus the command test conditions. In the copy test condition, many numbers were omitted on the left side of the drawing. The clock drawing to command did not demonstrate this behavior and was generally intact compared to the copy test condition. Kaplan (1988) demonstrated the opposite profile in a patient with a right temporal lesion. Here, there was differential impairment in the *command* condition. For this patient, the clock drawing to copy was generally intact. For the right parietal lesioned patient, the differential impairment in the *copy* condition likely reflected a deficit involving visually mediated neglect of left hemi-space. For the left temporal lobe patient, the visuospatial impairment seen in the *command* condition may be due to a deficit in visuospatial memory. Tranel, Rudrauf, Vianna, and Damasio (2008) administered the CDT to a large group of focal lesioned patients. Imaging studies found errors on the CDT were associated with right parietal (supramarginal gyrus) and left inferior frontal-parietal opercular brain damage. These researchers also noted that visuospatial errors were predominant in patients with right hemisphere damage, whereas time-setting errors were predominant in patients with left hemisphere lesions.

Dementia – As noted above, since the late 1980s, there has been a plethora of research demonstrating the value of the clock drawing test as both a screening test for dementia as well as a means of investigating cognitive constructs that may differentiate between dementia subtypes (see Cosentino et al., 2004 for a review). Rouleau et al. (1992, 1996) examined patients with Alzheimer's disease (VaD) and Huntington's disease (HD) administering a clock drawing test to command and copy with hands set for ten after eleven. An analysis of errors proved effective in differentiating between dementia subtypes. AD patients made more conceptual errors while HD patients produced more graphomotor errors. These authors speculated that semantic knowledge deficits might underlie the deficits produced on the CDT by AD patients whereas executive dysfunction might underlie the errors produced by HD patients. When the command and copy conditions were compared, AD, but not HD patients improved from the command to copy test conditions.

Rouleau's research was the impetus for a series of studies conducted by Libon and colleagues (Cosentino et al., 2004; Libon et al., 1993, 1996) that examined differences on the CDT between patients with AD and vascular dementia (VaD). In their original study, Libon et al. (1993) found no difference in errors between AD and VaD patients in the command condition. However, similar to Rouleau, AD patients improved, that is, made fewer errors than VaD patients in the copy condition. These findings were replicated in a second study (Libon et al., 1996), that is, AD patients generally improved from the command to copy test conditions compared to VaD patients. Cosentino et al. (2004) grouped dementia patients diagnosed clinically with either AD or VaD on the basis of MRI white matter alterations (MRI-WMA). These groups were compared to dementia patients with Parkinson's disease (PD). Patients presenting with minimal to mild MRI-WMA continued to improve from the command to copy test conditions, that is, produce fewer errors in the copy versus the condition test conditions, compared to patients with moderate to severe MRI-WMA and PD patients. Errors produced the copy condition were

correlated with poor performance on executive tests. Errors produced in the command condition were correlated with overall dementia severity and tests related to semantic knowledge. Cahn-Weiner (2003) correlated CDT performance with MRI measures of atrophy and found that impaired CDT performance was attributable to impairment in multiple cognitive domains but was primarily related to volume loss involving the right temporal cortex. Taken as a whole, this research suggests that different cognitive constructs underlie impaired clock drawing in patient with cortical versus subcortical dementia.

Cross References

► Constructional Apraxia

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C-Log

► Cognitive-Log

Clomipramine

John C. Courtney
Children's Hospital of New Orleans
New Orleans, LA, USA

Generic Name

Clomipramine

Brand Name

Anafranil

Class

Tricyclic antidepressant

Proposed Mechanism(s) of Action

Boosts neurotransmitters serotonin and norepinephrine/noradrelaline; blocks serotonin transporter; apparently desensitizes both serotonin 1A receptors and beta adrenergic receptors

Indication

Obsessive-compulsive disorder, depression, cataplexy syndrome

Off Label Use

Anxiety, insomnia, chronic pain

Side Effects

Serious

Paralytic ileus, hyperthermia, lowered seizure threshold and rare seizures, orthostatic hypotension, sudden death, arrhythmias, tachycardia, QTc prolongation, increased intraocular pressure, hepatic failure, extrapyramidal symptoms, mania, and suicidal ideation

Common

Blurred vision, constipation, increased appetite, urinary retention, dry mouth, nausea, diarrhea, heartburn, strange taste in mouth, weight gain, fatigue, weakness, dizziness, headache, anxiety, nervousness, restlessness, sedation, sexual dysfunction, sweating

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

Clonazepam

John C. Courtney
Children's Hospital of New Orleans
New Orleans, LA, USA

Generic Name

Clonazepam

Brand Name

Klonopin

Class

Anxiolytic

Proposed Mechanism(s) of Action

Binds to benzodiazepine receptors at the GABA-A ligand-gated channel, thus allowing for neuronal hyperpolarization. Benzodiazepines enhance the inhibitory action of GABA via boosted chloride conductance.

Indication

Panic disorders (with or without agoraphobia), Lennox–Gastaut syndrome, akinetic seizures, myoclonic seizure, absence seizures

Off Label Use

Atonic seizures and other anxiety disorders, acute psychosis, insomnia

Side Effects

Serious

Respiratory depression, hepatic dysfunction (rare), renal dysfunction and blood dyscrasias, Grand mal seizures

Common

Sedation, fatigue, depression, dizziness, memory problems, dysinhibition, confusion, ataxia, slurred speech

References and Readings

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

Clonidine

John C. Courtney
Children's Hospital of New Orleans
New Orleans, LA, USA

Generic Name

Clonidine

Brand Name

Duraclon, Catapres, Catapres-TTS, Clorpres

Class

Antihypertensive

Proposed Mechanism(s) of Action

Centrally acting alpha 2 agonist

Indication

Hypertension

Off Label Use

Attention deficit hyperactivity disorder, Tourette's syndrome, anxiety disorders including PTSD and social anxiety disorder, substance withdrawal including opiates and alcohol, menopausal flushing, clonadine-induced hypersalavation, severe pain in cancer patients

Side Effects

Serious

Sinus bradycardia, atrioventricular block during withdrawal, hypertensive, encephalopathy, cerebrovascular accidents, and death

Common

Dry mouth, dizziness, constipation, sedation, major depression, weakness, fatigue, impotence, loss of libido, insomnia, headache, dermatologic reactions, hypotension, occasional syncope, nervousness, agitation, tachycardia, nausea, vomiting

References and Readings

Physicians' Desk Reference (62nd ed.). (2007). Montvale, NJ: Thomson PDR.
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Pill Identification: http://www.drugs.com/pill_identification.html

Clorazepate

John C. Courtney
Children's Hospital of New Orleans
New Orleans, LA, USA

Generic Name

Clorazepate

Brand Name

Azene, Tranxene

Class

Anxiolytic

Proposed Mechanism(s) of Action

Binds to benzodiazepine receptors at the GABA-A ligand-gated channel, thus allowing for neuronal hyperpolarization. Benzodiazepines enhance the inhibitory action of GABA via boosted chloride conductance. Clorazepate is also hypothesized to inhibit neuronal activity in amygdala-centered fear circuits.

Indication

Anxiety Disorder, symptoms of anxiety, and acute alcohol withdrawal.

Off Label Use

Partial seizures (as an adjunct).

Side Effects

Serious

Respiratory depression, hepatic dysfunction (rare), renal dysfunction and blood dyscrasias, and Grand mal seizures.

Common

Sedation, fatigue, depression, dizziness, memory problems, dysinhibition, confusion, ataxia, and slurred speech.

References and Readings

Physicians' Desk Reference (62nd ed.). (2007). Montvale, NJ: Thomson PDR.
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Pill Identification: http://www.drugs.com/pill_identification.html

Closure

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Synonyms

Visual integration; Visual synthesis

Definition

Visual closure refers to the ability to perceive and recognize objects, shapers, features, or symbols from incomplete or degraded visual stimuli. It reflects the capacity of humans to fill in missing information from incomplete sensory input to achieve a meaningful percept.

Historical Perspective

The principle of visual closure had its roots in Gestalt psychology (Ellis, 1938; Harlow, 1938; Köhler, 1929). Gestalt psychology theorized that operationally brain

functions (i.e., perception and cognition) are holistic consisting of analog processes that occur in a parallel manner and are self-organizing. This led to the well-known conclusion regarding perception, and cognition more generally, that "the whole is greater than the sum of its parts." Based on this framework, all perceptual processes act to achieve optimal organization and reconciliation with the objects that are being perceived. A critical principle driving Gestalt perception is prägnanz (law of conciseness), which maintains that people organize their experience in an orderly, symmetric, and simple manner when possible. Visual closure was one of the five laws of prägnanz, with others including the laws of similarity, proximity, symmetry continuity, and common fate.

While each of these laws has potential value in accounting for elements of visual integration, the law of visual closure seems to have had the most direct impact, particularly with respect to clinical neuropsychology. Early clinical studies of the effects of posterior cortical lesions on visual perception indicated that certain patients had difficulty in simultaneously processing all the elements of their visual sensorium to achieve a unified percept, a syndrome that was labeled simultagnosia (Poppelreuter, 1990).

Current Knowledge

The idea that visual perception occurs as a function by-product of active self-organizing processes is now widely accepted by most visual scientists, though many would reject a pure holistic view. Instead, visual perception and higher-order visual processes tend to be conceptualized as the by-product of computational processes carried out by modular neural networks responsible for specific operations. Visual closure is thought to result from such processes occurring in extra-striatal systems found primarily in the parietal cortex. Psychometric studies of closure have tended to employ tests such as the Gollin Figures and Mooney test (Foreman, 1991; Holmes, 1968; Jones & Dennis, 1972; Mooney & Ferguson, 1951). In healthy adults, the ability to recognize line drawings that have been degraded has been shown to be a function of the size of gaps in the drawing (Jones & Dennis, 1972), which in turn reflects the amount of missing information. Performance on closure tests has been shown to not be strongly associated with visual search performance, suggesting that these are distinct visual processes (Foreman, 1991). While tests of closure have existed for over 40 years, there are relatively few studies demonstrating consistent impairments on tests such as the Gollin Figures and Mooney Closure tests. This may reflect the fact that impairments on this type of task tend to be embedded in other visual perception deficits that are more striking. However, it is also clear that closure paradigms have not been systematically implemented into standard neuropsychological batteries using modern computerized methods, so that definitive conclusions regarding impairments of closure secondary to localized and global brain disorders cannot be reached at this point.

Cross References

- ▶ Gollin Figures
- ▶ Hooper Visual Organization Test
- ▶ Simultanagnosia

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Clot Buster

- ▶ Recombinant Tissue Plasminogen Activator

Clot Busting

- ▶ Thrombolysis

Clotting

► Thrombosis

Clozapine

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Metarie, LA, USA

Generic Name

Clozapine

Brand Name

Clozaril, Leponex

Class

Atypical Neuroleptic

Proposed Mechanism(s) of Action

Blocks dopamine 2 receptors, inhibits serotonin 2A receptors, thus increasing presynaptic release of related catecholamines

Indication

Schizophrenia (treatment-resistant), reduction of suicidal behavior.

Off Label Use

Bipolar disorder (treatment-resistant), violence, and aggression associated with psychosis or brain dysfunction.

Side Effects

Serious

Agranulocytosis, neuroleptic malignant syndrome, seizures, pulmonary embolism, myocarditis, hyperglycemia.

Common

Increased risk for diabetes, sweating, and increased salivation.

References and Readings

Physicians' Desk Reference (62nd ed.). (2007). Montvale, NJ: Thomson PDR.
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Pill identification: http://www.drugs.com/pill_identification.html

Clumsiness

► Ataxia

Cluster Analysis

► Clustering

Clustering

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Synonyms

Cluster analysis

Definition

Clustering can be thought of as the obverse of factoring. In factor analysis, observations are correlated with each other and the correlation matrix is examined to see which items covary among themselves. In cluster analysis, the correlation matrix is examined to see which individuals or observations covary among themselves. Once the groups of individuals are composed, they are compared to each other in order to see if some external correlate exists.

For example, a large group of patients might be administered a series of cognitive measures. The scores on those measures are examined to see which individuals seem to be most similar to each other. Once the groups are empirically formed, their characteristics are examined to see if age or diagnosis or injury severity discriminates among the groups.

Cross References

► Correlation Coefficients
► Factor Analysis

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Cmax

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Synonyms

Maximum concentration; Peak concentration

Definition

Plasma concentrations reflect a time curve from the administration of a drug through its peak effect and eventual elimination. The maximum concentration of a drug in blood plasma represents a drug's peak effect. Cmax is one of the primary pharmacokinetic measures for evaluating how the body acts upon a drug.

The same dose of a drug may result on different plasma levels because the body is not a passive recipient. Plasma levels of a drug can be altered by the route of administration, the ease of absorption, the distribution of the drug with the body, the bioavailability or accessible concentration of the drug, and the efficiency with which a drug is metabolized or eliminated.

Common factors that affect plasma levels of a drug could include the size of the molecule and its fat solubility; the patient's gastric pH, physical health or age, and the presence of other medications or foods that may expedite or slow absorption from the gastrointestinal tract into general circulation or alter the drug's metabolism and excretion. These factors may require changes in dosing in order to achieve therapeutic levels of a drug or alterations in diet, such as to take a drug with or without food. It may also include recommendations to avoid particular foods that are known to induce or inhibit the specific enzyme pathways that metabolize the drug (e.g., grapefruit juice). Drug–drug interactions can alter the plasma levels of a drug, including its peak effect.

There are also mediations that inhibit metabolism within a particular liver-mediated enzyme system, thus altering the availability of other agents of that drug itself. This can alter the availability of a drug, including its Cmax. Fluoxetine's impact on codeine is a good example. Codeine is ultimately synthesized into morphine and via a transformation that requires an enzyme that makes this change possible. Fluoxetine appears to inhibit this process, thus dramatically reducing the pain control intended via the administration of codeine.

For some drugs, there is no clear relationship between the concentration of a drug in blood plasma and its pharmacological effect. For other drugs, the concentration must be tightly monitored, typically through regular plasma assessments of drug levels (assays). As a rule of thumb, adverse drug reactions and side effects become more likely as the dose of a drug increases. Consequently, adverse drug effects are more likely at the time of Cmax.

Cross References

► p450 Cytochrome System
► Pharmacokinetics
► Side Effects

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CMS

► Children's Memory Scale

CNC

► Coma/Near Coma Scale

CNS Lupus

► Lupus Cerebritis

Cochlea

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Definition

The cochlea, a small conical structure, is the part of the inner ear that converts mechanical energy (vibrations) into nerve impulses sent to the brain. It is also known as the organ of hearing. The word *cochlea* is a Latin word derived from the Greek *kokhlos*, which refers to the land snail. A coiled tube, the cochlea winds around a central axis, forming the anterior part of the labyrinth. It contains the organ of Corti, which includes the hair cells that constitute the primary mechanisms by which pressure waves in the cochlea are transduced into bioelectrical nerve impulses. The acoustic division of the eighth cranial nerve has its cell bodies in the spiral ganglion of the cochlea.

Cross References

► Auditory System

References and Readings

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Cochlear Nerve

► Vestibulocochlear Nerve

Cochlear Nuclei (Dorsal and Ventral)

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Definition

Cochlear nuclei is the nuclei that receive first-order auditory input from the organ of Corti in the cochlea of the inner ear.

Current Knowledge

The cochlear nuclei are divided into a dorsal and a ventral group. The dorsal cochlear nuclei give rise to the dorsal acoustic stria, which immediately cross the midline and contribute fibers that ascend contralaterally

in the lateral lemniscus. The ventral cochlear nuclei are the source of two other auditory pathways, the intermediate and the ventral acoustic stria. The former also crosses the midline and, like the dorsal acoustic stria, ascends in the contralateral lateral lemniscus. Fibers making up the ventral acoustic stria, the largest of these three pathways, take three different paths after leaving the nucleus. They (1) synapse in both the ipsilateral or contralateral superior olivary nuclei, which in turn send tertiary fibers to the inferior colliculi via the ipsilateral and contralateral lateral lemniscus, and (2) send fibers directly to the contralateral inferior colliculi, bypassing the superior olivary nuclei.

The crossing fibers of the ventral acoustic stria make up what is known as the **trapezoid body** of the pontine tegmentum. The dorsal and ventral cochlear nuclei themselves are located laterally in the rostral medulla at the pontine–medullary junction near the vestibulocochlear nerve.

Since the first-order neurons from the auditory nerve terminate in the cochlear nuclei and the decussations within the auditory system only begin with the second-order neurons originating in the cochlear nuclei, these nuclei receive input only from one ear. Hence, unilateral pontine lesions affecting the dorsal and ventral cochlear nuclei would be expected to result in ipsilateral hearing deficits (loss).

Cross References

► Auditory System

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Coding

► Digit Symbol Substitution Test

Cog-Log

► Cognitive-Log

Cogniform Disorder

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Synonyms

Conversion disorder; Malingering; Somatoform disorders

Definition

In neuropsychology, the assessment of test-taking effort has captured the focus of considerable research and debate. In the past 20 years, over 500 studies have been published in peer-reviewed neuropsychological journals that address the breadth and scope of this problem (see reviews by Hom & Denny, 2002; Iverson & Binder, 2000; Larrabee, 2005; Sweet, 1999). Considerable advances have been made in the development of empirically based methods for identifying individuals who are simulating cognitive problems, including the use of instruments designed specifically to assess cognitive validity (Binder, 1993; Frederick, 1997; Green et al., 1999; Tombaugh, 1996), analysis of atypical performances on standard ability tests (Larrabee, 2003; Millis et al., 1995), and analysis of test–retest profile inconsistencies (Hom & Denny, 2002; Iverson & Binder, 2000). In addition, specific guidelines and criteria have been developed for diagnosing suboptimal effort and malingering on neuropsychological tests (e.g., Slick et al., 1999). Using these methods and guidelines, neuropsychologists have found that the frequency of individuals exhibiting excessive or exaggerated cognitive symptoms in medicolegal evaluations often ranges from 20% to 40% (Miller, 2001; Millis et al., 1995; Mittenberg et al., 2002). In light of the pervasiveness of this problem, it is now generally accepted that cognitive validity testing is an important part of the neuropsychological assessment process, particularly for evaluations that occur in the context of medicolegal or disability-application settings (Bush et al., 2005).

While considerable advances have been made in the methods used to detect individuals who are exhibiting inadequate effort and symptom exaggeration on cognitive testing, neuropsychologists often find themselves in a quandary in terms of the diagnostic labels to ascribe to these individuals once they have been identified. The DSM-IV

offers several possible categories for diagnosing individuals with excessive cognitive symptoms (e.g., Malingering and Conversion Disorder); however, shortcomings of these conditions have been noted in the literature. In particular, Malingering has been the subject of considerable debate and criticism, especially with regards to the objectivity with which clinicians can assess if feigned symptoms were intentionally or unintentionally produced. Improvements have been made in establishing criteria for this condition (e.g., Slick et al., 1999), but clinicians nevertheless often remain reluctant to use any diagnosis that requires them to make judgments about intentionality of symptom exaggeration.

Clinicians often face three general problems in trying to use existing DSM-IV categories to classify individuals with excessive cognitive symptoms. These problems include (a) lack of a diagnostic category that adequately targets the specific features of this relatively common condition, (b) the use of criteria that *require* the clinician to make judgments about internal states that are exceedingly difficult to evaluate in an objective manner (e.g., intentional versus unintentional production of excessive symptoms), and (c) difficulties in determining the relative role that external incentive and sick-role factors may play in the symptom production.

Symptom Specificity. The existing DSM-IV categories addressing excessive symptomatology can be divided into two general types: symptom-specific versus symptom-nonspecific conditions. Symptom-specific conditions are those that require amplification of only certain types of symptoms. The DSM-IV offers a relatively small number of *symptom-specific* categories, which fall only among the somatoform disorders (e.g., Somatization Disorder and Conversion Disorder) and dissociative disorders (e.g., Dissociative Amnesia and Dissociative Fugue). In addition, the DSM-IV offers two *symptom-nonspecific* conditions, Malingering and Factitious Disorder, which are discussed in the next section.

A major problem in trying to subsume individuals with excessive cognitive complaints or invalid test performances into one of the *symptom-specific* diagnoses is that the cognitive symptoms of many of these cases simply fail to fit adequately in these categories. Following are explanations of this problem for each of the symptom-specific categories provided in the DSM-IV:

(a) **Somatization Disorder** requires at least four pain symptoms, two gastrointestinal symptoms, one sexual symptom, and one pseudoneurological symptom. However, many individuals who present with primarily excessive cognitive symptoms have few if any physical complaints (Larrabee, 2005).

(b) **Undifferentiated Somatoform Disorder** requires "one or more physical complaints," with no reference made to cognitive difficulties.

(c) **Conversion Disorder** requires "one or more symptoms or deficits affecting voluntary *motor* or *sensory* function" [emphasis added], without mention of cognitive or memory difficulties among the specific criteria.

(d) **Pain Disorder** requires only excessive pain symptoms.

(e) **Somatoform Disorder NOS** could conceivably include individuals with predominantly excessive cognitive symptoms; however, "soma" denotes physical rather than cognitive problems, and the list of example cases provided in the DSM-IV for this catchall category makes no reference to excessive cognitive symptoms.

(f) **Dissociative Amnesia** requires one *specific* type of cognitive problem, namely, "an inability to recall important *personal* information, usually of a traumatic or stressful nature" [emphasis added]. However, individuals presenting with excessive cognitive symptoms do so in a myriad of ways (Bush et al., 2005; Delis & Jacobson, 2000; Larrabee, 2003). Some people endorse problems in all cognitive domains queried, including attention, language, math, visual-spatial functions, higher-level executive functions, new learning and memory, and remote recall of important personal information. In contrast, other individuals endorse difficulties in only one or a few specific cognitive skills (e.g., short-term memory and concentration), while denying problems in other cognitive domains, including recall of important personal information. In fact, cases of isolated difficulty in remembering important autobiographical information are relatively rare, illustrating the limited utility of this diagnostic category for the vast majority of cases with excessive cognitive symptoms.

(g) **Dissociative Fugue** not only requires one specific cognitive difficulty ("inability to recall some or all of one's past"), but carries the added stipulation that this difficulty must surface in the context of a "sudden, unexpected, *travel* away from home or one's customary place of daily activities" [emphasis added]. These cases are extremely rare among individuals presenting with excessive cognitive symptoms, thereby precluding the use of this category for almost all cases.

(h) **Dissociative Identity Disorder** is thought to occur in individuals with multiple personalities in which they exhibit an inability to recall important

information about one or more personality states when they are in a different personality state. However, cases of multiple personalities are relatively rare, particularly in clinical–neuropsychological practice, and thus this diagnosis is seldom applicable to individuals with excessive cognitive symptoms.

(i) **Dissociative Disorder NOS** is another catchall category that, conceivably, could encompass individuals with excessive cognitive complaints. However, the tenor of this category is for individuals who exhibit an inability to recall personal information that was of a traumatic or stressful nature, thereby greatly limiting the utility of this category for most cases of excessive cognitive symptoms.

Taken together, the aforementioned nine symptom-specific categories either fail to include cognitive complaints, target only highly specific, relatively rare types of cognitive problems, or require that other, qualitatively different symptoms or conditions also be present (e.g., extensive physical symptoms for Somatization Disorder). For these reasons, these diagnoses generally fail to capture the vast majority of individuals presenting with excessive cognitive symptoms.

Intentionality. Another difficulty in using existing DSM-IV categories has to do with *required* criteria related to intentional/unintentional or voluntary/involuntary control over the production of the excessive complaints or symptoms. For example, a key required criterion for the two symptom-nonspecific categories – Malingering and Factitious Disorder – is that the clinician must determine if the excessive symptoms were generated in an intentional or volitional manner. The problem here is that this criterion reflects a *causative internal state that, for the majority of cases, is difficult if not impossible to assess in an objective manner.* That is, the degree to which a person may be exhibiting excessive symptoms or behaviors in an intentional, voluntary, or conscious manner versus an unintentional, involuntary, or unconscious manner represents an untestable diagnostic hypothesis for many cases (see also Slick et al., 1999). A clinician may have a "hunch" about whether an individual's excessive complaints or symptoms were under the voluntary or involuntary control of the person, but usually these impressions are not substantiated by objective data, such as a disclosure or confession made by the individual to a clinician or other uninvolved, reliable third party.

Another difficulty in this area of diagnosis is that intentionality is likely multifactorial in nature. For example, there may be at least two key components of intentionality that can be dissociated: conscious awareness and goal-directed motivation. An individual may be both *conscious* of producing feigned behavior (e.g., is capable of admitting to self and others that he or she is simulating symptoms) and *motivated* to do so for some type of personal gain; these features would meet criteria for a DSM-IV diagnosis of Malingering. However, someone may be largely unconscious of the feigned behavior (e.g., has convinced himself or herself that the excessive symptoms are real), yet the feigned behavior may still arise due to a specific, goal-directed purpose. For example, it was noted during World War II that some soldiers, when faced with the prospect of entering the frontlines of battle, would develop psychogenic paralysis (what would now be diagnosed as Conversion Disorder given that the symptom amplification occurred primarily in the motor domain). These individuals often appeared to truly believe they were paralyzed, thereby suggesting an unconscious (conversion) process. However, their exaggerated behavior (paralysis) was clearly goal-directed, because it was manifested in the context of an external incentive (avoidance of danger). In these cases, the *conscious component* of intentionality may have been absent, but the *goal-directed motivational component* for producing the symptom was likely present.

In neuropsychological practice, the same type of dissociation may occur in which individuals may produce excessive cognitive symptoms in reaction to an external incentive (e.g., litigation), thereby suggesting goal-directed motivation for the symptom production. However, these individuals may have nevertheless convinced themselves that their symptoms are real, thereby suggesting a lack of a conscious component to the symptom production. Thus, for these individuals, only certain components of intentionality may be present, with the lack of conscious awareness calling into question whether they would adequately meet the required criteria for a diagnosis of Malingering. Another complicating factor in the assessment of intentionality is that conscious awareness likely exists on a continuum, with individuals varying from being fully conscious, to semiconscious, to largely unconscious of the production of the feigned behavior. Although an operational definition of intentionality is beyond the scope of this chapter, the important point here is that intentionality of symptom production not only refers to an elusive internal state, but it likely has component features that exist on a continuum (e.g., levels of conscious awareness), thereby making this construct exceedingly difficult for clinicians to assess in an objective manner. Consequently, many clinicians are reluctant to use diagnoses such as Malingering, Factitious Disorder, and Conversion Disorder at least in part because of difficulty in objectively assessing the presence or absence

of intentionality in the generation of the excessive symptom.

External Incentive. A third difficulty in using existing DSM-IV categories to diagnose individuals with excessive cognitive symptoms is related to another *required* criterion for the two symptom-nonspecific categories – Malingering and Factitious Disorder – regarding the presence or absence of *external incentive* in the production of the symptoms. Specifically, external incentive is a *required inclusionary* criterion for Malingering and *required exclusionary* criterion for Factitious Disorder. (If there is an absence of external incentive, then the clinician must make a further determination of whether or not an individual has adopted the sick role in order to diagnose Factitious Disorders). However, the criterion of *external incentive* carries its own inherent difficulties for clinicians to identify when considering these diagnoses. First, for many cases, practitioners may not have access to sufficient background information about a person's life to be able to assess if external incentives are operative in the case. That is, a practitioner may be unaware that a patient has or is planning to apply for disability or to initiate a civil lawsuit in the future, or has committed a crime and fears that he or she may soon be apprehended. This lack of knowledge about possible covert sources of external incentives makes it difficult to utilize the diagnoses of Malingering or Factitious Disorder for a number of cases, especially given that such information is a *required criterion* rather than an optional one for these categories.

Second, as currently written, the DSM-IV criteria do not allow for the possibility that a comorbidity may occur between the adoption of the sick role and the presence of external incentives (see also Slick et al., 1999). For example, some individuals may gradually develop into a progressively worsening sick role without the presence of external incentives. However, after a period of time, these individuals may present as so "disabled" that they begin to receive disability payments, without necessarily having actively sought out such compensation. The financial gain, however, likely buttresses and propagates the continuation of the sick role. According to the DSM-IV, these individuals would have started out as having Factitious Disorder, but as soon as the external incentive was initiated and became a reinforcing factor, the diagnosis of Factitious Disorder would be called into question (again, because external incentive is a *required* exclusionary criterion for this condition). However, for these cases, the predominant causative factor for the excessive symptomatology may still be the adoption of the sick role, with the external incentive playing a secondary or supportive role in the continuation of the symptoms. As another example, some individuals may begin to feign symptoms intentionally and consciously in reaction to an external incentive (e.g., a lawsuit). However, these individuals may gradually, and perhaps unconsciously, assume a progressively worsening sick role due to (a) a prolongation in obtaining the external incentive (e.g., caused by delays in the lawsuit); and (b) increased skepticism and questioning on the part of family members, coworkers, or health providers about the authenticity of the individual's complaints. This prolonged scrutiny may be overwhelming to these individuals, compelling them to adopt the sick role and exhibit illness behavior in widespread areas of their lives, to the point where they may even convince themselves of the authenticity of their symptoms. In other words, while the DSM-IV treats *external incentive* and *sick role* as mutually exclusive diagnostic criteria for differentiating Malingering and Factitious Disorder, in reality, as is the case for most psychiatric conditions, they may co-occur in varying degrees (Slick et al., 1999).

Given these limitations in the DSM-IV, the following two diagnostic categories were proposed by Delis and Wetter (2007) to encompass cases of excessive cognitive complaints or poor (invalid) test performances in the absence of sufficient evidence of intentionality of symptom production to warrant a diagnosis of Malingering.

Neuropsychology of Cogniform Disorder

The essential feature of Cogniform Disorder is a pattern of cognitive complaints or low scores on psychometric cognitive tests that are considered to be excessive because they cannot be fully explained by a neurological disorder, by another mental disorder that is associated with CNS dysfunction (e.g., schizophrenia), by a general medical condition known to affect CNS function (e.g., renal disease), by the direct effects of a substance (e.g., opioid medications), or by other factors known to affect cognitive functioning (e.g., developmental learning disorder, insomnia, and normal aging process). If the cognitive complaints or poor test performances occur in the presence of a known neurological or mental disorder or any other factor known to affect CNS function (e.g., medication), the cognitive symptoms are in excess of what would be expected from the history, physical examination, laboratory tests, or psychometric validity testing. Findings from the clinical interview or psychometric testing of cognitive functions do not substantiate the degree of cognitive complaints or symptoms because of the presence of at least two of the following features:

(a) Cognitive complaints or poor test performances that are rare for patients with documented mild to moderate generalized brain damage (e.g., loss of remote autobiographical memories and inability to perform overlearned verbal skills like reading, spelling, or simple math)
(b) Inconsistencies between the individual's excessive cognitive complaints or poor test performances and the relatively mild nature of the injury or illness as documented in the medical records
(c) Inconsistencies between the individual's excessive cognitive complaints or poor test performances and observed behavior
(d) Delayed onset of excessive cognitive complaints or symptoms after an injury and/or significant worsening of symptoms over time without an adequate explanation for the decline in functioning (e.g., subsequent neurological complications)
(e) Significant inconsistencies in cognitive test scores or profiles across repeat evaluations
(f) Patterns of cognitive test scores within an examination that are rare for brain-damaged patients
(g) Significant inconsistencies in cognitive complaints or symptoms over time
(h) Evidence of insufficient test-taking effort or exaggeration on tests designed specifically to assess validity of cognitive performance
(j) Evidence of insufficient test-taking effort or exaggeration on specific measures obtained from standard ability tests that have been empirically found to assess validity of cognitive performance

Considerable individual differences are found in the performances of people with this condition on psychometric tests of cognitive skills (Larrabee, 2003; Slick et al., 1999). Some individuals obtain markedly low scores on most cognitive tests administered; these individuals are often less sophisticated about medical and psychological conditions and more blatant in their symptom amplification. Other people may obtain low and invalid scores on only a few tests administered (e.g., memory tasks); these individuals may be more subtle in their symptom exaggeration and, as a result, more difficult to identify. Occasionally, an individual may perform within expected ranges on most cognitive tests administered, including cognitive validity tests, and yet continue to complain of extensive cognitive problems and dysfunction in their daily lives. These individuals may have learned from other sources (e.g., Internet; attorney coaching) that neuropsychological tests are capable of detecting poor test-taking effort, and consequently exert adequate effort on psychometric tests despite reporting significant cognitive complaints and dysfunction in their daily lives.

The primary distinguishing feature between Cogniform Disorder and Cogniform Condition (see below) concerns the degree to which the individual presents as cognitively impaired in widespread areas of his or her life. Specifically, a diagnosis of Cogniform Disorder should be made if there is reasonable evidence that the individual exhibits excessive cognitive symptoms in most if not all areas of his or her life and seemingly at all times, thereby suggesting a conversion-like adoption of the sick role manifested primarily as cognitive dysfunction. In addition, in Cogniform Disorder, the degree of claimed disability in performing activities of daily living will often parallel the individual's complaints of cognitive dysfunction and poor (invalid) cognitive test performance. For example, the individual not only obtains severely deficient (and likely invalid) scores on tests of visual-motor and visual-spatial functioning, but he or she also ceases to drive a vehicle because of the perceived cognitive problems. In many ways, Cogniform Disorder is analogous to the somatoform condition of Conversion Disorder, but with the excessive symptoms manifested primarily in terms of cognitive dysfunction rather than deficits affecting primarily motor or sensory functions (e.g., nonepileptic seizures). For this reason, Cogniform Disorder should be considered as a new subtype of the somatoform disorders.

Neuropsychology of Cogniform Condition

The essential features of Cogniform Condition are the same as those of Cogniform Disorder in every respect, with the exception of the degree to which the individual exhibits cognitive dysfunction in widespread areas of his or her everyday life. That is, in Cogniform Condition, there is (a) a lack of reasonable evidence that the individual presents as cognitively dysfunctional in many areas of his or her life, and (b) evidence of significant inconsistencies between the individual's excessive cognitive complaints or poor test performances in an evaluation and his or her higher level of everyday functioning. For example, an individual may obtain severely deficient (and likely invalid) scores on tests of visual-motor and visual-spatial functioning and yet continues to drive a vehicle without apparent difficulty. In other words, in Cogniform Condition, the individual is not given a diagnosis of "disorder," because there is a lack of reasonable evidence that the individual is acting out the "sick role" of being cognitively dysfunctional in widespread areas of his or her life despite presenting to the clinician in a manner that suggests that

he or she should be markedly impaired in everyday functioning.

Cogniform Disorder and Condition Versus Malingering: Similarities and Differences

Cogniform Disorder, Cogniform Condition, and Malingering (when manifested in the form of cognitive dysfunction) are similar in that the individual may present with excessive cognitive complaints or exhibit evidence of inadequate effort and exaggeration on formal neuropsychological testing. However, a diagnosis of Cogniform Disorder or Cogniform Condition should *not* be made if there is reasonable evidence that the excessive cognitive symptoms are produced in an intentional or volitional manner, in which case a diagnosis of Malingering may be warranted. As noted above, however, this determination can be difficult to make for many cases due to inherent problems in objectively assessing the internal state of the intentionality of simulated behavior. For this reason, it is likely that many cases of excessive cognitive symptoms would receive the more neutral diagnosis of Cogniform Condition, and possibly a diagnosis of Cogniform Disorder if the individual exhibits cognitively dysfunctional behavior in widespread areas of his or her life. However, when evidence emerges that implicates at least a conscious component in the production of the excessive cognitive symptoms, then a diagnosis of Malingering (or Malingered Neuropsychological Dysfunction; Slick et al., 1999) may be warranted. Following are different examples of evidence that can be supportive of a diagnosis of Malingering:

(a) On psychometric testing, an individual obtains an accuracy score on a forced-choice recognition memory test that falls significantly below a chance level. Such a score provides empirical evidence that the individual correctly remembered the right answers above a chance level and used this knowledge to frequently select the wrong answer (Larrabee, 2003; Millis, 1992).
(b) A person who is involved in two separate personal-injury lawsuits for different accidents complains of one set of symptoms and injuries to doctors associated with one lawsuit and different symptoms and injuries to other doctors associated with the second lawsuit. Such selective reporting of symptoms that correspond to the different lawsuits suggests a conscious component to the symptom amplification.
(c) An individual "confesses" to intentionally performing poorly when taking cognitive tests.

As proposed here, *a diagnosis of Cogniform Disorder or Cogniform Condition does not exclude the possibility of intentional production of the excessive symptoms; rather, these categories imply only that there is insufficient evidence at the time of the assessment to formulate a diagnosis of intentionality and therefore Malingering.* Indeed, an advantage of having diagnostic categories such as Cogniform Disorder and Cogniform Condition is that they allow the clinician to label the cognitive symptoms as excessive using more neutral terms that avoid the accusatory implications of Malingering when there is a lack of clear evidence to make that diagnosis. In addition, as discussed above, intentionality is likely multifactorial in nature and is comprised of at least two key components: conscious awareness and goal-directed motivation. The individual who has convinced himself or herself that the feigned behavior is real may not be fully or even partially conscious of his or her symptom amplification, but this person may nevertheless have developed the symptoms in reaction to the presence of external or interpersonal incentives for personal gain. The categories Cogniform Disorder and Cogniform Condition allow the clinician to acknowledge the presence of incentives that may have played a significant role in the goal-directed motivation for the excessive symptomatology without having to make the difficult determination of whether the individual is conscious or unconscious of these dynamics.

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Cognistat

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Synonyms

Neurobehavioral cognitive status examination (NCSE)

Description

The Cognistat test battery (Kiernan, Mueller, & Langston, 1995), formerly called the Neurobehavioral Cognitive Status Examination, is a screening tool designed to assess a number of different cognitive domains including Orientation, Attention, Language Abilities (Comprehension, Repetition, Naming), Construction, Memory, Calculations, and Reasoning (Similarities, Judgment). A rating for Level of Consciousness is also provided. The various subscales are modeled after more extensive and well-validated neuropsychological tests but in an abbreviated from. For example, Attention is assessed using digit repetition similar to that used on the Wechsler scales. Unlike other screening procedures that yield a single summary score, Cognistat is designed to yield a score for each domain and thus produce a differentiated profile of cognitive abilities. Cognistat also employs an adaptive testing approach (referred to as a screen and metric approach) to decrease the time spent in administration. In this approach, an item that is of average difficulty is first administered for each subtest. If that item is passed, no other items are administered from that subtest, but if it is failed, additional easier items are administered. The raw scores for each subscale are then plotted on a standard profile form, and performance is classified as being in the average range or as indicative of mild, moderate, or severe impairment.

Current Knowledge

Because extensive validity and normative information were not available when Cognistat was originally published, a number of studies have subsequently examined its sensitivity to brain damage as well as the influence of demographic variables on test performance. Research revealed that the Cognistat may be more sensitive to brain damage than the Mini Mental State Exam, Cognitive Capacity Screening Examination, and Mattis Dementia Rating Scale (e.g., Drane et al., 2003). Cognistat has also been found to be sensitive to a variety of neurological and psychiatric disorders, and to age-related changes in cognitive abilities (for brief review see Doninger, Ehde, Bode, Knight, Bombardier, & Heinemann, 2006). Correlations between its various subtests and neuropsychological measures of similar abilities provide evidence for its construct validity (Nabors, Millis, & Rosenthal, 1997).

The Cognistat battery also has limitations. For example, performance is influenced by demographic variables, including age and education. Although the original validation study of the Cognistat demonstrated no differences in performance between age groups (Kiernan, Mueller, Langston, & Van Dyke, 1987), subsequent investigations found that increased age is associated with poorer performance on the Construction, Memory, Similarities, Attention, and Calculation domains, with Construction and Memory appearing to be the most consistently impacted by age (Drane & Osato, 1997). Additionally, years of education and low levels of educational attainment are

associated with diminished performance (Macauly et al., 2003; Ruchinskas et al., 2001). With only limited normative data available to correct for these influences (Drane et al., 2003), interpretation of performance in the elderly and those with limited education must be tentative. Finally, a recent study of community dwelling and hospitalized individuals with traumatic brain injury suggests that the Cognistat should not be used to profile neurocognitive strengths and weaknesses because measurement error accounted for the majority of variance in subtest scores (Doninger et al., 2006). Thus, the Cognistat provides a useful method to screen patients with a variety of neurological and psychiatric disorders for neurocognitive impairment, although additional research appears warranted before that it can be used to make inferences regarding impairment of discrete cognitive abilities, and more extensive normative data is needed.

Cross References

► Mattis Dementia Rating Scale (DRS)
► Mini Mental State Exam (MMSE)

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Cognition

► Cognitive Functioning

Cognitive Affective Syndrome

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Synonyms

Cerebellar cognitive affective syndrome

Definition

First described by Schmahmann and Sherman (1997), cerebellar cognitive affective syndrome (CAS) refers to a cluster of impairments involving higher-order cognitive processes and affective functioning. Symptoms tend to cluster in executive dysfunction, including problems with planning, set shifting, verbal fluency, abstract reasoning, perseveration, attentional dysregulation, hyperactivity, impulsivity and disinhibition, and deficits in working memory. However, symptoms may also include visuospatial disorders, expressive language disorders, affective abnormalities, difficulties with visuospatial organization, visual memory, logical sequencing, and blunted or inappropriate affect (Schmahmann & Sherman, 1997).

Current Knowledge

Causes and Correlates of CAS

The co-occurrence of these cognitive and affective symptoms arises from the disruption of neuroanatomical circuits connecting the cerebellum with frontal, parietal, temporal, and limbic cortices. Damage to these connections can occur in association with cerebellar infarct (Schmahmann and Sherman, 1997), cerebellar atrophy associated with severe alcoholism (Fitzpatrick, et al., 2008), cerebellar tumor or tumor resection (Levihson, et al., 2000; Konczak, 2005), trauma, neurodegenerative disorders, or cerebellitis. Affective symptoms have been associated with damage to the cerebellar vermis (Levihson, et al., 1997). Lesions of the anterior lobe of the cerebellum

tend to produce only minor changes in executive and visual–spatial functions. Children with a cognitive affective syndrome can also have autistic characteristics, and diagnosis of autism can be confounded by cerebellar lesions.

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Cognitive Archives

► Cognitive Correctors

Cognitive Assessment System

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Synonyms

CAS

Description

The Das–Naglieri Cognitive Assessment System (CAS; Naglieri & Das, 1997a, 1997b) is a cognitive assessment to assess children aged 5 years, 0 months to 17 years, 11 months. Individual administration time is approximately 1 h. The CAS is arranged in three separate, yet interrelated levels of scores: individual subtests, PASS (Planning, Attention, Simultaneous, and Successive) composite scales, and a Full Scale quotient. Twelve subtests comprise the CAS and each subtest generates a scaled score ($M = 10$; SD = 3). The Standard Battery utilizes 12 subtests with three subtests per PASS process, while the Basic Battery includes eight subtests, two subtests for each PASS process. Each of the four PASS composite scores ($M = 100$; SD = 15) is a combination of the subtests included in each respective process. Finally, the Full Scale Score ($M = 100$; SD = 15) is the aggregate total of the four PASS cognitive processes scales, which are equally weighed.

The *Planning* scale consists of: matching numbers, planned codes, and planned connections.

1. Matching numbers – The individual is asked to locate and underline a pair of matching numbers. The task begins with 1 digit and progressively moves into 7 digit numbers.
2. Planned codes – A client is requested to complete a series of boxes according to a corresponding code provided at the beginning of each item.
3. Planned connections – This subtest is similar to the original trail making task. In this subtest, both numerical and alphabetical sequences are employed.

The *Attention* scale encompasses: number detection, expressive attention, and receptive attention.

1. Number detection – This subtest consists of rows of numbers with both target and distracter stimuli. At the top of each item page, a key is printed with the target numbers. Children are instructed to underline only the target specified.
2. Expressive attention – Children aged 5–7 are to identify the size of an assortment of animals, in spite of the size depicted on the page. For children aged 8–17, this subtest is similar to the Stroop Test. Color words are presented in a different colors of ink (e.g., the word "red" might be in blue ink) and the children are requested to name the color of the ink.
3. Receptive attention – First, a series of pictures or words are presented in which the client must identify and underline identical stimuli. Second, children are requested to recognize and identify two items that share a common characteristic. This subtest is omitted for the Basic Battery.

The *Simultaneous Processing* scale includes: nonverbal matrices, verbal–spatial relations, and figure memory.

1. Nonverbal matrices – A variety of pictures with geometrical shapes or patterns are shown to the student. The student needs to select one option that is consistent with the presented relationship or pattern.
2. Verbal spatial relations – The individual receives auditory information and determines which picture best represents the verbal description given. Presented in a multiple-choice format, the series of pictures allows the student to demonstrate understanding of logical, grammatical, and spatial information.
3. Figure memory – A client is required to trace geometric design previously observed, which is embedded within a larger and more intricate geometrical design. This is omitted from the Basic Battery.

The *Successive Processing* scale consists of: word series, sentence repetition, sentence questions, and speech rate.

1. Word series – Individuals are instructed to repeat a series of commonly used words in the same consecutive order given. The difficulty level increases as the word list starts with two words and ends with nine words.
2. Sentence repetition – This subtest demands that individuals repeat sentences that gradually become longer. The sentences in this subtest utilized color words to reduce the contextual meaning and possible interference with simultaneous processing. The score is based on the total number of correctly repeated items.
3. Speech rate – This subtest is administered only to children aged 5–7. Children are verbally presented with a series of three word combinations and are requested to repeat each combination as quickly and as many times as possible within 30 s.
4. Sentence questions – Administered to children aged 8–17 instead of speech rate. In an extension of the sentence repetition task, this subtest requires that children respond to a question about a nonsensical sentence.

Historical Background

The CAS is one of few cognitive processing instruments which incorporate a neuropsychological foundation. The theoretical basis of the CAS is an extension of Alexander S. Luria's work relating to the brain's three functional units (Naglieri, 1999, 2005). It was modified and refined by Das, Naglieri, and Kirby into four processing components: Planning, Attention, Simultaneous, and Successive Processing, otherwise known as PASS, to explain differences in cognitive processing of children (Das & Naglieri, 2001).

The *Planning* subtests require individuals to engage in a problem-solving sequence to complete novel tasks (Naglieri, 1999). The development, selection, application, and evaluation of strategies are crucial to the success of performance (Naglieri & Das, 1997b). Subtests in the *Attention* scale require a combination of three components: focused, selective, and sustained attention (Naglieri, 2005). Focused attention involves the act of attending to presented stimuli in the environment. Selective attention is the concentration of attention to chosen stimuli while disregarding nonessential or competing stimuli. Sustained attention is the differential effort (over time, especially) an individual applies toward task completion. *Simultaneous Processing* subtests require the ability of an individual to incorporate and comprehend unconnected entities and its relation/position to a collective whole (Naglieri, 2005). *Successive Processing* is described as the unidirectional, consecutive organization of stimuli (Naglieri & Das, 2005).

Psychometric Data

The standardization sample of 2,200 children is representative of the US population on nine criteria (Naglieri & Das, 1997b). The internal consistency of the CAS subtests range from 0.75 to 0.89, with a median reliability of 0.82. The Standard Battery had average reliabilities of 0.88 (Planning), 0.88 (Attention), 0.93 (Simultaneous), and 0.93 (Successive). The reliability coefficient range for the Full Scale score was 0.95–0.97.

The theoretical premise of the CAS was constructed on a four-factor model; however, factor analyses generated empirical support for both three- and four-factor models depending upon the age category. Further research has provided support for the CAS as a measure of *g*. Weak correlations were found between the CAS Standard Battery Full Scale and PASS scales (ranging from 0.37 to 0.67) with the Wechsler Intelligence Scale for Children-third edition (WISC-III; Wechsler, 1991). Predictive validity was moderately established with cluster and subtest scores from the Woodcock–Johnson Psycho-Educational Battery-Revised Tests of Achievement (Woodcock & Johnson, 1989).

Clinical Uses

The purpose of the CAS is to provide an analysis of an individual's cognitive abilities through the measurement of the PASS processes (Naglieri, 2005). The authors suggested that the CAS is a valuable alternative tool to the traditional Wechsler or Stanford–Binet scales, when assessing individuals who may have attention-deficit/hyperactivity disorders (ADHD), LD, mental retardation, traumatic brain injury, serious emotional disturbance, giftedness, and planning problems (Naglieri & Das, 1997b). Furthermore, possessing an understanding of an individual's PASS profile will provide essential information for the selection and evaluation of instructional recommendations (Das & Naglieri, 2001; Naglieri & Das, 1997b).

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Cognitive Assessors

► Cognitive Correctors

Cognitive Assistive Technology

► Prosthetic Memory Aids

Cognitive Awareness

► Metacognition

Cognitive Behavioral Couples Therapy

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Synonyms

Behavioral marital therapy; CBCT; Cognitive behavioral marital therapy; Couples therapy; Marital therapy

Definition

Cognitive Behavioral Couples Therapy (CBCT) has become one of the most well researched approaches for the treatment of marital and couple distress, with growing empirical support for it effectiveness. Theoretically grounded in both social learning and social exchange theories, the premise of CBCT is that an individual's behavior both influences and is influenced by his/her environment. When applied to a marriage or other long-term relationship, this premise suggests that one partner's behavior influences and is influenced by the actions of the other. CBCT typically focuses on two aspects of this process: (a) exchanges of positive and negative behaviors; (b) communication skills that influence the interaction process (Epstein, Baucom, & Daiuto, 1997).

Current Knowledge

Couples and Health

A patient's ongoing, long-term relationship can influence a range of psychosocial variables related to health behaviors. The health-enhancing properties of intimate and long-term relationships have been repeatedly documented (Kiecolt-Glaser & Newton, 2001; Wilson, 2001). Various mechanisms of action for this relationship have been proposed, including selection and protection (Kiecolt-Glaser & Newton, 2001). That is, healthier people are more likely to be in and stay in intimate relationships, and they tend to have more resources and take care of themselves better than their counterparts without such relationships. Additional research has investigated other mechanisms for the protective benefits of long-term relationships, including partner attitudes or behaviors, and caretaking (Keefe et al., 1996; Wilson, 2001).

Treatment Procedures

A CBCT approach to treatment with both relationship distressed and health impaired couples focuses on three factors: behavioral factors, affective/emotional factors, and cognitive factors. The behavioral component includes increasing positive behaviors such as spending more time together and decreasing negative behaviors such as criticizing or nagging. In addition, because communication problems are the most commonly reported presenting complaint of distressed couples, the behavioral aspects of the treatment also typically involve a skills-oriented approach to communication change where the

value and skills of working together to solve a problem are the foci.

Affect is a focus of therapy insomuch as it is an indicator of significant relationship distress and for its ability to direct the therapist in exploring links between the emotions of the partners and their behaviors. Affect can be approached with a skills approach in helping partners learn to express their own and listen to the other person's emotions and by linking specific emotions to specific relationship issues.

The third factor in CBCT is cognition. As Epstein et al. (1997) note, "the importance of cognitive factors in relationship functioning lies in the fact that objectively observable behavioral events are often subjectively experienced quite differently by the partners". The therapist works to uncover underling cognitive factors shaping the behavior and affect of the partners in order to increase understanding and promote behavioral change.

Efficacy Information

CBCT, and its predecessor, Behavioral Marital Therapy (BMT) have been one of the most researched forms of couples therapy (Shadish & Baldwin, 2003, 2005). Results of efficacy trials repeatedly demonstrate that those who receive either CBCT or BMT report less distress than those who receive no treatment and that this finding remains not only for couples presenting with general marital distress, but also for depression, agoraphobia, and alcohol abuse (Baucom, Shoham, Mueser, Daiuto, & Stickle, 1998). Based upon recent meta-analyses of studies using CBCT, Shadish and Baldwin (2003, 2005) reported an overall mean effect size ranging from 0.59 to 0.84 for couples therapy generally, with no differential effectiveness across theoretical orientation found.

Qualifications of Providers

CBCT can be conducted by a variety of treatment providers, specifically trained in its use with various populations. This includes neuropsychologists, clinical psychologists, marriage and family therapists, counselors, social workers, and clergy.

Cross References

► Behaviorism
► Cognitive Behavior Therapy

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Cognitive Behavioral Marital Therapy

► Cognitive Behavioral Couples Therapy

Cognitive Behavioral Therapy

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Definition

Cognitive behavioral therapy (CBT) is a theoretical framework based on the premise that a person's cognitions influence their emotions and behavior. CBT

provides considerable utility in addressing a variety of common emotional consequences of neurological disorders including anxiety and depression, as well as behavior modification for brain injury survivors.

Historical Background

CBT grew out of Albert Ellis's (Ellis, 1975) work on Rational Emotive Behavioral Therapy (REBT) and examination of irrational beliefs in the 1950s. Ellis concluded that irrational beliefs (e.g., I am powerless to solve my problems; I am unlovable) were associated with the development of mood disorders. Beck, Rush, Shaw, and Emery (1979) developed Cognitive Therapy on the premise that cognitive errors (e.g., over-generalizing, magnification, personalization) were associated with the development of depression and anxiety. Further, they viewed depression as accompanied by of a triad of negative cognitions consisting of a negative view of self, the world, and the future.

Arnold Lazarus (1971) was the first to introduce the term "behavior therapy" into the professional literature. Further expanding the lens of CBT, Lazarus and Folkman (1984) developed the stress, appraisal, and coping model (1984) that acknowledges the importance of how an individual views the environment (primary appraisal) and their available coping resources (secondary appraisal). Finally, attribution theory, proposed by Fritz Heider (1958) posits that people can interpret events as caused by internal (i.e., factors within the person such as a person's own intelligence and behavior) or external factors (i.e., factors external to the person such as the weather or luck). When internal attributions are made, people are said to have an internal locus of control, or hold cognitions that support their sense of efficacy in affecting what happens to them. When external attributions are paramount, people are said to have an external locus of control, that is, they believe that they exert less control over their environment.

Goals and Objectives

Neuropsychologists using CBT as a conceptual framework work toward modifying maladaptive cognitions such as negative attributions, unattainable expectations and standards, and faulty belief systems. Through the modification of these problematic (i.e., schemas), CBT seeks to alter people's emotional and behavioral responses in service of symptom management, reduction, and alleviation. To reduce the emotional experience of frustration, a neuropsychologist working with a brain-injured patient would encourage the patient to appreciate the gains made since the injury. In order to avoid unrealistic expectations for post injury functioning that could create frustration; the patient would be cautioned against comparing present capabilities to preinjury functioning.

Treatment Participants

CBT is broadly applicable to a variety of clinical neurological diagnoses including anxiety, depression, obsessive compulsive disorder (OCD), and posttraumatic stress disorder (PTSD). CBT can be used with individuals, couples, and families. Further, CBT can be used with patients at developmental levels from young children to older adults. CBT therapists act more as coaches collaborating with patients alter processes of thinking, feeling, and action as opposed to an analyst making expert interpretations.

Treatment Procedures

CBT encompasses a variety of clinical interventions for individuals with a neurological disorder as well as couples and families in which a member has a neurological diagnosis or injury. Within all CBT interventions, behaviors, cognitions, and emotions are integrally related; though interventions may focus on behavior or cognition, these distinctions are often made for heuristic purposes (Epstein & Baucom, 2002). Further, CBT posits that change in one domain will produce changes in others domains. In the past, behavior-oriented therapies have been ripe with skills training in areas such as problem-solving, communication, and relaxation with the idea being that maladaptive patterns arise around areas of skill deficits. Though CBT therapists also deliver a judicious amount of skills-based training, therapy moves beyond behavior modification to the meaning and interpretations made as a result of interactions and experience.

Behavior-Focused Interventions

Patients with neurological diagnoses have a host of behavior patterns amenable to CBT intervention. One such pattern, negative reciprocity, is the idea that negative behavior increases the propensity that a person will respond to expressed negative behavior with more

negative behavior (Epstein & Baucom, 2002). For example, family members may respond angrily toward a patient who acts aggressively after a brain injury. Over time negative reciprocity pervades relationships, invading cognitions and emotions such that family members make global negative attributions about another person's intentions and behavior (Epstein & Baucom, 2002).

An important aspect of CBT is skills-based training; meant to enhance positive behavior and decrease negative behavior. In an instance where a person receives a diagnosis of schizophrenia, a CBT clinician may intervene with a family to teach communication skills. Empirical literature has discussed the detrimental impact of expressed emotion (EE) in families with a schizophrenic family member. Further, the family members may be overwhelmed and have many questions, concerns, and emotions they need to express surrounding the diagnosis. Communication skills training teaches families how to communicate more productively with one another about complex issues and reduce detrimental patterns of interaction such as EE.

Cognition-Focused Interventions

The manner in which a person cognitively ascribes meaning to behavior is an important factor in CBT. Therapy often focuses on reassessing and amending these cognitions. Areas of inquiry often include attributions, expectancies, assumptions, standards, and beliefs. In order to evaluate and modify existing cognitions, clinicians intervene using guided discovery during which clients are asked to identify and evaluate their cognitions (Epstein & Baucom, 2002). When a patient is depressed about a neurological diagnosis or injury, clinicians may intervene at the cognitive level challenging catastrophic thinking (e.g., I will never get better). A therapist may focus decreasing negative self talk (e.g., I am worthless the way I am now) by encouraging the patient to keep a journal of thoughts occurred and the impact of the thoughts on the patient's mood (Epstein & Baucom, 2002).

Emotion-Focused Interventions

Interventions within the realm of emotions may range from expanding minimized emotional experience to containing heightened emotional experience. Clinicians may draw on a variety of strategies to access, heighten, or limit emotional experience including normalizing emotional responses, metaphor, acceptance of emotional expression, enhancing tolerance for distressing emotions, and encouraging healthy compartmentalization of emotion (Epstein & Baucom, 2002). In cases of neurological diagnosis or injury, emotions may either be exacerbated or minimized. To enhance the expression of emotions, therapists may ask probing questions to bring awareness to emotional experience such as: What happens to you when. . .What is it like for you when. . .How do you feel as you listen to your son expressing his experience. . . (Epstein & Baucom, 2002). In this way, individuals and family members can be encouraged to share their emotional experience in the context of a safer therapeutic environment.

Efficacy Information

CBT has been empirically validated for the treatment of many disorders including anxiety (Barlow, O'Brien, & Last, 1984), sexual dysfunction (Baucom, Shoham, Mueser, Daiuto, & Stickle, 1998), depression (Beach, Sadeen, & O'Leary, 1990), bipolar disorder, schizophrenia, and bulimia nervosa (Baucom et al., 1998). CBT is often used in conjunction with medication for a variety of mental health concerns. In addition, CBT effectively enhances coping skills for adults with chronic illness (Rybarczyk, DeMarco, DeLaCruz, Lapidos, & Fortner, 2001) and caregivers (Gallagher-Thompson, Lovett, Rose, McKibbin, Coon, et al., 2000).

Recently, advances in computer software have given rise to computerized versions of CBT. Though computerized cognitive behavioral therapy (CCBT) is not meant t o replace face-to-face therapy, it does provide an additional treatment option. CCBT allows clients to participate in therapy when there is a paucity of available therapists, the associated costs are prohibitive, or the prospect of speaking to someone face-to-face seems off-putting. In 2006, the United Kingdom's National Institute of Health and Clinical Excellence (NICE) provided guidelines recommending CCBT as a result of randomized controlled trials for mild to moderate depression and anxiety (NICE, 2006).

Outcome Measurement

Many clinicians used standardized instruments to assess the presence and severity of a variety of neuropsychological diagnoses. With regard to depression, clinicians may use self-report inventories such as the Beck Depression Inventory (BDI II; Beck, Steer, & Brown, 1996) Finally, the Beck Anxiety Inventory (BAI: Kabacoff, Segal, Hersen, &

Van Hasselt, 1997) has shown considerable utility in diagnosing anxiety and identifying the severity of the anxiety symptoms.

Qualifications of Treatment Providers

CBT can be used by a variety of treatment providers such as neuropsychologists, clinical psychologists, marriage and family therapists (MFTs), counselors, and social workers. Treatment providers using CBT should have appropriate clinical training in the model. Further, clinical providers learning to use CBT as a conceptual framework to guide therapy should seek supervision from an experienced individual trained in the model of CBT.

Cross References

- ▶ Behaviorism
- ▶ Behavior Modification
- ▶ Psychotherapy

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Cognitive Behaviorism

▶ Behaviorism

Cognitive Control

▶ Controlled Attention

Cognitive Correctors

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Synonyms

Behavioral memory aids; Behavioral prothestics; Cognitive archives; Cognitive assessors; Cognitive monitors; Cognitive orthotics; Cognitive robots; Cognitive trainers; External aids; Prosthetic devices

Definition

A device, external to the mind, that enhances or replaces a memory or cognitive function.

Current Knowledge

Parente and Herrmann (2010) defined several different external aids that take over a deficient cognitive or memory function. Table 1 surveys the various types of cognitive correction devices.

Cognitive prosthetics refer to a general class of devices that take over some memory process. Examples of particularly useful prosthetics include: *color coding,* which creates simplistic visual organization; *checklists,* which improve consistency and sequential ordering; *medication organizers,* – which organize medications and remind a person which ones to take on each day; *notepads and post-it notes,* –which are useful for quickly writing a message or for placing a written message somewhere where it will be seen later; *appointment calendars and diaries,* – which remind the person of important appointments and provide a record of daily activities.

Cognitive robots carry out a repetitive task for an individual. For example: *calculators* perform the same mathematical operations; *cycling timers* turn appliances on and off at preset times; *telememo wrist watches* remind the person of appointments at the same time every day, week, month, or year; *motion-sensitive detectors* automatically turn lights on and off when motion or the lack thereof is sensed; *smart irons* turn themselves off after a short period of disuse.

Cognitive Orthotic Devices replace some cognitive function. For example: *spelling machines* find the correct spelling by keying in a close match; *internet search engines* search the internet using key words and Boolean logic; *grammar checkers* check a document for accuracy of grammar.

Cognitive Correctors. Table 1 Kinds of cognitive correctors

Behavioral prosthetics	Changes in behavior that are specifically designed to remind a person to do something
Cognitive robot	Carries out the same task consistently and correctly
Cognitive orthotic	Performs a thinking task
Cognitive trainers	Help the person to learn new skills and to practice them
Cognitive archives	Maintain knowledge and records of past experience so that a person does not have to store and retain this information internally in his or her memory

Cognitive trainers and *archives* teach a skill and/or store large searchable databases. For example: *academic remediation software* teaches basic academic skills; *cognitive remediation software* provides drill and practice exercises that improve cognitive skills; *skills training software* teaches skills like typing or the use of specific software packages (e.g., Microsoft Office). *Cognitive archives* (e.g., electronic encyclopedias) store large amounts of information and provide a search engine for finding specific topics.

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Cognitive Exercises

▶ Homework

Cognitive Flexibility

▶ Mental Flexibility
▶ Stroop Effect

Cognitive Functioning

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Synonyms

Cognition; Mentation; Thinking

Definition

"Cognitive functioning" is a general term used to describe the different ways that people think. It refers to faculties such as attention, mental processing speed, executive functions, language, visual spatial skills, memory, and

fine motor dexterity. Different cognitive functions are supported by distinct cortical and subcortical brain regions. Disruption of neural processes in these brain regions can result in a range of cognitive deficits and syndromes.

Historical Background

In the 1600s, Descartes, a philosopher, was one of the first scholars to establish the idea that the brain controls behavior. In the late 1700s, Franz Gall, a forefather of phrenology (the study of behavior based on the size and shape of the skull) helped identify that different parts of the brain regulate distinct aspects of thought, personality, and behavior. Later on in the 1800s and 1900s,Wilder Penfield, Hughlings Jackson, Paul Broca, and Carl Wernicke, to name only a few researchers who contributed significantly to the field of neuropsychology, used epilepsy and lesion models to delineate distinct neuroanatomical correlates of cognitive and motor functions. More recently, functional (e.g., fMRI, PET) and structural (e.g., MRI, CT) neuroimaging techniques have provided an even more well defined understanding of brain-behavior relationships.

Current Knowledge

Attention

Attention refers to the ability to concentrate on information that is heard and seen in the environment in both the presence and absence of distractions. It also allows us to concentrate on two things at once, such as balancing a checkbook while talking on the phone. It is regulated by the frontal lobes, although pathways involving the pons, parietal lobe, and thalamus are involved in the mediation of attention, as well. Dysfunction along these pathways can contribute to various types of attention problems.

Attention is hierarchically organized, and disruption of the most basic attention skills leads to disruption of more complex attention abilities. Focused attention is the most rudimentary level of attention that permits us to concentrate or be vigilant to something in the environment for a very brief time period. Sustained attention refers to maintenance of concentration for minutes or hours. Selective attention requires even greater attention capacity that allows us to attend to a particular task while filtering out irrelevant, background information. For example, this form of attention is used when we read the newspaper and are not distracted by noises such as the radio or television nearby. Divided attention allows us to pay attention to two or more tasks simultaneously. At the top of the hierarchy is alternating attention, which is the most complex form of attention that involves shifting of attention from one task to another.

Many neuropsychological measures have been designed to evaluate attention. One such measure is the digit span test. This task assesses basic attention capacity through repetition of series of numbers of increasing length (e.g., 1-2-3-etc). Continuous performance tests are computerized assessment tools that evaluate attention capacity over an extended time period of 10–15 min. Higher level attention skills can be examined using tests involving mental arithmetic, more complex mental arithmetic, connecting numbers and letters in alternating sequence (e.g., 1-A-2-B-3-etc), and resequencing of numbers and letters in numeric and alphabetic order (e.g., transform "8-K-2" to 2-8-K).

Attention problems can be seen across the lifespan. In children, poor concentration, distractibility, and trouble regulating behavior can be related to an Attention Deficit Disorder, a diagnosis that is made when pervasive attention difficulties are demonstrated prior to age seven years and are observable in at least two different environments. However, attention difficulties in children also may develop secondary to anxiety, language disorders, or neurological disorders such as epilepsy. In adults, attention deficits may manifest secondary to a variety of neurological and medical conditions, including subarachnoid hemorrhage, epilepsy, dementia, head injury, diabetes, and hypothyroidism. Stress, depression, and medication side effects often contribute to attention problems in adults, as well.

Mental Processing Speed

Mental processing speed, a term used synonymously with reaction time, refers to the speed at which an individual thinks and completes activities. It is largely regulated by the frontal lobes and subcortical regions, and it has global effects on cognition. That is, if mental processing speed is poor slowness also is observed in areas of attention, language, and spatial processing abilities. This mental slowing is referred to as bradyphrenia.

Reaction time can be evaluated in several ways. Measures requiring manual transcription of numbers and shapes are very sensitive to bradyphrenia. Other tasks may involve rapid reading of color names and naming of colors, timed symbol search, and rapid generation of words.

Bradyphrenia is commonly observed in medical and neurological conditions across the lifespan, including dementia, brain injury, and Parkinson's disease.

Executive Functions

Executive functioning refers to some of the most highly complex aspects of thinking. It includes skills such as problem solving ability, task execution and efficiency, self-monitoring, strategizing, mental flexibility, planning, and conceptualization. Executive functions are largely mediated by the frontal lobes, and they are highly sensitive to cerebral dysfunction.

Deficits in executive functioning can occur secondary to neurodevelopmental immaturity, such as seen in attention deficit disorder. For the most part, however, executive dysfunction is either acquired through brain injury (e.g., stroke, head injury) or associated with neurodegenerative diseases, such as Alzheimer's disease, multiple sclerosis, and Huntington's disease.

Language

At the most fundamental level, language can be broken down into two categories: receptive and expressive language. Receptive language refers to the ability to understand language; it is mediated by the posterior region of the superior temporal gyrus, which is known as Wernicke's area. Expressive language, on the other hand, pertains to oral and written expression and it is regulated by the posterior frontal/anterior temporal lobe. This region commonly is referred to as Broca's area.

Deficits in receptive and expressive language can be idiopathic (i.e., of unknown origin) or acquired. If language problems are idiopathic, they typically are identified during a child's development and are referred to as a developmental language delay. As some children mature, their language difficulties might resolve; however, other children experience residual language deficits throughout adulthood. On the other hand, acquired language deficits such as those resultant from stroke, head injury, and tumor are characterized as aphasia. Depending on the location of brain injury, different aphasia syndromes can be observed. Listed below are three of the most commonly referenced syndromes.

Traditionally, language skills in adults are assessed with an aphasia battery (e.g., Boston Diagnostic Aphasia Examination (BDAE)), letter and category fluency, and visual confrontation naming tasks. As one of the most commonly observed language problems in adults is a naming deficit (which is synonymous with word finding difficulty, dysnomia, and "tip of the tongue" experiences), auditory naming tests have proven to be useful for identification of anomia, as well. Similar measures are used during evaluation of pediatric populations. In addition, examination of children also should include assessment of phonics, grammar, syntax, language formulation, and language organization skills.

Cognitive Correctors. Table 1

Aphasia syndrome	Neuroanatomy	Symptom(s)
Broca's (expressive) aphasia	Posterior, lateral frontal lobe	Poor articulation, telegraphic, non-fluent, limited speech, intact comprehension
Wernicke's (receptive) aphasia	Anterior, lateral temporal lobe	Poor comprehension, fluent speech, nonsensical speech content
Anomic aphasia	Temporal-parietal border	Poor naming, intact comprehension, intact fluency

Visual Spatial Processing

Cognitive abilities such as basic spatial perception, visual construction, and nonverbal problem solving ability are characterized as spatial processing skills. These faculties involve identifying "what" and "where" items are in space. The "what," or ventral, pathway is mediated by the right (nondominant) temporal lobe. Disruption of this pathway by acquired brain injury can result in agnosia, which is a loss of awareness and inability to recognize entities in the environment. Agnosias can be domain specific, affecting auditory, visual, or somatosensory functioning. Prosopagnosia is a unique agnosia in which there is an inability to recognize faces.

The "where" or dorsal pathway is regulated by the parietal lobe. Dysfunction in the parietal lobe can contribute to spatial neglect in which a portion of space is neglected or ignored. Neglect can be detected during visual field screening and on line bisection and drawing tasks. An individual with spatial neglect will not respond to information presented in part or all of the visual field. Left hemispatial neglect, in which the entire left side of space is ignored, is the most commonly observed form of neglect that results from damage to the right parietal lobe.

Memory

Memory involves learning, retrieval, retention, and recognition of information. Memory functions are lateralized, with verbal memory regulated by left (dominant) temporal regions and visual-spatial memory regulated by the right temporal lobe. In order for new learning to occur, we must be able to attend to the material and store it temporarily in our short-term memory. Consolidation of this information into long-term memory stores occurs within the CA1-CA3 regions of the hippocampus, a subcortical brain region rich in acetylcholine. Once learned, information is then stored in various neural networks throughout the brain.

Learning deficits can be attributed to depletion of acetylcholine or loss of brain cell within the hippocampus. This type of memory problem typically is associated with cortical dementia, such as Alzheimer's disease. Cholinesterase inhibitors, which are medications that promote cholinergic availability, are common treatment options to delay or slow the progression of a learning deficit. Retrieval deficits, on the other hand, are characterized by poor spontaneous recall, yet adequate recognition of newly learned material. They are associated with cell loss and neurochemical depletion of dopamine and glutamate in the basal ganglia and other subcortical regions. Retrieval deficits are observed in subcortical dementias, including Parkinson's disease and Huntington's disease. Although cholinesterase inhibitors can be used to treat subcortical memory dysfunction, their efficacy is relatively weaker in subcortical versus cortical dementia populations.

Motor

Motor abilities refer to skills such as hand-eye coordination, manual dexterity, and visual-motor integration. These skills all have contralateral representation within the parietal lobes. That is, right sided motor functions are regulated by left parietal lobe regions, and vice versa. Motor dysfunction can be observed in children with developmental delay, as well as in adults and children with medical conditions including cerebral palsy, spasticity, and dementia. It often manifests as clumsiness, poor handwriting, or trouble tying shoe laces and buttoning shirts. Motor abilities can be evaluated using tasks requiring rapid placement of pegs into a pegboard, drawing, imitation of hand movements, finger tapping, and grip strength.

Cross References

- ▶ Agnosia
- ▶ Agraphia
- ▶ Alexia
- ▶ Amnesia
- ▶ Anomia
- ▶ Aphasia
- ▶ Attention
- ▶ Learning
- ▶ Memory
- ▶ Processing Speed
- ▶ Syndrome
- ▶ Verbal Fluency
- ▶ Visuoperceptual

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Cognitive Functioning in the Elderly

- ▶ Normal Aging

Cognitive Impairment No Dementia (CIND)

- ▶ Mild Cognitive Impairment

Cognitive Log

- ▶ Cognitive-Log

Cognitive Monitors

► Cognitive Correctors

Cognitive Orthotics

► Cognitive Correctors
► Prosthetic Memory Aids

Cognitive Potential

► Best Performance Method

Cognitive Processing

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Definition

Cognitive processing is a general term to describe a series of cognitive operations carried out in the creation and manipulation of mental representations of information. Cognitive processes may include attention, perception, reasoning, emoting, learning, synthesizing, rearrangement and manipulation of stored information, memory storage, retrieval, and metacognition. These functions can be conscious (e.g., learning a concept) or unconscious (e.g., learning a skill) and can be internally generated (e.g., recalling a memory) or initiated by a novel sensory input from the environment (e.g., solving a problem).

From a cognitive psychology perspective, cognitive processing is approached as a sequence of ordered stages wherein sensory input is transformed, reduced, elaborated, stored, recovered, and utilized. Early views of cognitive processing emphasized linear temporal processing, whereas contemporary models assume a less linear, more complex flow of dynamics, including bottom-up (sensory-driven) and top-down (concept-driven) processes. One cognitive psychology approach to better understanding cognitive processing has been through the development of computational models, such as artificial intelligence.

In cognitive neuroscience research, cognitive processing concepts are used to explore the relation between brain and behavior, as exemplified by George Miller's research (1956) on the capacity of short-term memory to hold seven plus or minus two items and Baddeley's theory (1974) of a central executive, phonological loop, and visuospatial sketchpad.

Current Knowledge

The cognitive processing model is currently used in the assessment and treatment of learning disabilities, alcohol and drug addictions, and trauma and abuse. This model emphasizes how new information is processed, internalized, and retrieved in the context of a person's existing mental representations of information, and of his/her beliefs, desires, knowledge, preferences, and intentions.

Cross References

► Attention
► Cognitive Functioning
► Learning
► Memory
► Metacognition
► Perception
► Reasoning
► Retrieval, Retrieval Techniques
► Short Term Memory

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Cognitive Processing Speed

► Information Processing Speed

Cognitive Rehabilitation

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Synonyms

Cognitive Remediation

Definition

Cognitive rehabilitation (CR) can be defined as efforts to promote maximal adaptive cognitive functioning in people with neurologically induced cognitive deficits (Barrett & Gonzalez-Rothi, 2002).

Historical Background

The field of CR has grown rapidly over the last few decades, but historically can be traced to the 1800s (Ponsford, 2004; Sohlberg & Mateer, 2001). For example, Broca administered language rehabilitation in the 1800s and until the 1980s most rehabilitation programs focused on remediation of language deficits (as reviewed by Ponsford, 2004). Many further developments in CR were a result of the confluence of societal influences and scientific and technological advances. During WWI, Goldstein established CR programs for brain-injured soldiers. During WWII, Luria advanced the field of CR through his theoretical model of brain functioning, recovery, and rehabilitation (Ponsford, 2004).

Advances in medical practice and an increase in the number of survivors of traumatic brain injury (TBI) led to a greater awareness of the needs of people who sustained TBI, and an expansion of the number and focus of CR programs (the "era of proliferation"; Coelho, 1997, as cited by Sohlberg & Mateer, 2001). However, current trends in service delivery have resulted in an "era of consolidation," a term describing the significant downsizing of CR programs. It has been suggested that the reduced length of inpatient stays, outpatient health coverage, and more limited support for CR programs have made evidence and theory-based CR practices increasingly relevant to contemporary practice (Cicerone et al., 2005; Levine & Downey-Lamb, 2002; Sohlberg & Mateer, 2001).

Rationale or Underlying Theory

CR is multidisciplinary and draws from a range of fields, including neuropsychology, learning theory, cognitive behavioral therapy and psychotherapy, among many others. One main category of theories underlying CR is that specific to the cognitive or behavioral domain of CR focus. For example, attention rehabilitation programs are based on theoretical models of attention, memory rehabilitation programs on theoretical models of memory, and so on. A theory-driven approach provides a rational and empirical basis for intervention, and guidance on the structure and delivery of CR (Levine & Downey-Lamb, 2002; Sohlberg & Mateer, 2001).

Another major theory underlying many forms of CR is neuroplasticity (Kolb & Cioe, 2004), the concept that the brain is amenable to change in structure and function. These changes occur at multiple levels, and are manifest in various ways, including changes at the synapse, changes in neurotransmitter systems, and changes in neural networks (Barrett & Gonzalez-Rothi, 2002; Sohlberg & Mateer, 2001). Neuroplasticity has many implications to CR, including the type and timing of CR, and the effect of environmental factors on recovery of cognitive function following brain injury (Barrett & Gonzalez-Rothi, 2002).

Goals and Objectives

CR aims to foster natural recovery, decrease the development of maladaptive patterns, and increase functional recovery (Sohlberg & Mateer, 2001). The primary goal of CR is to help people achieve an optimal level of functioning in the context of impairments. CR emphasizes improving function in everyday contexts, rather than on specific cognitive tasks per se.

Treatment Participants

CR has been used with a variety of populations, including but not limited to: TBI, stroke, acquired brain injury (ABI) of varying etiology, developmental disorders, Alzheimers' dementia, and schizophrenia. CR has been most commonly used among people who have sustained TBI and stroke. Research on CR aimed at neurocognitive deficits associated with schizophrenia has been growing over the last 10 years (Kurtz & Nichols, 2007).

Variables contributing to the pattern and degree of recovery following brain injury include: demographic

(e.g., age, education, gender, culture), injury-related (e.g., time since injury, extent, and severity of injury), and psychological characteristics (e.g., therapeutic alliance, comorbid psychological disorders, awareness).

Demographic Variables

Age: Younger adults show better levels of recovery than older adults (Teuber, 1975 as cited by Sohlberg & Mateer, 2001). ABI in older adults may be complicated by a number of factors, including the superimposition of effects of ABI on declining cognitive abilities (Richards, 2000 as cited by Sohlberg & Mateer, 2001), and psychosocial difficulties more prevalent in the population, including reduced levels of social support and financial resources (Goleburn & Golden, 2001). However, it has also been suggested that older adults often have a greater degree of stability, coping skills, fewer life demands, and effective compensatory techniques, which may be helpful to promoting recovery (Sohlberg & Mateer, 2001).

Education and Intelligence: Premorbid intelligence and education are significantly related to recovery and adjustment (Anson & Ponsford, 2006).

Gender: Some research suggests that women have better recovery following left hemisphere lesions than men (Kimura, 1983), and circulating sex hormones have also been shown to have neuroprotective effects (e.g., Roof, Duvdevani, & Stein, 1993).

Culture: Culture influences beliefs regarding the nature and cause of loss, service utilization, degree of personal responsibility for health, role of family, and many other facets of psychological and behavioral functioning relevant to recovery and participation in CR (Sohlberg & Mateer, 2001).

Injury Related Variables

Time since Injury: Spontaneous recovery typically occurs at a faster rate immediately following brain injury, particularly within the first 6 months, with significant recovery also occurring up to 2 years following injury (Sohlberg & Mateer, 2001). However, it is important to note that compensatory techniques can be implemented and underlying motor and cognitive skills improved years after injury (e.g., Shaw et al., 2005).

Extent and Severity of Injury: Relatively mild injuries are associated with faster recovery rate and better outcomes. Focal injuries are often associated with more rapid recovery than diffuse injury (Sohlberg & Mateer, 2001).

Psychological Factors

Therapeutic Alliance: CR should be an interactive partnership between the client, their significant others, and the therapist. Cultivating a relationship characterized by attentiveness, respect, trust, commitment, and rapport is a critical component of CR. Open communication and involvement of the client and family in goal setting can also enhance engagement in rehabilitation (Sohlberg & Mateer, 2001).

Comorbid Psychological Disorders: Depression and anxiety are frequently associated with brain injury (e.g., Anson & Ponsford, 2006). These can impede CR and adjustment following injury due to their propensity to decrease motivation and contribute to a feeling of hopelessness (Sohlberg & Mateer, 2001).

Awareness: Lack of awareness can occur following brain injury, and has been associated with poor self-regulation, disengagement in CR programs (Allen & Ruff, 1990), and poorer outcome.

Although these factors provide important information related to the pattern and degree of recovery following brain injury, it should also be noted that much more research is needed to better identify therapy factors and client characteristics that optimize clinical outcomes of CR (Cicerone et al., 2005).

Treatment Procedures

Examples of domains that have been a focus of CR include: attention, memory, language, visuoperceptual difficulties, executive functions, and socioemotional and behavioral disturbances. CR encompasses a range of interventions. These can be broadly divided into two types of techniques. The first are those that aim to restore or enhance function, by targeting the underlying impairment (Glisky & Glisky, 2002; Sohlberg & Mateer, 2001). For example, Attention Process Training (APT) is a theoretically-driven program that contends that attention can be improved through repeated activation of attentional systems (Sohlberg & Mateer, 1987, 2001). APT consists of a group of hierarchically organized tasks that exercise different components of attention (e.g., sustained, selective, alternating, divided attention). In CR of memory deficits, restorative/generalized memory approaches aim to improve specific memory systems

across tasks and contexts (e.g., prospective memory training, Raskin & Sohlberg, 1996 as cited by Sohlberg & Mateer, 2001). Various approaches to executive function rehabilitation provide practice in executive skills (e.g., planning) and guiding behavior through self-talk (e.g., self instructional training, Cicerone & Giacino, 1992).

The second category of CR interventions is compensatory techniques, which aim to compensate for, or bypass deficits (Sohlberg & Mateer, 2001; Wilson & Zangwill, 2003). These include environmental supports (e.g., organization of physical space, manipulation of physiological factors such as sleep, nutrition etc.) and external aids (e.g., calendars, pagers, checklists, etc.; Manly, Ward, & Robertson, 2002; Wilson & Zangwill, 2003). Compensatory techniques can be helpful in managing diverse types of cognitive difficulties (Sohlberg & Mateer, 2001).

A third approach involves the use of specialized approaches to teaching and stabilizing new behaviors and knowledge in people with memory difficulties. These include instructional techniques such as errorless learning, in which mistakes are minimized (Wilson, Baddeley, Evans, & Shiel, 1994), the method of vanishing cues (Glisky & Glisky, 2002) and traditional behavioral shaping and training techniques.

Psychosocial support or psychotherapy (e.g., supported listening, brain injury education, relaxation training) can also be an integral part of a rehabilitation program, depending on the needs of the client (Sohlberg & Mateer, 2001). Computer programs can be used as an adjunct to CR, but should not be the sole form of CR (Cicerone et al., 2005). It has been recommended that computer CR programs be focused, structured, monitored, and ecologically valid. A successful rehabilitation program typically involves a combination of interventions, specifically tailored to the individual's level of disability and personal goals (Manly et al., 2002; Solhberg & Mateer, 2001).

The duration and frequency of CR varies widely (e.g., Geusgens, Winken, van Heugten, Jolles, & van den Heuvel, 2007; Kurtz & Nichols, 2007). CR has been delivered both on an individual and on a group basis. Significant others (e.g., family) are viewed as an integral part of treatment (Sohlberg & Mateer, 2001).

Efficacy Information

Cicerone et al. (2005) examined differential treatment effects of CR compared to alternate treatment conditions in 46 class I studies with TBI and stroke populations. Their review suggested a clear differential benefit of CR compared to a number of alternate treatment conditions, with no comparison demonstrating a benefit for an alternate treatment condition.

Although implementation of CR programs results in positive change, a number of methodological problems have been identified in the CR literature, including but not limited to:

1. Variability in client characteristics and treatment settings
2. Insufficient description of samples
3. Small sample sizes
4. Inadequate description of interventions
5. A lack of standardized treatment protocols and treatment approaches (including type of intervention, length, and intensity)
6. Lack of appropriate control conditions (e.g., no treatment or alternate treatment)
7. Lack of uniform outcome measures

Evidence-based standards of CR are frequently identified as important in advancing the field of CR, both in terms of quality of treatment and for fiscal support at an organizational level (e.g., Sohlberg & Mateer, 2001).

Cicerone et al. (2005) reviewed evidence for CR interventions used with TBI and stroke populations. A number of interventions with strong empirical support were identified. Strategy training (e.g., APT or Time Pressure Management) was effective during postacute rehabilitation for TBI. A number of visuospatial rehabilitation techniques were empirically supported for improving neglect following right hemisphere stroke (e.g., visuospatial rehabilitation, scanning training). Specific gestural or strategy training was found to be effective in the treatment of apraxia following left hemisphere stroke. A number of language interventions were shown to have empirical support, including cognitive linguistic therapies, pragmatic conversational skills, interventions for specific language impairments (e.g., reading comprehension), and adjunctive computer-based interventions. Strong empirical support was shown for memory strategy training (e.g., visual imagery and external aids, such as memory notebooks). Some empirical support was found for self-instruction and self-monitoring interventions for executive functions. Empirical support was found for comprehensive-holistic CR programs, which address multiple aspects of impairment.

Interventions that meet criteria for inclusion as an empirically supported treatment do not represent an exhaustive list of CR interventions that may be effective, but reflect the empirical status of the field of CR research and practice. A successful rehabilitation program typically

involves a combination of interventions, specifically tailored to the individual's level of disability and personal goals (Manly et al., 2002; Solhberg & Mateer, 2001). Therefore, clinical considerations may demand CR interventions with strong empirical support, as well as those that have less empirical support but may be beneficial to the client. A discussion of limitations and strengths of empirically supported and empirically validated interventions can be found elsewhere (e.g., Chambless & Ollendick, 2001).

There is less empirical study of CR with populations beside TBI and stroke. A review of CR with children who have sustained ABI concluded that there is a sufficient amount of evidence to recommend attention remediation, involvement of family members in the treatment plan, and provision of psychoeducation to guardians (Laatsch et al., 2007). A review of studies of CR with people with early stage Alzheimer's disease suggested CR could be beneficial, particularly when flexible to the needs of clients, of sufficient duration, and involving caregivers (Clare, 2003). A review of studies of CR with people with schizophrenia has suggested improvements on memory and executive functions, although it should be noted that this is an emerging field so is based on a small number of studies (Kurtz & Nichols, 2007). A review of CR for people with multiple sclerosis suggested empirically based support for verbal learning and memory intervention, and suggested additional research support is needed (O'Brien, Chiaravalloti, Govereover, & DeLuca, 2008).

Outcome Measurement

Levine and Downey-Lamb (2002) recommend inclusion of both specific outcome measures that relate closely to the construct addressed by the intervention, and well as general measures of functional outcomes, such as return to work/school, interpersonal relationships, and leisure activities. When possible, measures should have known psychometric properties, and be completed by both the client and significant others.

Neuropsychological assessment can be a valuable tool at various points to: predict outcome, guide appropriate rehabilitation strategies, guide vocational and educational planning, explain behavior, and help evaluate the extent of injury in conjunction with other information (Bergquist & Malec, 2002).

Measurement of transfer of training to everyday life has been measured through assessment of performance on tasks similar to tasks used during training, standardized observations of simulated performance of daily tasks in a laboratory environment, and standardized and nonstandardized reports of transfer to daily functioning (Geusgens et al., 2007). There is a need for further development of standardized measure of transfer with good psychometric properties (Geusgens et al., 2007). It has been recommended that generalization should not be "expected," but should be "programed" throughout the CR program (Sohlberg & Mateer, 2001).

Qualifications of Treatment Providers

CR is typically provided by registered psychologists, and occupational therapists. Speech and language therapists typically provide rehabilitation for language and communication deficits.

Cross References

- ▶ Assistive Technology
- ▶ Attention Training
- ▶ Behavioral Memory Aids
- ▶ Brain Plasticity
- ▶ Compensatory Strategies
- ▶ Environmental Modifications
- ▶ Errorless Learning
- ▶ Head Injury
- ▶ Insight, Effects on Rehabilitation
- ▶ Interdisciplinary Team Rehabilitation
- ▶ Memory
- ▶ Neglect and Heminattention
- ▶ Neuropsychological Rehabilitation
- ▶ Prosthetic Memory Aids
- ▶ Rehabilitation Psychology
- ▶ Traumatic Brain Injury

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Cognitive Remediation

► Cognitive Rehabilitation
► Neuropsychological Rehabilitation

Cognitive Reserve

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Synonyms

Brain reserve; Neural compensation; Neural reserve

Definition

Cognitive reserve is a concept often used to describe how individual differences mediate the clinical expression of brain damage. In this context, some individuals may cope better than others and function within relatively normal limits, despite the presence of neuropathology.

Historical Background

Historically, one of the earliest observations of cognitive reserve was described in a study that found characteristic senile plaques and neurofibrillary tangles commonly associated with Alzheimer's pathology present in healthy, cognitively unimpaired elderly (Blessed, Tomlinson, & Roth, 1968). Similar observations between brain pathology and performance variability frequently have been described in the extant literature.

Current Knowledge

While the underlying mechanisms that support cognitive reserve remain unclear, current theories focus on how the brain may develop alternative or more efficient networks to compensate for pathology. One theory proposed by Satz (1993) holds that brain mass and neuronal count – or brain reserve capacity (BRC) – may raise or lower the brain's threshold to withstand a lesion or degenerative process. In this model, lower BRC would make an individual more vulnerable to neurological insult and increase test sensitivity in detecting impairment. Similarly, greater BRC would provide a higher threshold before the effects of neuropathology are observed. For instance, one study (Katzman et al., 1988) compared a high-functioning group of nursing home residents with Alzheimer's pathology with a group of healthy nursing home controls. When compared across several cognitive domains, the Alzheimer's group performed at or above the levels of healthy controls. In this study, the observed cognitive deficits were much lower than predicted, given the level of brain pathology in these individuals. This was explained, in part, by the finding that those in Alzheimer's group had larger brains with more neurons than the control group, which may have helped those individuals compensate for their neuropathology. Genetics may play a primary role in building greater BRC by increasing overall brain mass, synaptic density, or neurogenesis, all of which could provide greater resiliency against lesions or a degenerative process.

By contrast, the concept of cognitive reserve suggests that individual differences in genetics or life experiences provide a buffer to the effects of brain disease, including dementia. Neural reserve or neural compensation models (Stern, 2002) are proposed as potentially active processes that facilitate the brain's attempts to adapt to disease pathology. Various lines of animal studies have supported this model noting the benefits of enriched environments on neurogenesis and plasticity in the brain. In humans, years of education and occupational attainment are associated with the preservation of cognitive functions. For instance, individuals with lower education often demonstrate clinical manifestations of dementia earlier in the disease process compared to those with higher education (Stern, 2003).

One of the important implications of the cognitive reserve construct is that lifetime experiences can influence individuals' level of functioning. It is important to highlight that current research does not suggest that cognitive reserve prevents neuropathology from developing. Rather, it is believed that a lifetime spent engaging in stimulating activities, such as education, occupation, exercise, or social involvement, may provide buffers within neural networks to help delay the behavioral symptoms associated with brain pathology.

Future Directions

An important implication of this concept may be the development of interventions that aim enhances cognitive reserve. In aging research, for instance, growing interest has revolved around the effectiveness of brain "training" games, exercise, or lifestyle modifications that may strengthen and/or expand neural networks, and improve cognitive functioning.

Cross References

- ▶ Brain Plasticity
- ▶ Brain Reserve Capacity

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Cognitive Robots

- ▶ Cognitive Correctors

Cognitive Stimulation

- ▶ Reality Orientation

Cognitive Trainers

► Cognitive Correctors

Cognitive-Behavioral Modification

► Behavior Modification

Cognitive-Communication Disorder

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Synonyms

Cognitive-communication impairment; Language of confusion; Language of generalized intellectual impairment; Right hemisphere impairment/disorder

Short Description or Definition

The American Speech-Language-Hearing Association (ASHA) has defined cognitive-communication disorders (CCDs) as those that, "...encompass difficulty with any aspect of communication that is affected by disruption of cognition. Communication may be verbal or nonverbal and includes listening, speaking, gesturing, reading, and writing in all domains of language (phonologic, morphologic, syntactic, semantic, and pragmatic). Cognition includes cognitive processes and systems (e.g., attention, perception, memory, organization, executive function). Areas of function affected by cognitive impairments include behavioral self-regulation, social interaction, activities of daily living, learning and academic performance, and vocational performance. Cognitive-communication disorders may be congenital or acquired. Congenital etiologies include but are not limited to genetic disorders and pre, peri, and postnatal neurologic injuries and diseases. Acquired etiologies include but are not limited to stroke, brain tumor, traumatic brain injury, anoxic or toxic encephalopathy, and nondegenerative and degenerative neurologic diseases (including the dementias)" (ASHA, 2005).

CCDs were recognized in the early 1980s as research mounted, illustrating the interdependency of cognition and language, particularly with regard to the roles of attention and memory in language processing and with regard to the pervasive impact that cognitive impairments have on functional communication abilities (Bayles & Tomoeda, 2007; Myers & Blake, 2008; Ylvisaker, Szekeres, & Feeney, 2008). Speech-language pathologists (SLPs) now routinely assess and treat those aspects of cognition that either support, or are influenced by, speech, language, and communication; other clinicians such as neuropsychologists, rehabilitation psychologists, and cognitive remediation specialists may do so as well.

Categorization

CCDs are differentiated from linguistic impairments (aphasias) and from motor speech disorders (dysarthrias and apraxia of speech) by the use of overly concrete, poorly organized, and socially insensitive communication despite preserved speech and language skills. CCDs may be caused and/or complicated by impairments of attention, memory, executive functions, and pragmatics. Symptoms will vary by etiology, patterns of brain damage, and individual differences in the neural organization of cognitive functions.

Epidemiology

Approximately 1.4 million individuals will suffer stroke-related CCDs each year (CDC, 2007; Tomkins, 1995). One to two million children will acquire post-traumatic CCDs annually (Blosser & DePompei, 2003; CDC, 2001; Ylvisaker et al., 2008), whereas adult and elderly individuals will sustain head injuries at rates approximating 100–200 per 100,000, respectively (Bruns & Hauser, 2003). The number of individuals expected to be living with dementia of the Alzheimer's type (DAT), and thus living with CCDs, is expected to increase from 4 million (in the year 2000) to 31.2 million people by the year 2050.

Natural History, Prognostic Factors, and Outcomes

The natural history and prognosis for improvement of cognitive-communication dysfunction are tied to many factors, including etiology, initial severity of disorder,

the presence or absence of comorbid illnesses, and the nature of specific individual variables such as age, gender, and psychological state. In the absence of confounding circumstances, improvement of CCD is anticipated when caused by stroke, excisable tumor, remitting disease, or traumatic brain injury. When caused by progressive debilitating conditions such as non-excisable tumors or dementing diseases, CCD will worsen over time. CCD of greater initial severity has a poorer prognosis than CCD of mild or moderate initial severity, although in the case of recovery from TBI, the degree of functionality at hospital discharge may be more predictive than the initial severity of injury (Testa, Malec, Moessner, & Brown, 2005). The presence of co-occurring illnesses may compromise the speed and extent of recovery from CCD at any severity level; CCDs arising from traumatic injuries causing diffuse brain damage, for example, are likely to be accompanied by paralysis, motor speech disorders, and injuries to vital organs.

Recovery from CCD differs by etiology. Recovery after stroke is usually most rapid in the first 3 months post onset. Recovery from thrombo-embolic stroke may continue after 6 months post onset, whereas recovery from hemorrhagic stroke may plateau at the 6-month point. Functional improvement in cognitive-communication abilities after severe traumatic brain injury is generally slower at the outset when compared with stroke and typically proceeds in a stairstep fashion over months or years. Recovery from mild stroke or brain injury often seems rapid in comparison. Many individuals with mild brain injuries appear to recover quickly (Ylvisaker et al., 2008), but as many as 15–20% suffer from persistent fatigue and reduced information processing speed for years after injury. Frequently unrecognized and untreated, these deficits cause lifelong cognitive challenges that threaten social adjustment and successful community reentry. In contrast to stroke and TBI, recovery from dementing illness is not expected. Early stages of cognitive decline in DAT are accompanied by forgetfulness, word-finding difficulties, and changes in social pragmatics; mid stages are characterized by increased memory loss, anomia, and social withdrawal; and late stages are associated with loss of most useable cognitive and physical functions (Bayles & Tomoeda, 2007).

Neuropsychology and Psychology of Cognitive-Communication Disorder

Damage to the prefrontal and frontal association regions of the right (or nonlanguage-dominant) hemisphere of the cerebrum may cause difficulty with pragmatics, context-sensitive semantics, and expressive affective prosody, obscuring indications of mood and compromising the ability to communicate successfully in social situations. Egocentrism may impair recognition of these problems and reduce insight into the communication needs of others. Deficits in vigilance, sustained and selective attention, and/or in attention switching can cause salient information to be missed, and reduce the sequential or simultaneous processing of information from multiple sources. Difficulties drawing inferences, extracting themes and topics in discourse, interpreting nonliteral language, and/or reading affective and prosodic cues for meaning during conversational exchanges may be present (Ylvisaker et al., 2008). A tangential communication style often emerges, where excessive, vaguely relevant details are inserted inappropriately into narratives and discourse (Myers & Blake, 2008).

Damage to the right parietal association cortex and to the right parietal-temporal-occipital (PTO) cortex can lead to contralateral inattention, impeding reading, writing, and listening for stimuli in left hemispace. Lesions in PTO cortex can also cause visual-spatial perception and recognition deficits (including topographical and geographical agnosias), which can impair navigation in familiar environments if verbal mediation strategies are not used to compensate. Damage to secondary (superior temporal) association cortex may reduce the efficiency of auditory language processing if stimuli are complex or if they must be processed quickly. Damage may also impair the interpretation of affective prosody when produced by others. Lesions to prefrontal, parietal, and temporal cortex have been associated with anosognosia, the failure to recognize the existence or presence of illness and a problem that can reduce compliance with treatment activities (Myers, 2001; Myers & Blake, 2008; Tomkins, 1995).

When damage to the cerebral cortex is bilateral, deficits across multiple systems may interact to impair communication to different degrees. Bilateral cortical damage, for example, can cause impairments in the self-regulation of communicative behaviors that range from failure to organize discourse efficiently to failure to inhibit inappropriate utterances and actions. It can also diminish the ability to focus attention and memory so as to make them useful during conversational exchanges (Ylvisaker et al., 2008). However, if bilateral damage involves subcortical hippocampal structures, then fundamental disruptions of declarative (semantic, episodic, and lexical) and explicit memory may occur. Hippocampal damage/deterioration is common with brain injury/dementing disease, and it can lead to a host of

impairments ranging from difficulty learning new information to the presence of a severe, unremitting amnesia.

Bilateral damage to lower brain stem reticular activating circuits can severely compromise arousal, alertness, and awareness. This can lead to brief losses of consciousness or to intractable coma. When caused by dementing disease, coma is most likely followed by death. However, recovery from traumatic coma frequently leads to return of function through increasing levels of responsivity, communication, orientation, self-regulation, and cognitive integration. A common sequela of traumatic brain injury is a period of post-traumatic amnesia (PTA), that is, inability to form new memories of events happening after brain injury with disorientation to time, place, or person. In individuals who are verbal, confabulation may be present until disorientation and confusion diminish. As cognitive functioning improves, individuals with brain injuries will benefit from environmental structure and external direction to support increasingly purposive, flexible, and goal-oriented behavior.

Evaluation

Evaluation of a suspected CCD requires examination of cognition as it affects and interacts with skills of speech planning and execution, language comprehension and production, and pragmatic/discourse aspects of communication in everyday social contexts (Turkstra, Cohelo, & Ylvisaker, 2005).

Tests of cognitive-communication skills are used to evaluate the effects that deficits in attention, orientation, perception, memory, organization, and executive functions can have on communication. Examples of tasks frequently included in formal and informal assessments are provided for illustration:

1. Attention to left hemispace is frequently tested with line-bisection, cancellation, and drawing tasks as well as with more complex reading, writing, and listening tasks that require individuals to process communication stimuli from both right and left sides of body midline.
2. Inferencing abilities are tested by asking patients to interpret humor, to recognize indirect requests for actions, and to follow the themes of conversations.
3. Orientation is assessed by asking patients to respond to questions about time, place, and person.
4. Memory for facts, events, and procedures is evaluated with yes/no questions, narratives, and performance of familiar routines.
5. The ability to discriminate relevant from irrelevant detail and integrate disparate parts into a coherent whole can be evaluated with scene description tasks.
6. Cognitive flexibility and functional problem-solving abilities can be assessed with tasks that require the generation of multiple strategies for achieving a goal and that require repairs of failed communicative interchanges with others (Myers, 1999).

Standardized tests used to assess cognitive-communication functions subsequent to right hemisphere impairment include the *Mini-Inventory of Right Brain Injury* (Pimental & Kingsbury, 1989), *The Rehabilitation Institute of Chicago Clinical Management of Right Hemisphere Dysfunction* (Halper, Cherney, & Burns, 1996), and *The Burns Brief Inventory of Communication and Cognition* (Burns, 1997). While these instruments will elicit symptoms of CCD associated with right hemisphere damage, findings must be interpreted with a view toward evaluating impairments of underlying neuropsychological processes, for it is here that treatment will be most profitably directed (Myers, 1999).

Standardized tests used to assess CCD after TBI include the *American Speech-Language-Hearing Association Functional Assessment of Communication Skills in Adults* (Frattali, Thompson, Holland, Wohl, & Ferketic, 1995), the *Behavioral Rating Inventory of Executive Function* (Roth, Isquith, & Gioia, 2005), the *Brief Test of Head Injury* (Helm-Estabrooks & Hotz, 1991), the *Ross Information Processing Assessment-2* (Ross-Swain, 1996), and the *Scales of Cognitive Ability for Traumatic Brain Injury* (Adamonovich & Henderson, 1992). The *Glasgow Coma Scale* (Jennett & Teasdale, 1981) and the *Rancho Los Amigos Levels of Cognitive Function Scale* (Hagen, Malkmus, Durham, & Bowman, 1979) or its revisions, including a version adapted for children under 14 years of age (Blosser & DePompei, 2003), are frequently used to assess consciousness and track recovery of cognitive functions after coma.

Informal situational assessments of cognition and communication may yield valuable information that cannot be obtained from formal standardized tests (Blosser & DePompei, 2003; Turkstra et al., 2005). This naturalistic approach to assessment has been termed, "functional, collaborative, context-sensitive, hypothesis-testing assessment," and it should be conducted with the purpose of identifying situational variables that can be manipulated to improve the successful participation of injured individuals as they operate within their everyday environments (Ylvisaker et al., 2008). Best-practice assessment procedures for individuals who have suffered brain injuries

should, therefore, include (1) completion of formal testing in specific skill domains and (2) completion of observational checklists (or quality of life inventories) where children (or adults) may be evaluated in natural environments (Blosser & DePompei, 2003; Turkstra et al., 2005). This type of assessment might include administration of the *Functional Assessment of Verbal Reasoning and Executive Strategies* test (MacDonald, 2005), a tool that was developed for evaluation of cognitive-communication skills specifically related to reading, writing, and reasoning (Turkstra et al., 2005), and the *Quality of Communication Life Scale* (Paul et al., 2005).

Assessment of CCD in dementia employs standardized screening tests, severity staging instruments, and comprehensive assessment batteries (Hopper & Bayles, 2008). Screening tests include the *Story Retelling Subtest of the Arizona Battery for Communication Disorders of Dementia* (Bayles & Tomoeda, 1993) and the *FAS Verbal Fluency Test* (Borkowski, Benton, & Spreen, 1967). Tests for estimating the severity of cognitive decline include the *Mini-Mental State Examination* (Folstein, Folstein, & McHugh (1975)) and the *Global Deterioration Scale* (Reisberg, Ferris, deLeon, & Crook (1982)). Comprehensive assessment batteries include the *Arizona Battery for Communication Disorders of Dementia* (Bayles & Tomoeda, 1993) and the *Functional Linguistic Communication Inventory* (Bayles & Tomoeda, 1994), the latter being most useful for individuals with severe dementia (Hopper & Bayles, 2008). Assessment findings can help families work with SLPs to understand and compensate for the symptoms of cognitive decline.

Treatment

Treatment for CCD has traditionally been decontextualized (implemented in rehabilitation settings or at bedside) and deficit oriented (designed to improve/support impaired cognitive-communication processes). Conventional pencil/paper or computer tasks are used to stimulate underlying cognitive processes (e.g., attention, memory) with the expectation that as they improve, so will the related functional skills (e.g., readiness to listen to speakers taking turns, remembering a set of instructions given by one's boss). Myers (1999) advocates the use of decontextualized treatments to *facilitate* cognitive-communication skills in individuals who have acquired CCD from right hemisphere stroke. She argues that although the value of any treatment approach "rests on its functional merits" (p. 209), improving fundamental cognitive processes will have the greatest automatic generalization to the many untrained tasks where those processes are needed.

Generalization may be more difficult to achieve when CCD is associated with diffuse brain injury versus focal stroke. Rehabilitation approaches that are more contextualized (implemented in natural settings) and more function oriented (designed to support activities of daily living) than traditional methods are ideal for promoting rapid skill mastery and transfer. Ylvisaker et al. (2008) suggest that treatment strategies designed to *compensate* for impaired executive functions and self-regulation abilities will most often lead to successful rehabilitation after brain injury. They advocate practicing essential cognitive-communication tasks (such as conversing with family members or taking notes during lectures) within supportive real-world environments, where stimuli likely to trigger errors have been removed and where the use of external aids is encouraged.

Intervention for CCD in DAT emphasizes management rather than rehabilitation. Goals are designed to help individuals maintain functional competence for as long as possible. Decontextualized errorless learning activities that drill attention and memory can bolster the encoding and retrieval of factual information in early stages of dementia. Intervention tasks that employ external memory aids that recruit long-term (remote) memory (such as reminiscing about past events) and that draw on procedural memory for familiar routines (such as describing how to get home) can help promote social communication and safety while capitalizing on those aspects of cognition that are available in mid-stage dementia. Tangible sensory stimuli (including dolls and stuffed animals) can help support communication and reduce agitation as dementia advances (Bourgeois, 1990, 1991, 1992; Hoerster, Hickey, & Bourgeois, 2001; Hopper & Bayles, 2008).

Cross References

- ▶ Attention
- ▶ Brain Injury
- ▶ Cognitive Functioning
- ▶ Cognitive Processing
- ▶ Cognitive Rehabilitation
- ▶ Dementia
- ▶ Executive Functioning
- ▶ Functional Neuroanatomy

- ▶ Memory Impairment
- ▶ Mild Brain Injury
- ▶ Mild Cognitive Impairment
- ▶ Moderate Brain Injury
- ▶ Pragmatic Communication
- ▶ Severe Brain Injury

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Cognitive-Communication Impairment

- ▶ Cognitive-Communication Disorder

Cognitive-Log

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Synonyms

C-Log; Cog-Log; Cognitive Log

Description

The Cog-Log is a ten-item scale designed for serial bedside measurement of cognitive functions in individuals completing inpatient rehabilitation. The scale includes items assessing orientation, immediate and delayed verbal recall, concentration, executive function, response inhibition, and praxis. All responses are scored according to the following criteria: 3 points = spontaneously correct response, no errors; 2 points = correct on logical cueing (e.g., "That was yesterday, so today is?") or in the presence of 1 error; 1 point = correct on multiple-choice cueing or in the presence of 2 errors; 0 points = incorrect despite cueing, more than 2 errors, unable to complete. Points for time estimation are calculated as follows: 3 points = correct within ± 5 s; 2 points = correct within ± 10 s; 1 point = correct within ± 15 s. Incorrect/absent responses are followed by cueing at the next level for the orientation and delayed memory items. Multiple-choice cueing, used only with the orientation items, involves provision of three choices, varying the location of the correct response. Item scores are summed to provide a total score ranging from 0 to 30. Daily scores can be plotted to permit quick visual analysis of recovery trends.

The following specific items are included in the Cog-Log: three items assessing the orientation to the date, time, and hospital name; two items assessing immediate and delayed recall for a short address; counting backwards from 20; reciting the months in reverse order; and estimating when 30 s has passed. Two motor tasks involving hand gestures – a movement sequence (fist-edge-palm) and a response inhibition task (go/no-go) – are also included.

Historical Background

Measurement of orientation and higher neurocognitive processes are important aspects of early neurorehabilitation. Reliable serial assessment is crucial to document the rapid changes in behavior and cognition during recovery from acquired brain disorders. Therefore, there is a need for a brief, bedside evaluation instrument to assess other areas of cognition that are frequently affected by acquired brain injuries. Existing scales are typically too lengthy or involved to present as part of morning rounds. Other scales fail to adequately capture the primary limitations resulting from acquired brain injury. The brief scales that have been created for serial administration in an inpatient setting have been presented without adequate psychometric properties or scaling. The Cog-Log was created to serve as a brief bedside measure to chart neurocognitive recovery and assist in planning rehabilitation interventions with a wide variety of patients.

Psychometric Data

The reliability and validity of the Cog-Log have been assessed in several ways. A sample of 150 individuals with acquired brain injury was examined with the Cog-Log. Most of the sample (80%) had sustained moderate to severe TBI, with the remainder including patients with CVA and anoxia. Internal consistency analysis (Cronbach's alpha) was conducted for the total Cog-Log score. Inter-rater reliability estimates (Spearman's rho) were calculated using a subset of 19 patients (75 total observations). High internal consistency (Cronbach's alpha = 0.778) was demonstrated with a standard error of measurement of 0.53. Inter-rater reliability coefficients for each of the ten Cog-Log items ranged from 0.749 (Go/No-go task) to 1.0 (Time Estimation). Standard errors of measurement were no more than 0.10 for single item scores, which range from 0 to 3.

Factor analysis of the Cog-Log using principal components extraction (N = 150) revealed a unitary factor (Eigenvalue = 3.48), with all items loading on that factor. The highest loadings were for delayed recall of verbal information and recitation of months backwards, suggesting a stronger contribution of complex working memory and verbal recall to this unitary factor.

The Cog-Log exhibited a significant correlation with neuropsychological assessment completed on the same day, including tests such as immediate and delayed recall of the Wechsler Memory Scale-III Logical Memory subtest, Rey Auditory Verbal Learning Test, Digit Span subtest of the Wechsler Adult Intelligence Test-III, and the Trail Making Test. The lowest Cog-Log score obtained during acute rehabilitation also significantly predicted 1-year outcome in three of six neuropsychological domains (attention, executive functioning, and visuospatial abilities) after

controlling for demographics and injury severity. The Cog-Log was also significantly correlated with the results of the Mini Mental State Examination, a well-known cognitive screening test ($r = 0.75$, $P < 0.001$).

Clinical Uses

Individuals without known neurological insult received average total Cog-Log scores of 28 ($\pm$ 2), and mean individual item scores were greater than or equal to 2.4. Age, education, and gender did not predict total or individual item scores ($p > 0.05$). Stepwise discriminant analyses on a sample of 82 persons with brain injury and 82 normal controls matched for age, education, and gender revealed that a cut-off score of 25 correctly classified 88.4% of individuals in their respective groups.

The Cog-Log is a companion instrument to the Orientation Log (O-Log). Generally, the Cog-Log is not administered until a score of at least 15 is achieved on the O-Log, indicating that the person is responding and able to respond correctly to some orientation questions. Administering the Cog-Log prior to this point has not proven fruitful. The Cog-Log can be administered every day, but typically three times a week is sufficient to monitor progress or detect deterioration.

Efficiency and ease of assessment were considered when choosing items; tasks requiring additional stimuli (e.g., block construction) or extended administration times were not included. The Cog-Log was designed for flexible administration to patients with severe cognitive and behavioral disturbances. Administration time ranges from 7 to 10 min for confused patients, but can be as short as 5 min for those who perform well.

Cross References

- ▶ Cognitive Functioning
- ▶ Galveston Orientation and Amnesia Test
- ▶ Mini Mental State Examination
- ▶ Traumatic Brain Injury

References and Readings

Alderson, A. L., & Novack, T. A. (2003). Reliable serial measurement of cognitive processes in rehabilitation: The Cognitive-Log. *Archives of Physical Medicine and Rehabilitation, 84*, 668–672.

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Lee, D., LoGalbo, A. P., Baños, J. H., & Novack, T. A. (2004). Prediction of cognitive abilities one year following TBI based on cognitive screening during rehabilitation. *Rehabilitation Psychology, 49*, 167–171.

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Collaborative Care

▶ Family-Centered Care

Collagen Vascular Disease

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Synonyms

Connective tissue disease

Definition

Collagen vascular diseases are a group of conditions that are characterized by malfunction of the tendons, bones, and connective tissue, that are supported by collagen. Their pathogenesis is autoimmune in nature.

Current Knowledge

The most common collagen vascular disorders include rheumatoid arthritis, systemic lupus erythematosus, scleroderma, and dermatomyositis. Others include polymyositis, polyarteritis nodosa, ankylosing spondylitis, and a number of vasculopathies. These diseases are frequently associated with diffuse inflammatory changes, abnormal immunity. Vascular abnormalities that result from these conditions serve as frequent causes of various types of vasculitis. Common features include arthritis, skin changes, eye changes, pericarditis, pleuritis, myocarditis, nephritis, and vasculitis of the brain, peripheral nerves, or extremities. They also may have a variety of hematological changes causing clotting or bleeding, and a number of abnormal circulating blood proteins.

The cause of most of these diseases is unknown. Hereditary factors and deficiencies, autoimmunity, environmental antigens, infections, allergies, and antigen-antibody complexes in various combinations are probably involved.

Cross References

- ▶ Cerebral Angiitis
- ▶ Lupus Cerebritis
- ▶ Vasculitis

References and Readings

Klippel, J. H., Stone, J. H., Crofford, L. J., & White, P. H. (Eds.). (2008). *Primer on the rheumatic diseases* (13th ed.). New York: Springer.

Collapsed Lung

- ▶ Pneumothorax

Colliculi

- ▶ Inferior Colliculi

Color Agnosia

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Definition

Literally, a loss of *previous* color knowledge.

Current Knowledge

In pure color agnosia, patients have difficulty naming or pointing to named colors, despite relatively preserved color perception (i.e., retaining the ability to match colors or to identify the numbers on the Ishihara plates). They also have difficulty matching colors, either verbally or visually, to familiar colored objects (e.g., identifying the color normally associated with cherries, lettuce, or bananas).

Relatively rare, pure color agnosia must be distinguished from other disturbances of color perception and color naming (*color anomia*). In *color blindness*, the individual is unable to perceive or distinguish either certain colors or possibly all color. In the latter case, the world is seen in shades of black and white. While color blindness is usually congenital, it can also be acquired, a condition known as *central achromatopsia*. The latter is a perceptual deficit thought to result from lesions in the visual cortices (e.g., lingual gyrus and the occipitotemporal (fusiform) gyrus). In this disorder, the patient may have difficulty verbally naming a visually presented color, pointing to a color named by the examiner, or simply matching or sorting colored objects to others of a similar hue, yet still be able to indicate (name) the colors normally associated with common objects (e.g., the colors of the outside, inside, and seeds of a watermelon). In a milder form of this condition (*dyschromatopsia*), colors are described as "dull," "washed out," or "faded." In *color anomia*, the problem is not one of perceptions, but of language. The patient can perceive and match colors, but has difficulty naming specific colors or pointing to colors named by the examiner.

In the few published cases, lesions associated with color agnosia tend to occur in the left or bilateral occipitotemporal area.

Cross References

- ▶ Achromatopsia
- ▶ Color Anomia

References and Readings

Bauer, R. M., & Demery, J. A. (2003). Agnosia. In K. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (4th ed., pp. 236–295). New York: Oxford University Press.

Tranel, D. (2003). Disorders of color processing. In T. E. Feinberg & M. J. Farah (Eds.), *Behavioral neurology and neuropsychology* (pp. 243–256). New York: McGraw-Hill.

Color Anomia

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Definition

Anomia is the inability to name colors in the absence of a more global anomia associated with an aphasic disorder.

Current Knowledge

To be classified as a color anomia, the disorder should occur in the absence of problems with color perception or recognition (i.e., the patient should be able to match or sort colors). Two subtypes of the disorder have been identified. In one, the problem is limited to an inability to name colors that are visually presented or to point to colors named by the examiner. This type of color anomia is usually associated with the syndrome of alexia without agraphia and results from lesions involving the primary visual cortex of the dominant hemisphere (resulting in a right homonymous hemianopsia) and the splenium of the corpus callosum. Visual information is thus restricted to the left visual field (right hemisphere) and the color information cannot cross the involved splenium of the corpus callosum to reach the left (verbal) hemisphere. In the second subtype, *specific color anomia*, the patient has difficulty with purely verbal color naming tasks, in addition to difficulty in naming visually presented colors. Thus, there would be a naming deficiency if the patient were asked to name the colors associated with the inside and outside of a watermelon. As in the first case, color matching or sorting should be intact. In specific color anomia, other aphasic (naming) deficits may be present, but color names are most affected.

Cross References

► Alexia Without Agraphia
► Color Agnosia

References and Readings

Bauer, R. M., & Demery, J. A. (2003). Agnosia. In K. Heilman, & E. Valenstein (Eds.), *Clinical neuropsychology* (4th ed.) (pp. 236–295). New York: Oxford University Press.
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Color Blindness

► Achromatopsia

Color Imagery

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Definition

The ability to visualize a color in its absence. When asked, most individuals would be able to identify the outer color of a watermelon as well as that of the inside of the rind, the fleshy part of the melon, and its seeds. Color imagery is more than an ability to simply recall a particular visual image; it also involves the capacity to mentally conjure up and manipulate colors at will. For example, one may imagine a blue horse or a person wearing an article of clothing of a specific color, never having seen either before. While disturbances of color perception and color imagery are frequently linked, in some cases the two can be distinguished clinically. Thus, while a given patient may be able to name or match colors presented visually, that same patient may not be able to name the color of an apple in its absence. Similarly, while accurately identifying the color red from an array, the patient may not be able to match it to a black-and-white picture of the fruit, although the latter may be identified by its shape. While the exact anatomical site responsible for disturbances of color imagery has not been firmly established, the left temporal-occipital cortex is believed to be involved in most cases.

Cross References

- ▶ Apperceptive Visual Agnosia
- ▶ Associative Visual Agnosia
- ▶ Color Agnosia
- ▶ Color Anomia

References and Readings

Farah, M. J. (2003). Disorders of visual-spatial perception and cognition. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (4th ed., pp. 146–160). New York: Oxford University Press.

Tranel, D. (2003). Disorders of color processing. In T. E. Feinberg & M. J. Farah (Eds.), *Behavioral neurology and neuropsychology* (2nd ed., pp. 243–256). New York: McGraw-Hill.

Colored Progressive Matrices

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Description

- The colored progressive matrices (CPM) are an alternate form of the Raven's progressive matrices (RPM) that was published in the 1940s. Shorter and simpler than the original, this version was designed for younger children (ages 5–11 years), the elderly (over 65 years), and people with moderate or severe learning difficulties. As such, it also tends to be used more frequently in research protocols, although it is important to note that the CPM and the RPM are not interchangeable, nor may derived scores from the two tests be interpreted the same. CPM contains 36 items, grouped into three sets (A, Ab, B) of 12 items each: A and B from the original version, with the addition of set Ab. Most items are presented on a colored background to make the test visually stimulating for participants; the bright background does not seem to detract from the clarity of the stimuli. As the last few items in set B are exactly the same as they appear in the standard version, an examinee who succeeds on these may go on to Sets C, D, and E of the standard progressive matrices (SPM) so that intellectual capacity can be more accurately assessed (the score for set Ab would be omitted to determine a percentile based on SPM scoring).

Historical Background

For information about the historical background of the original test, please refer to ▶ Raven Progressive Matrices.

Psychometric Data

Norms for ages 5.5–11.5 for North America are presented by Raven, Raven and Court (1998, 2000). By age 9 years, a nearly perfect score is obtained (35/36) by the upper 5% of the sample. Education corrected norms are available for an abbreviated version (sets A and B, excluding Ab which is correlated >0.90 with the sum of A and B) for ages 55–85. In children, split-half reliability has been shown to be high (>0.80) (Raven et al. 1998), as is test-retest reliability following days or weeks (>0.80). Over longer intervals (6 months to 1 year) these values decline (0.59–0.79).

Clinical Uses

The CPM was designed for use with children, older people, for anthropological studies, and for clinical work. Its format makes it valuable for individuals who cannot understand English; people with physical disabilities, aphasias, cerebral palsy, or deafness; and people with below normal intelligence. The colored backgrounds were introduced to attract the patient's attention; the test can be administered in the form of illustrations in a booklet or as boards with moveable pieces. Patients with left hemisphere damage perform better on the colored matrices than on the standard form (as described in Lezak, Howieson, & Loring, 2004). This is likely attributable to the fact that while only one-fifth of RPM items test visuospatial skills almost exclusively, fully one-third of items on the CPM are predominantly visuospatial, with other items involving more problem-solving. Also likely due to the visuospatial task demands, individuals with Lewy body dementia tend to have more difficulty on the CPM than Alzheimer's patients with similar levels of dementia.

Cross References

- ▶ Advanced Progressive Matrices
- ▶ Raven's Progressive Matrices
- ▶ Standard Progressive Matrices

References and Readings

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

Raven, J. C. (1938, 1996). Progressive Matrices: A perceptual test of intelligence. Oxford: Oxford Psychologists Press Ltd.

Raven, J., Raven, J. C., & Court, J. H. (1998). *Raven manual: Section 2. Colored progressive matrices.* Oxford: Oxford Psychologists Press Ltd.

Raven, J., Raven, J. C., & Court, J. H. (2000). *Raven manual research supplement 3: American norms, neuropsychological applications.* 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.

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Coma

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Definition

Coma is a state of unconsciousness in which the patient is incapable of being awake, and is unarousable, even with vigorous stimulation. Coma is usually the result of disease or injury, and rarely lasts for more than 4 weeks. While comatose, a patient may respond to painful stimuli but lack the ability to demonstrate localized response or defensive movements (Posner, Saper, Schiff, & Plum, 2007).

Current Knowledge

Diagnosis

The diagnosis of patients in coma necessarily involves examination of physiological functions, including arousal, pupillary responses, respiration, motor function, and reflexes. Several diagnostic scales are available for severity rating, and include the Glasgow Coma Scale (Teasdale & Jennett, 1976) and the FOUR Score (Wijdicks, Bamlet, Maramattom, Manno, & McClelland, 2005).

Prognosis

Mortality rates for patients in coma vary with etiology, but commonly reach or exceed 50%. Early prognostic variables of poorer outcome include lower Glasgow Coma Scale scores (Teasdale & Jennett, 1976), increased age, absent pupillary responses, systolic blood pressure <90 mm Hg, and computed tomography abnormalities, including compression, effacement, or blood within the basal cisterns, or extensive traumatic subarachnoid hemorrhage (Posner et al., 2007).

Cross References

- ▶ Decerebrate Posturing
- ▶ Decorticate Posturing
- ▶ Glasgow Coma Scale
- ▶ Loss of Consciousness
- ▶ Minimally Conscious State
- ▶ Stupor
- ▶ 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.

Teasdale, G., & Jennett, B. (1976). Assessment and prognosis of coma after head injury. *Acta Neurochirurgica, 34*, 45–55.

Wijdicks, E. F. M., Bamlet, W. R., Maramattom, B. V., Manno, E. M., & McClelland, R. L. (2005). Validation of a new coma scale: The FOUR score. *Annals of Neurology, 58*, 585–593.

Coma Recovery Scale

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Synonyms

CRS; CRS-R; JFK coma recovery scale

Definition

The Coma Recovery Scale (CRS-R) is a 23-item instrument used to assist with differential diagnosis, prognostic assessment, and treatment planning with patients having disorders of consciousness.

Current Knowledge

The Coma Recovery Scale was initially developed by Giacino and colleagues in 1991 and thereafter was revised in 2004 as the JFK Coma Recovery Scale-Revised (Giacino, Kalmar, & Whyte, 2004). The scale was developed with the goal of helping to identify the neurobehavioral characteristics of persons diagnosed with disorders of consciousness, thus allowing for increased prognostic clarity and refinement of treatment methods. In its revised form, the CRS-R is composed of 23 items, which coalesce into six subscales addressing auditory, visual, motor, oromotor, communication, and arousal functions. Each subscale is composed of hierarchically arranged items associated with the brain stem, subcortical, and cortical processes. Items receiving higher scores are more likely to reflect cognitively mediated activity. The test syllabus offers behavioral criteria, which provide the user with operationalized characteristics used to determine whether a specific response to sensory stimuli has been demonstrated by the patient. A rating form is included as part of the syllabus, which allows the user to perform serial assessments.

Based on the 2004 study by Giacino, Kalmar and Whyte, interrater and test–retest reliability were described as high for CRS-R total scores, although some systematic scoring differences between raters were found on the visual and oromotor/verbal subscales. The CRS-R has been translated into Spanish, Italian, German, French, Dutch, and Norwegian. The CRS-R offers a reliable method for measuring the trajectory of consciousness following severe brain injury, and in assisting in the differential diagnosis of patients in a minimally conscious state from those in a vegetative state.

Cross References

► Coma/Near Coma Scale

References and Readings

Giacino, J. T., Kalmar, K., & Whyte, J. (2004). The JFK coma recovery scale- revised: measurement characteristics and diagnostic utility. *Archives of Physical Medicine and Rehabilitation, 85*(12), 2020–2029.

Kalmar, K., & Giacino, J. (2005). The JFK coma recovery scale-revised. *Neuropsychological Rehabilitation, 15*(3–4), 454–460.

The CRS-R, including administration and scoring information and rating forms, is available as a PDF file at www.tbims.org/combi/crs/index.html.

Coma Vigile

► Vegetative State (Persistent)

Coma/Near Coma Scale

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Synonyms

C/NC; CNC

Description

The Coma/Near Coma (CNC) Scale was designed to measure small changes in neurobehavioral status among patients who have sustained severe brain injuries and whose clinical functioning is consistent with vegetative or near-vegetative states. The CNC Scale is useful when reliable, valid, and economical assessment of patient's functioning is required so as to substantiate clinical change or lack thereof in patients whose functioning is at markedly low levels. The CNC Scale expands the levels of the Disability Rating Scale (DRS). It has five levels and is composed of 11 items. The items assess dysfunction in the sensory and perceptual dimensions and describe the severity of primitive response deficits.

Historical Background

Recognizing a need for rehabilitation professionals to systematically identify and chart changes in functioning in patients whose clinical status is best characterized as approximately "vegetative," the CNC Scale was developed as a supplement to the Dementia Rating Scale (DRS). Use

Coma/Near Coma Scale. Table 1 Coma/near coma scale overview

Item #	Dimension assessed	Stimulus used	Response elicited
1	Auditory	Ringing bell	Eye opening/orientation
2	Command Responsivity	Verbal request	Response to command
3	Visual	Flashing light	Fixation or avoidance
4	Visual	Say "Look at me"	Fixation & Tracking
5	Threat	Quick movement of hand to eyes	Eye blinking
6	Olfactory	Ammonia capsule	Withdrawal or grimacing
7	Tactile	Shoulder tap	Head/eye orientation or shoulder movement
8	Tactile	Swab to each nostril	Withdrawal/eye blink or mouth twitch
9	Pain	Firm pinch to finger tip	Withdrawal or agitation
10	Pain	Firm ear pinch	Withdrawal or agitation
11	Vocalization	None; listen for any verbalization	Words or sounds

of the CNC Scale is recommended whenever the DRS score is greater than 21 (Extremely Severe Disability). The scoring form also notes that in cases in which DRS scores are < 21, CRC Scale ratings should be conducted monthly in conjunction with the DRS.

Current Knowledge

As described by Rappaport, Dougherty, and Kelting (1992) and as detailed by the Center for Outcome Measurement in Traumatic Brain Injury (COMBI; www.tbims.org/combi/cnc/index.html), interrater reliability calculated at three time periods was 0.97. The lowest interrater intercorrelation of 0.86 was found to exist on item 8 (nasal swab). The internal consistency as determined using alpha coefficients were 0.43, 0.65, and 0.65 for CNC Scale scores taken at 1, 8, and 16 weeks post injury, respectively (Rappaport, Dougherty, & Kelting, 1992).

A rank order correlation between CNC Scale and DRS scores was 0.69 ($p < 0.02$). The correlation between CNC Scale ratings and the prominence of sensory (auditory, visual, and somatosensory) abnormality as determined through evoked potential studies was 0.52 ($p < 0.05$) (Rappaport, Dougherty, & Kelting, 1992).

Discussion regarding training in the use of the CNC Scale is offered by COMBI (www.tbims.org/combi/cnc/cnctat.html). It is recommended that proper use of the instrument follows a period during which evaluations are performed simultaneously with several raters independently offering judgments for each item. Interrater comparisons and discussion concerning the reasons for making specific judgments are encouraged to refine observational accuracy. Following this training, it is noted that while single ratings can be used, "a minimum of two independent ratings per patient is encouraged" for purposes of promoting reliability (Rappaport, 2000). It is also recommended that rating be taken at approximately the same time each day whenever possible.

The CNC Scale includes 11 items representing 8 response dimensions (see Table 1). Each item includes 3 score options (0, 2, 4); 4 referring to "no responsivity to sensory stimulation." The CNC Total Score is computed by adding the ratings (0, 2, 4) from each of the 11 items and dividing that score by the number of items rated. Categorical descriptions are offered to coincide with the Total Score attained (i.e., No Coma through Extreme Coma).

Cross References

- ▶ Coma Recovery Scale
- ▶ Dementia Rating Scale

References and Readings

Rappaport, M. (2000). The *coma/near coma scale. The center for outcome measurement in brain injury.* Accessed 1/6/08 from www.tbims.org/combi/cnc.

Rappaport, M. (2005). The disability rating scale and coma/near coma scales in evaluating severe head injury. *Neuropsychological Rehabilitation, 15*(3/4), 442–453.

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Commission on Accreditation of Rehabilitation Facilities (CARF)

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Membership

Commission on Accreditation of Rehabilitation Facilities (CARF) is an independent, nonprofit organization that is an accreditor of human service providers. It was formed in 1966 when the Association of Rehabilitation Centers (ARC) and the National Association of Sheltered Workshops and Homebound Programs (NASWHP) joined forces to further develop their interests in setting standards. Since its inception, CARF has grown significantly in size and informally adopted CARF International as its registered name after accrediting programs in Canada, Europe, and South America.

CARF International currently provides accreditation services in the following areas, called customer service units: aging services; child and youth services; behavioral health; durable medical equipment, prosthetics, orthotics, and supplies (DMEPOS); employment and community services; medical rehabilitation; and opioid treatment programs. The CARF family of organizations currently accredits more than 6,000 providers at more than 20,000 locations on five continents. More than 8.3 million persons of all ages are served annually by CARF-accredited providers. In 2002, CARF Canada was developed as a private, nonprofit organization serving Canadian providers. CARF acquired the Continuing Care Accreditation Commission (CCAC) in 2003. The CCAC provides accreditation of continuing care retirement communities and aging services networks. Financial support for CARF includes fees from accreditation surveys, sales of publications, grants from public and private entities, fees from seminars and conferences, and contributions from International Advisory Council (IAC) members.

CARF is governed by its President/CEO and a 12-member Board of Directors. The Board of Directors consists of individuals with expertise, experience, and perspective on CARF-related issues. Current CARF leadership includes individuals with a diverse range of expertise, including individuals with psychology, physical therapy, and speech-language pathology expertise, social work backgrounds, as well as past surveyors and individuals with business and legal training. Other perspectives on issues related to CARF-accreditation and other matters are gained through individuals and organizations that are members of CARF's International Advisory Council (IAC).

The International Advisory Council (IAC) creates a partnership for CARF and IAC members to promote quality in human services and enhance the lives of persons served. It also provides a forum for guidance and input into standards development and the accreditation process and insight on issues affecting the fields in which CARF provides accreditation services. IAC members support CARF's mission, purposes, values, and vision. The IAC is composed of organizational and individual members who represent a broad spectrum of stakeholders, including persons served, providers, professionals in the field, and purchasers. Current members of the IAC include American Academy of Neurology, American Academy of Orthopedic Surgeons, American Academy of Pain Medicine, American Academy of Physical Medicine and Rehabilitation, American Occupational Therapy Association, Inc., American Physical Therapy Association, American Psychological Association, American Speech-Language-Hearing Association, American Therapeutic Recreation Association, and the U.S. Psychiatric Rehabilitation Association, in addition to many other established agencies and associations documented on the CARF web site, http://www.carf.org/About/IAC/.

Major Areas or Mission Statement

The mission of CARF is "to promote the quality, value, and optimal outcomes of services through a consultative accreditation process that centers on enhancing the lives of the persons served" (retrieved June 29, 2010, from http://www.carf.org/About/Mission/). CARF has identified three core values as central to its mission, and these values are prioritized in all CARF accreditation, research, and educational activities. The following are the three CARF core values: (1) all people have the right to be treated with dignity and respect, (2) all people should have access to needed services that achieve optimum outcomes, and

(3) all people should be empowered to exercise informed choice.

In support of its mission, CARF has identified numerous purposes that are the focus of the agency, including the following (retrieved June 29, 2010, from http://www.carf.org/About/Mission/).

- To develop and maintain current, field-driven standards that improve the value and responsiveness of the programs and services delivered to people in need of rehabilitation and other life enhancement services.
- To recognize organizations that achieve accreditation through a consultative peer-review process and demonstrate their commitment to the continuous improvement of their programs and services with a focus on the needs and outcomes of the persons served.
- To conduct accreditation research emphasizing outcomes measurement and management, and to provide information on common program strengths as well as areas needing improvement.
- To provide consultation, education, training, and publications that support organizations in achieving and maintaining accreditation of their programs and services.
- To provide information and education to persons served and other stakeholders on the value of accreditation.
- To seek input and to be responsive to persons served and other stakeholders.

CARF also collaborates with partners who share CARF vision and goals, while remaining politically neutral. CARF partners include professional associations, advocacy groups, governmental agencies, or individuals who are committed to enhancing the lives of persons who receive services.

Landmark Contributions

One of the key developments in the history of CARF was the passing of a resolution by the Council of State Administrators of Vocational Rehabilitation in April 1970, which urged state agencies to support a CARF accreditation requirement for all organizations providing rehabilitation services. As CARF's services became increasingly recognized by organizations and agencies, additional support of CARF accreditation services has occurred. In November 1974, Goodwill Industries of America recognized CARF as the accrediting organization for all Goodwill organizations. Another resolution, adopted in November 1980 by the Association of Trial Lawyers of America, urged state workers' compensation agencies to require CARF accreditation for rehabilitation organizations providing services to workers with occupational disabilities.

CARF has also expanded the types of services for which it provides accreditation. In September 1997, CARF was awarded a contract by the Department of Health and Human Services, Substance Abuse and Mental Health Services Administration for the development and implementation of opioid treatment program accreditation. After successful development of the program accreditation, CARF was recognized in November 2001 by the Substance Abuse and Mental Health Services Administration (SAMHSA)/Center for Substance Abuse Treatment (CSAT) as an approved accrediting organization for opioid treatment programs. As noted previously, CARF acquired the Continuing Care Accreditation Commission (CCAC) in 2003. Acquisition of the CCAC promoted CARF's vision of being an independent resource that identifies high-quality care providers for individuals of all ages, including children and older adults. More recently, in February 2007, CARF began accrediting suppliers of certain durable medical equipment, prosthetics, orthotics, and supplies (DMEPOS) after the Centers for Medicare & Medicaid Services (CMS) approved CARF as a national authority for DMEPOS suppliers.

Over the decades, CARF has steadily grown and expanded its accreditation services to serve individuals in other countries. In 1969, CARF accredited its first program in Canada and the first program in Europe was accredited in August 1996. CARF Canada was incorporated in September 2002 in Edmonton, Alberta. Further expansion of services included accreditation of the first program in South America in December 2005. The value of CARF accreditation is extensive and includes benefits such as business improvement, risk management, funding access, positive visibility, accountability, peer network, in addition to other benefits detailed on the CARF web site.

Major Activities

CARF accreditation outcomes for CARF and CARF Canada consist of a three-year accreditation, a one-year accreditation, provisional accreditation, nonaccreditation, and preliminary accreditation. CARF-CCAC accreditation outcomes include a five-year accreditation and nonaccreditation. CARF currently has close to 1,500 surveyors throughout the North and South America and Europe.

The steps to accreditation involve a year or more of preparation prior to the site survey and ongoing quality improvement following the survey. CARF designates a CARF Resource Specialist to organizations seeking

accreditation to provide guidance and technical assistance regarding the accreditation process. Other resources are also available to facilitate the preparation process, including standards manuals for the various programs and services an organization can seek accreditation for and the CARF Accreditation Sourcebook, which explains the accreditation process. CARF accreditation requires that an organization must implement and use the standards in its programs for at least 6 months prior to the survey. During this time, an organization conducts a self-study and evaluation of conformance to the standards using the standards manual in conjunction with the survey preparation questions. Then the organization submits the Intent to Survey, which includes detailed information about leadership, programs, and services that the organization is seeking to accredit and the service delivery location(s). CARF selects a survey team by matching program or administrative expertise and relevant field experience with the organization's unique requirements. During the survey, the survey team determines the organization's conformance to all applicable standards on site through the observation of services, interviews with persons served and other stakeholders, and review of documentation. The survey team shares findings related to the standards with the organization at an exit conference before the team leaves the site and this information is also submitted to CARF. CARF then renders the accreditation decision and a written survey report approximately 6–8 weeks after the survey. Organizations have 90 days after notification of an accreditation award to fulfill an accreditation condition by submitting to CARF a Quality Improvement Plan (QIP) outlining actions that have been or will be implemented in response to the recommendations made in the survey report. CARF maintains communication with organizations throughout the tenure of accreditation.

CARF has published numerous standard manuals which have identified program evaluation standards, resulting in improved services and programs provided to clients receiving rehabilitation services. The first publication of a new section of standards manuals for Rehabilitation Facilities in November 1973 became a springboard for future CARF contributions to program evaluation. In January 1986, standards for two new program areas (Respite Programs and Alcoholism and Drug Abuse Treatment Programs) were published in the *1986 Standards Manual for Organizations Serving People with Disabilities*. The *1988 Standards Manual for Organizations Serving People with Disabilities* included new standards in areas of Post-Acute Brain Injury Programs and Community Mental Health Programs. CARF published a separate volume of its *1995 Standards Manual and Interpretive Guidelines* for each of its accreditation areas (Behavioral Health, Employment and Community Support, and Medical Rehabilitation). Then in 1996, CARF published the first edition of the *Accreditation Sourcebook*. A significant contribution to accreditation services occurred in 1998, when CARF rewrote its standards to be unidimensional as part of its commitment to implementing the Standards Conformance Rating System. Additional standards were published in 1999, including standard manuals for Adult Day Services Programs and the Veterans Health Administration Comprehensive Blind Rehabilitation Services. Moreover, that same year, CARF extended its practice of disclosing information to the public regarding an organization's survey report from just the *Medical Rehabilitation Standards Manual* to also include the *Behavioral Health Standards Manual* and the *Adult Day Services Standards Manual*. CARF's public information policy was extended to include all areas later in 2003. Standards manuals for assisted living programs were published in 2000, followed by the *Child and Youth Services Standards Manual* in 2005, the *Aging Services Standards Manual* in 2006, and Standards for Dementia Care in 2006. In addition to these publications, CARF has continued to implement new standards in many of its areas of accreditation.

CARF has implemented new electronic programs designed to support and further develop its mission. In 2007, CARF released the uSPEQ® (pronounced *you speak*; http://www.uspeq.org/) Consumer Experience Survey, followed by the uSPEQ Employee Climate Survey. Both survey tools are designed to assist organizations with their well-being and performance improvement. CARF launched its extranet Customer Connect (http://customerconnect.carf.org) for accredited organizations and those seeking accreditation to manage their individual contact information, view up-to-date information about their organization's surveys, and access information about the accreditation process. Most recently in 2008, CARF's ASPIRE to Excellence™ quality framework has been implemented in all standards manuals. This framework provides a logical, action-oriented approach to ensure that organizational purpose, planning, and activity result in desired outcomes.

Each year, CARF sponsors numerous in-person and web-based seminars to help providers maintain conformance to the CARF standards. The in-person seminars are held in cities across the United States, Canada, and Europe.

Cross References

- ▶ Americans with Disabilities Act of 1990
- ▶ Individuals with Disabilities Education Act

- ▶ National Center for the Dissemination of Disability Research
- ▶ National Dissemination Center for Children with Disabilities
- ▶ National Institute on Disability and Rehabilitation Research
- ▶ National Institute of Neurological Disorders and Stroke
- ▶ National Rehabilitation Information Center
- ▶ No Child Left Behind Act of 2001
- ▶ Rehabilitation Counseling
- ▶ Rehabilitation Psychology
- ▶ Section 504 of the Rehabilitation Act of 1973

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CARF overview (http://www.carf.org/About/).
CARF partners (http://www.carf.org/Resources/IACResources/).
CARF structure (http://www.carf.org/About/WhoWeAre/).
Value of CARF Accreditation (http://www.carf.org/Accreditation/ValueofAccreditation/).

Commissural Fibers

- ▶ Commissures, Cerebral

Commissural Magna

- ▶ Corpus Callosum

Commissures, Cerebral

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Synonyms

Brain commissures; Commissural fibers

Definition

The right and left cerebral hemispheres are connected by three tracts of nerve fibers or axons, which are collectively referred to as commissures, cerebral. These transverse, nerve fiber bundles connect to the homologous regions of each hemisphere and are known as the corpus callosum, the anterior commissure and the posterior commissure. The corpus callosum, also referred to as the great cerebral commissure, is the largest, and it interconnects the greatest portion of the cerebral hemispheres, permitting the cerebral cortex to operate as a whole. The corpus callosum is often the site for commissurotomy, surgical bisection, to treat certain psychiatric disorders and epilepsy. The anterior commissure is a smaller bundle of nerve fibers that interconnect parts of the temporal lobes. The posterior commissure is another fiber bundle that crosses beneath the pineal gland to connect the midbrain regions of the cerebral hemispheres.

Cross References

- ▶ Commissurotomy
- ▶ Corpus Callosum

Commissurotomy

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Synonyms

Split-brain

Definition

The term referring to the medical procedure in which interconnecting fibers between the cerebral hemispheres are lesioned. The anatomic location includes either or both anterior and posterior fibers. When posterior fibers are resected, connections to the hippocampus may also be affected.

Current Knowledge

The neurosurgical technique is based on the premise that when stimuli enters the brain it is rapidly communicated via the corpus callosum to the other hemisphere. In refractory epilepsy, severing the corpus callosum may prevent the spread of electrical activity between hemispheres and generalization of the seizure activity. This procedure prevents the communication of the two hemispheres thus resulting in a clinical scenario of a "split brain." Clinical disconnection syndromes in these patients have been studied across a large number of neurocognitive tasks. These surgical lesions have allowed for the study of sensory perceptual processes and lateralization. Deficits have been seen in the area of perception, attention, memory, language, and reasoning. In studies of these patients, it has become clear that the left hemisphere has limitations in perceptual functions while the right hemisphere has more striking limitations in cognitive functions. The split brain studies have also led to theories of consciousness and a neurocognitive framework for the human experience.

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Communicating Artery

▶ Anterior Communicating Artery

Communication Ability

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Definition

Commonly refers to the ease, efficiency, and accuracy of the exchange of information or content between parties regardless of form, i.e., spoken, written, gestured, drawn, or using augmentative and assistive devices (communication boards or computers). Human communication relies primarily on the preservation and maintenance of key social and interpersonal bonds (staying close and connected with individuals who matter most in daily life). Thus, effective communication depends as much on nonverbal behaviors such as facial expression, touch, and vocal intonation as on the exchange of words. Acquired deficits to communication ability can occur in the absence of a notable loss in intelligence or nonlinguistic cognitive functions, as in aphasia, dysarthria, dysphonia/aphonia, and apraxia of speech.

Cross References

▶ Language
▶ Speech

Communication Disorders

▶ Speech/Communication Disabilities

Community Adjustment

▶ Community Re-entry

Community Integration

▶ Community Re-entry

Community Integration Questionnaire

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Synonyms

CIQ

Description

The Community Integration Questionnaire (CIQ) measures what the International Classification of Disability, Functioning and Health (ICF) currently designates as

Participation. The CIQ consists of 15 items relevant to home integration (H) (living), social integration (S) (loving), and productive activities (P) (working). It is scored to provide subtotals for each of these domains, as well as a total community integration score. The basis for scoring is primarily frequency of performing activities or roles, with secondary weight given to whether or not activities are done jointly with others and the nature of these other persons (for example, with/without disability). The CIQ can be completed by either the index person or a proxy in 10–15 min. The optimal method of data collection is an in-person interview, but telephone interviewing is quite common, and the Traumatic Brain Injury Model Systems (TBIMS) have also utilized self-administered CIQs. No formal training and credentialing process for the administration of the CIQ exists.

Historical Background

The CIQ was developed in the early 1990s by Barry Willer, Ph.D. and a group of professionals and consumers to provide a measure of community integration after traumatic brain injury (TBI) that could be used in the TBIMS research program (Willer, Linn, & Allen, 1994; Willer, Ottenbacher, & Coad, 1994; Willer, Rosenthal, Kreutzer, Gordon, & Rempel, 1993). They used the following design criteria: brevity; suitable for use in in-person or telephone interviews with the person with TBI or a proxy; focus on behaviors rather than feeling states; no biases resulting from age, gender, or socioeconomic status; sensitive to a wide variety of living situations; and value neutral. The instrument has been translated (not always using formal backtranslation methods) into French, Spanish, Japanese, Korean, Dutch, Swedish, and Norwegian, among others. It is the most extensively used measure of community integration/ participation in research on TBI, but has also been applied in investigations of other types of brain injury (including stroke and brain tumors), spinal cord injury, burns, trauma in general, "mobility disabilities", and post-polio syndrome. The most common applications are in program evaluation and the study of the natural history of TBI and other impairments, but the CIQ has also been used in clinical trials of interventions to improve post-injury functioning, and in construct validation studies of newly proposed participation measures.

Because the assignment of items to subscales by Willer et al. was based on a correlation matrix calculated for a small sample, Sander et al. performed a factor analysis involving all CIQ items for a much larger sample. The resulting instrument, sometimes designated the CIQ-R, dropped one item and reassigned two to different subscales (Sander et al., 1999). The CIQ-R has been used infrequently. In 2002, The TBIMS dropped the CIQ, and replaced it (in 2007) with the Participation Assessment with Recombined Tools (PART).

Psychometric Data

Psychometric information for the CIQ is spread out over a number of sources. In addition to the three papers by Willer et al. (Willer, Linn, et al., 1994; Willer, Ottenbacher, et al., 1994; Willer, Rosenthal, et al., 1993) key texts are those by Corrigan and Deming (1995), Sander et al. (1999); Sander et al. (1997) and Tepper, Beatty and DeJong (1996). Dijkers critically summarized psychometric information available through 1997 (Dijkers, 1997), and some later sources are given in Reistetter and Abreu's 2005 article (Reistetter and Abreu, 2005).

Reliability. Results of reliability studies have been mixed. Based on the (Pearson) correlations reported in the earliest study, the *interrater reliability* of the CIQ appears to be in the "acceptable" range. However, the intraclass correlation coefficient (ICC), a more appropriate measure, resulted in much lower numbers, according to a later investigation, especially for the Home (H) dimension. More recent research also suggests that in home integration there is the greatest discrepancy between reports by subjects with TBI and those by their proxies. In the latter study, the person with TBI tended to report higher values than the proxy for all three components. Subscale ICCs ranged from 0.43 (H) to 0.81 (P) – fairly low values.

For the CIQ total score, three out of four studies have reported *internal consistency* levels that exceed the criterion of a coefficient alpha above 0.80. However, the corresponding values for the S and P dimensions are much lower, especially for the latter – quite likely due to the fact that alpha is based on only two variables, which are very dissimilar from one another: work/study/volunteering, and travel.

Distribution issues. Corrigan and Deming noted negatively *skewed distributions* for the premorbid data for CIQ total, positive skews for H for all four samples, negative skews for all samples for the S dimension, and negative skew for P for the TBI-premorbid sample, but positive skew for the three others. Various *kurtosis* problems were also noted. They recommended that the P subscale not be used independently from its contribution to the CIQ total score. Distribution problems were also

noted by Willer et al. and Sander et al., among others. Unless these can be resolved through transformations, nonparametric statistics need to be used in order to deal with the nonnormal distributions.

Validity. No formal *content or face validity* studies of the CIQ have been done, but it was developed utilizing a panel consisting of both consumers and professionals with expertise in TBI outcome studies.

CIQ subscales and total score have been found to correlate with subscales and total score on CHART (another measure of community integration), impairment and disability, time since injury, CIM (a measure of subjective community integration), and subjective quality of life, according to multiple reports. Most researchers find negative correlations between CIQ and its subtotals (except sometimes H) and age. Females tend to have higher CIQ scores for total, H and S than males, but lower P scores.

Sensitivity. The available research shows that the CIQ can validly distinguish between persons with TBI and nondisabled people. Persons with TBI are less integrated along all dimensions than nondisabled comparison groups in most research. In one study, CIQ scores distinguished between three groups of persons with TBI living in settings differentiated by supervision/support level: independent in the community, in the community with some (natural) support, and in an institution such as a nursing home, rehabilitation facility, etc. Willer et al. reported gain in CIQ (sub)scores for people with TBI receiving residential rehabilitation services between the second and third anniversary of injury, while a control group who received no or minimal home-based services did not show a gain. Corrigan et al. found trends toward improvement in all three subscales and total score in a cross-sectional study covering injury anniversaries one through four. However, in another investigation, a very small increase from the first to the second anniversary of injury was reported for a group that in large majority did not receive rehabilitation services any longer.

Clinical Uses

In several TBI community integration programs, the CIQ is used to monitor patients' progress. However, due to the lack of norms and the dependence of community functioning on the environment, the clinical applicability of the CIQ is very much limited.

Note

Barry Willer Ph.D., who was the principal investigator in developing the CIQ, holds the copyright. Permission for use of the CIQ is freely given but should be requested, by contacting him at the Center for Research on Community Integration at the Ontario Brain Injury Association, 3550 Schmon Parkway, Thorold, Ont L2V 4Y6, Canada, email: willer@vaxxine.com

Cross References

- ▶ CHART Short Form
- ▶ Community Re-entry
- ▶ Craig Handicap Assessment and Report Technique
- ▶ Frenchay Activity Index
- ▶ Glasgow Outcome Scale
- ▶ Glasgow Outcome Scale – Extended
- ▶ Impact on Participation and Autonomy Questionnaire
- ▶ Instrumental Activities of Daily Living (I-ADL)
- ▶ Lawton–Brody IADL scale
- ▶ LIFE-H
- ▶ Outcome, Outcome Measurement
- ▶ Participation Measure for Post Acute Care (PM-PAC)
- ▶ Participation Objective – Participation Subjective
- ▶ Reintegration to Normal Living Index
- ▶ Social and Occupational Functioning Scales (SOFAS)
- ▶ Traumatic Brain Injury Model System

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Community Re-entry

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Synonyms

Community adjustment; Community integration

Definition

Community re-entry is the extent that an individual who is initially unable to fully function in the community due to disability is eventually returned to the community to work and live independently, using natural supports and exercising full access, choice, autonomy, and striving for actualization. Actualization is the degree to which there is the achievement of a respectable quality of life.

The Americans with Disabilities Act (ADA) of 1990 attempted to legislate community integration by removing physical barriers related to access and making it illegal to discriminate on the basis of disability. However, 30 years since the ADA's passage and after the recent passage of the ADA Restoration Act in late 2008, there is still significant concern that United States society does not fully possess nor embrace the attitudes and values commensurate with full inclusion and community integration for individuals with disabilities.

Community re-entry programs teach skills necessary along a continuum of services that afford persons the opportunity to learn behaviors needed to live full lives with a disability. Such programs provide services and supports to enable the successful transition of an individual with a disability from facility-based services to a viable community placement.

Note

The authors are not related by blood or marriage.

Cross References

▶ Quality of Life

References and Readings

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Community-Based Rehabilitation

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Synonyms

In vivo services; Milieu-based services; Outpatient rehabilitation

Definition

The provision of restorative or rehabilitative services targeted toward the amelioration of a disability (disability: any illness, condition, or impairment [congenital or acquired] that interferes with functioning in a major life domain) and/or the mitigation of functional limitations resultant from a disability to the maximum extent possible in a non-acute care, non-facility-based service setting. Community-based rehabilitation services attempt to maximize the individual's ability to function "in vivo" within major life pursuits such as independent daily living and work.

Community-based rehabilitation attempts to enhance the quality of life for individuals with disabilities and their families. This includes meeting basic needs, but also going beyond to promote inclusion and participation. Community-based rehabilitation typically involves several domains of functioning such as health, education, work, socialization, and one's sense of empowerment. Reaching a point of social acceptance in the 1970s and 1980s, the original mission of community-based rehabilitation was to offer individuals with disabilities access to programming and supports in their own home communities using local resources. Further, community-based rehabilitation ostensibly attempts to optimize the provision of rehabilitative services, equalize opportunity, reduce poverty, and promote full social inclusion.

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Comorbid

► Dual Diagnosis

Compartmentalization

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Definition

Drugs do not saturate tissues within the body at the same rate. They are quickly absorbed into blood plasma and into well-perfused organs. Muscle, fat, and bone are saturated last. These tissues can serve as storage depots for substances, contributing to the half-life of a drug and potentially to interaction effects days or weeks after a drug has been discontinued.

The body may be inappropriately be treated as a single entity or compartment. In a single-compartment model, drugs are theoretically presumed to diffuse through tissues and the differences in absorption and retention of a drug are not treated as significant. This single-compartment model is sufficient in many situations, but is less useful when drugs may be preferentially retained in one type of tissue. Dosage predictions fail if they do not account for the gradual ambient leaching of the drug from fat or bone into general circulation.

The instances in which a multi-compartment model is most relevant are for individuals who are medically ill, have unusually high or low body fat, are pregnant, have an abnormality in pH or illnesses which impact the liver or kidneys (and thus profoundly alter the pharmacokinetics of drug metabolism). Differences in the proportion of fatty tissue can influence dosing decisions since mg/kg may be less appropriate for drugs that are less fat-soluble. One common example of this reduced alcohol tolerance in women since fatty tissues do not readily absorb alcohol.

Another form of compartmentalization is ion trapping, in which the pH alters the ease with which a drug is transferred into or out of a particular tissue. For example, neonatal blood is typically more acidic than adult blood, making it more difficult for slightly alkaline drugs to diffuse back into maternal circulation. Similarly, the GI tract is more acidic than blood plasma. Weakly acidic drugs diffuse more readily to blood than weakly alkaline drugs.

Cross References

► pH
► Pharmacokinetics

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Compensation Neurosis

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Synonyms

Accident neurosis

Definition

Compensation neurosis is a condition in which symptoms are associated with a real or presumed disability (possibly exaggerated) that may bring financial compensation. This type of neurosis is believed to be motivated by the desire for, and hope of, monetary or interpersonal gain. Many factors are involved in compensation neurosis, such as the psychological factors before and after the presumed injury, the additional effects on the quality of life, and the possible influences of legal or insurance processes. The term is often used in litigation and seems to be quite controversial in psychiatry.

Cross References

- ▶ Fake Bad Scale
- ▶ Malingering

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Compensatory Education Approach

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Definition

Compensatory educational approaches require teachers to present material in a different format, providing students an alternative way to master a particular concept and demonstrate knowledge.

Current Knowledge

The goal of education is to help students acquire skills and master concepts that will be useful in daily living. Generally, two approaches are used when instruction fails to help students learn. These include compensatory education approaches and/or remedial education approaches (▶ Remedial Education Approach). Compensatory educational approaches require teachers to present material in a different format, providing students an alternative way to master a particular concept and demonstrate knowledge. They are used when students lack the ability to acquire a certain skill or concept. For example, a deaf student who is unable to speak may be taught sign language as an alternate form of communication; or, a student with a math calculation disability may be taught to use a calculator. When students fail to make progress in a certain skill area, evidence-based strategies and methods must be employed to help the student attain expected learning outcomes. In these cases, education must be adjusted by offering different activities that facilitate the same results. This approach in isolation does not represent best practice, and in keeping with the educational and neuro-developmental literature, practitioners should offer both remedial and compensatory approaches, depending on students' specific academic needs. Educators must be careful to distinguish between the educational content in which a student struggles (e.g., reading or math), and the method in which a particular concept may be successfully integrated. Offering a compensatory approach should be a last resort on the educational

continuum, and should be supported by a comprehensive neuro-psychological evaluation.

Cross References

- ▶ Academic Competency
- ▶ Academic Skills
- ▶ Compensatory Education Approach
- ▶ Remedial Education Approach
- ▶ Strength-Based Education Approach

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Compensatory Strategies

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Definition

Compensatory strategies are environmental modifications or behavioral strategies designed to bypass persistent impairment in attention, memory, executive-function, and/or other cognitive skills as a means to achieve desired rehabilitation goals. Environmental modifications could include the use of external aids or modifying the setting in which activities take place. The use of an alphanumeric pager and a checklist for a person with memory and executive-function deficits to ensure completion of daily tasks at specific times would be an example of external aids. Working in a distraction-free room to enhance concentration skills in a person with symptoms of disinhibition would be an example of modifying an environment. Examples of behavioral strategies would include repeating phrases during social interactions to ensure accurate processing of conversation, or associating words with images to enhance recall.

Current Knowledge

Compensatory strategies have shown efficacy in randomized, controlled studies for improving the ability to remember to complete activities in the future (prospective memory) in neurologically impaired people with cognitive difficulties. For example, Wilson, Emslie, Quirk, and Evans (2001) used an external pager in 143 people between 8 and 83 with a range of neurological illnesses or trauma. Results revealed that more than 80% of clients improved in carrying out self-care activities and meeting appointments, as well as other life skills, relative to the baseline period, and effects were maintained seven weeks after return of the pager. Environmental modifications, including use of signs, removal of distracting environmental stimuli, and checklists have been used effectively in randomized, controlled trials to enhance adaptive functioning and quality of life in people with severe psychiatric illnesses as well (Velligan, Prihoda, Ritch, Maples, Bow-Thomas, & Dassori, 2002).

Cross References

- ▶ Cognitive Rehabilitation
- ▶ Environmental Modifications
- ▶ Mnemonic Techniques
- ▶ Neuropsychological Rehabilitation
- ▶ Prosthetic Memory Aids

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Competence to Testify

► *Jenkins v. U.S. (1962)*

Competency

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Synonyms

Capacity

Definition

Competency is a legal determination to be made by a legal professional (i.e., judge). It relates to a person's capacity (a clinical status as judged by a health care professional) to make determinations/decisions or to perform certain functions. In a legal context, competency typically relates to one's understanding of issues related to involvement in a legal proceeding (Reisner & Slobogin, 1990). Such an understanding necessitates some degree of acknowledgment regarding the nature of the procedure, the risks involved, success estimates, possible alternative options/approaches, and the pros and cons of specific courses of action. Issues related to competency can be raised at any time during the criminal judicial process. Specific competencies in the criminal realm include: competence to confess (or waive rights at pretrial investigations), competency to plead guilty, competency to waive right to counsel, competency to stand trial, competency to be sentenced, competency to waive further appeal (when facing capital punishment), and competency to be executed. Competency evaluations are the most common referral of mental health professional in criminal forensics. A defendant's competency can be questioned by attorneys and judges. In the civil realm, impairment of competency, or decision-making capacity, is often a consequence of various types of dementias and other organic brain syndromes. Impairments in memory, judgment, reasoning, and planning can affect a person's capacity to: make medical decisions, consent to research, manage financial affairs, execute a will, drive, manage medications, live independently, and handle even the most basic activities of daily life (ADLs).

In the criminal realm, competency relates to a defendant's *current* ability to understand and partake in legal proceedings. The *Dusky* standard states that: "The test must be whether he (the defendant) has sufficient present ability to consult with his lawyer with a reasonable degree of rational understanding – and whether he has a rational as well as factual understanding of the proceedings against him." (p. 402). The *Dusky* standard outlines the minimal level of competency necessary in accordance with the U.S. Constitution for all criminal jurisdictions. Grisso (1988) outlined five areas of analysis suggested for psychologists/neuropsychologists conducting competency evaluations: (1) functional description of specific abilities, (2) causal explanations for deficits in competency abilities, (3) interactive significance of deficits in competency ability, (4) conclusory opinions about legal competency and incompetency, and (5) prescriptive remediation for deficits in competency abilities. In an attempt to operationalize the *Dusky* ruling, the Group for the Advancement of Psychiatry (1974) developed a 21-point list of abilities derived from competency assessment instruments available at the time and included items such as: understand current legal situation, understand legal defenses available in the defendant's behalf, tolerate stress at the trial and while awaiting trial, and protect self by using available legal safeguards. As a result of *Wieter v. Settle* (1961), the U.S. District Court outlined several minimal ability requirements for criminal competency including (but not limited to): mental capacity to appreciate his or her presence in relation to time, place, and thing; apprehends that there is a judge on the bench; apprehends that there is, or will be, a jury present to pass on evidence adduced as to his or her guilt or innocence of such charge; he or she has memory sufficient to relate those things in his or her own personal manner.

Currently, there are a number of instruments designed to assess competency (primarily in the criminal realm), which are referred to as competency assessment tools (CATs). Examples include: the Competency Assessment Inventory-Revised, the Fitness Interview Test-Revised, the Georgia Court Competency Test, the Competency Assessment for Standing Trial for Defendants With Mental Retardation, and the MacArthur Competency Assessment Tool-Criminal Adjudication. It is important to note that none of the aforementioned CATs have been validated with brain-injured or neurological populations. Marson and colleagues (cf. Marson & Hebert, 2005)

have developed instruments to assess consent capacity in older adults.

Cross References

► Financial Capacity

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Competency to Proceed

► Legal Competency

Complex Figure Test (CFT)

► Rey Complex Figure Test

Complex Partial Seizure

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Synonyms

Partial epilepsy; Psychomotor seizures; Temporal lobe seizure

Definition

Formerly known as "psychomotor seizures," complex partial seizures (CPS) are defined by the International League Against Epilepsy as focal seizures with impaired consciousness as the central feature.

A CPS may begin with a simple partial seizure, also referred to as an aura. This commonly consists of feelings of emotional, ideational, or sensory changes. Once consciousness becomes impaired, individuals with CPS may experience automatisms, which are often characterized as repetitive, semi-purposeful involuntary movements. These may be oropharyngeal (e.g., lip smacking), emotional (e.g., fear), gestural (e.g., picking), ambulatory (e.g., walking), or verbal in nature. In some cases, focal CPS secondarily progresses into a generalized event (i.e., tonic–clonic seizure). The specific paroxysmal behavior changes correspond to the seizure focus and the progression represents the spread of epileptiform activity. Individuals with CPS often experience brief postictal confusion and are usually amnestic for their ictal behavior.

Current Knowledge

Epidemiologically, CPS comprises an estimated 42% of all partial seizures and 20% of all epilepsies. Approximately 60–80% of CPS arise from the temporal lobe, particularly the neocortex and inferomedial (limbic) structures. While commonly equated with temporal lobe seizures, it is now known that forms of CPS can originate from other brain regions, most notably the frontal lobes. A diagnosis of CPS can be identified through a neurological history and description of the events. However, the electroencephalogram (EEG) remains the "gold standard" for diagnosis. Patients with CPS often exhibit abnormal discharges on interictal recordings and a characteristic pattern of onset and spread of activity when seizures are recorded. CPS is most commonly treated with antiepileptic drugs. Surgical intervention for intractable CPS has been increasingly pursued in recent years. Successful outcomes are most often achieved following temporal lobectomy or lesion resection in CPS patients with underlying mesial temporal sclerosis.

Cross References

► Aura
► Generalized Tonic-Clonic Seizures

► Temporal Lobe
► Temporal Lobe Epilepsy

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Complex Partial Seizures with Automatisms

► Psychomotor Epilepsy

Comprehensive Driving Assessment

► Driving Assessment

Comprehensive System (CS)

► Rorschach

Compulsory Self-Incrimination

► Self-Incrimination

Computed Tomography

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Synonyms

CAT Scan; Computerized axial tomography; CT

Definition

CT is an imaging modality that generates cross-sectional images of the body with very good anatomical detail. This is accomplished by an X-ray tube rotating 360° around the body as it moves through the CT scanner. During rotation a collimated (focused) X-ray beam is transmitted through the body which is attenuated by the tissues. The attenuated X-rays are sensed by an array of detectors on the opposite side as they leave the body. The signal from the detectors is processed by a computer to reconstruct a cross-sectional image. Images in axial (dividing the body into superior and inferior sections), cornal (dividing the body into anterior and posterior sections), and sagittal (dividing the body into left and right) planes, as well as 3D, can be reconstructed the data accquired. Images in the axial (dividing the body into inferior and superior segments), sogittal (dividing the body into left and right segments), and coronal (dividing the body into anterior and posterior segments) planes, as well as 3D images, can be reconstructed from the data accquired. The image is stored electronically and displayed on the computer screen. Hard copies of the image can be photographed onto film. The image is made up of a matrix of 2D cells called pixels, similar to a digital camera. The pixels are 2D representations of 3D volumes of tissue called voxels.

Different scanning geometries can be obtained including helical (or spiral) and multi-slice acquisition. In helical scanning contiguous slices are obtained with simultaneous continuous X-ray tube rotation and patient movement. Multiple rings of detectors can be activated by single rotation of the X-ray tube enabling multiple slices to be obtained. CT images are produced so that you view the films as if looking toward the patient's feet with the patient lying on their back; therefore, the right side of the body is to one's left and vice-versa.

The density of tissues (the *attenuation coefficient*) is expressed in Hounsfield units. All tissues are measured relative to the density of water which is set at 0. Less dense materials, such as air, appear black; more dense materials, such as bone, appear white. Tissue densities in between appear as various shades of gray.

CT in Identifying Neurologic Disorders

Traumatic brain injury: CT is useful in moderate to severe acute head trauma. It can accurately diagnose hemorrhage, pneumocephalus, foreign bodies, and fractures (C1-C3 should be included, as upper cervical spine fracture is associated with 10% of severe head injuries). Secondary effects of trauma such as edema and cerebral herniation are easily detected by CT. MRI may show subtle vascular dissections, diffuse axonal injuries and infarctions not initially evident on CT, as well as, evolution of hemorrhages that allows better dating of injuries than CT.

Hemorrhage/stroke: Non-contrast CT is the preferred investigative modality for presentation of acute stroke, in order to rule out a bleed. CT has a higher sensitivity than spin-echo MRI for detecting acute hemorrhage. MRI can detect site and volume of acute infarct. Some literature suggests that a T2-weighted MRI has the same sensitivity for acute hemorrhage as CT. However, rapid imaging, less motion artifact, availability, cost, and compatibility with ferromagnetic metals (pacemakers, foreign bodies), maintain CT as the primary imaging modality in acute stroke.

Gray/White matter differentiation and subtle lesions: CT may not be sensitive enough to detect lesions with subtle contrast. MRI has better gray/white differentiation and can detect subtle lesions within white matter. Therefore, it is preferred for subtle lesions such as encephalitis, hippocampal sclerosis, suspected intracranial tumors, multiple sclerosis, and acute ischemic stroke.

Bony Detail: Bony pathology such as a fracture, mastoiditis, or sinusitis is better imaged with CT than an MRI. However, since X-ray photons are preferentially absorbed by bone this can cause streaky bone artifact (called "cupping") when trying to view brain tissue at the skull base. MRI is the preferred modality for brainstem lesions as there are fewer bony artifacts.

Hydrocephalus and Cerebral Atrophy: CSF containing spaces are clearly shown on a CT and thus ventricular/sulcal size, shape, and proportionality are easily evaluated.

Current Knowledge

Advantages of CT

- Excellent anatomical detail
- Rapid imaging time
- Less expensive than MRI
- Less movement artifact than MRI

Disadvantages of CT:

- Relatively high dose of radiation
- Bony areas can cause artifact, limiting use in the posterior fossa, spine, bone/parenchyma interface, and pelvis
- Metallic objects also cause artifact if not MRI compatible
- Not good at allowing relative dating and evolution of intraparenchymal hemorrhage

Cross References

▶ Angiography, Cerebral
▶ Magnetic Resonance Imaging
▶ Neuroimaging

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Computer-Aided Assessment

▶ Test Interpretations: Computer Based

Computer-Based Test Interpretation

► Test Interpretations: Computer Based

Computerized Assessment of Response Bias

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Synonyms

CARB

Description

The Computerized Assessment of Response Bias (CARB) is a computer-based tool for evaluating effort. The CARB is one of many measures characterized as symptom validity tests (SVT), indices of response bias used to assess possible malingering, poor effort, or exaggeration of deficit. The test is a computerized version of the digit recognition paradigm in a forced choice format, akin to the procedures described by Hiscock and Hiscock (1989) and Binder (1990). Frequently, statistical determination of below chance performance (based on the binomial theorem) has been used with tests such as these to identify persons showing poor effort. This method requires an extremely low score to reach a statistically significant level. However, as the CARB is an easy test, it allows the clinician to detect inadequate effort using above-chance cutoff scores (Millis, 2008). The test has been studied with a wide variety of populations, including persons with head injury, chronic fatigue syndrome, musculoskeletal injuries, pain disorders, and psychiatric or emotional disturbances such as depression. Published studies have included both adults and children. Administration time can vary depending on the protocol used; however, it typically can be completed in no more than 10–15 min. CARB performance results include percent correct for each group of trials as well as the overall percent correct. Also reported are the response latency for each item, average latency per group of trials, and overall, as well as other, information. An interpretive summary can also be provided, if desired.

Historical Background

CARB was designed and published by Conder, Allen, and Cox (1992) to provide a computerized tool for administration of the forced-choice, digit recognition tasks that were gaining popularity in assessment of effort during neuropsychological testing. Hiscock and Hiscock (1989) had described the use of forced-choice methods in detecting malingering, and Binder (1990) had also studied this method of assessing effort. The authors of CARB elected to name the test with the phrase "response bias" (as opposed to malingering or effort) to clarify that the test was not "diagnosing" malingering, but measuring a bias in the response.

Several versions of CARB have been published and studied. The original version (Conder et al., 1992) utilized three distinct trials of 37 items each. Each item includes presentation of a five digit number that is to be remembered followed by a short delay during which the examinee is to count *silently* backward from 20. The examinee then selects the presented number from two choices – the target and a foil. The groups of trials differ from one another in that the delay between stimulus presentation and forced-choice presentation increases from 3 to 6 and 9 s, respectively. Initial studies of this version of CARB determined that performance at or below 89% correct falls more than two standard deviations below the sample of individuals with documented brain injury (Conder et al., 1992); performance below 90% was therefore deemed to be biased.

Subsequent versions of CARB include CARB-97 (Allen, Conder, Green, & Cox, 1997) and another revision referenced in Green and Iverson (2001). CARB-97 added auditory feedback to the original version such that correct responses are followed by a pleasant tone and incorrect responses are followed by an unpleasant tone. The third revision shortened the delays between stimulus presentation and forced choice presentation to 1.5, 2.5, and 3.5 s. Each CARB version has been utilized in published studies, although the original version may continue to be the most widely used (Larrabee, 2007).

Psychometric Data

As with many, if not most, symptom validity tests the nature of CARB – presentation of a relatively easy task that may be perceived to be much more difficult than it actually is – leads to a distribution of scores that is highly skewed toward the upper end. Individuals who put forth effort on the test get all, or nearly all, of the items correct.

This appears to be true regardless of the presence or absence of brain injury or other cognitive deficits. CARB was originally validated on a small sample (8) of individuals who had documented severe brain injury. That group performed at 98.6% (SD = 3.6) correct (Conder et al., 1992). Internal consistency across items is therefore also quite high among individuals putting forth adequate effort; biased responding with suboptimal effort is presumed, by the nature of the construct, to be more variable. Individuals who perform at a "passing" level on CARB yielded 96.6–97.6% accuracy across the blocks of trials, whereas those who performed at a non-passing level yielded accuracy scores ranging from 62.4 to 74.6% (Allen et al., 1997; Allen, Richards, Green, Iverson, & Conder, 1998). An accuracy difference of 10% or more between any two blocks of the test correlates highly with poor (non-passing) scores on CARB (Allen et al., 1997). Test–retest reliability (1-week interval) is quite high ($r = 0.97$) (de Armas, 1996).

Clinical Uses

Clinically, CARB is used to assess response bias and level of effort and assist in the detection of symptom exaggeration and/or malingering. Published studies have primarily involved populations of individuals alleging brain injury via closed head trauma, although other diagnostic groupings have also been examined. These include chronic fatigue syndrome, fibromyalgia, stroke, chronic pain, multiple sclerosis, brain aneurysm, and brain tumor (Allen, Iverson, & Green, 2003).

Additional studies have used samples of persons with brain injury, alleging brain injury, or with alleging other disorders that may affect cognition. The variable of "seeking versus not seeking compensation" as a result of an injury or condition is a significant one in the realm of assessing effort. Lending support to the notion that secondary gain can be a major factor in these cases. Gervais, Green, Allen, and Iverson (2001) reported that 100% of the individuals with fibromyalgia and rheumatoid arthritis who were *not* seeking compensation performed in a fashion that indicated adequate effort. Age does not appear to be a significant factor influencing performance on CARB. Flaro, Green, and Allen (2000) reported on children performing at or above normal adult performance levels on CARB. Allen et al. (2003) summarized a number of studies providing validation for the utility of CARB with a variety of diagnostic groupings.

CARB appears to be an appropriate measure of effort across a very large age range, and would in theory be appropriate in any clinical population. The concept of the binomial theorem and evaluation of performance against chance levels is a useful and reasonably valid measure. Clearly, however, there is support for the use of CARB with cutoff scores at above-chance levels in assessment of those groups of whom scientific studies have been published.

Cross References

- ▶ Dissimulation
- ▶ Effort
- ▶ Forensic Neuropsychologist
- ▶ Forensic Neuropsychology
- ▶ Forensic Psychology
- ▶ Hiscock Forced-Choice Test
- ▶ Malingering
- ▶ Portland Digit Recognition Test
- ▶ Response Bias
- ▶ Rey 15-Item Test
- ▶ Symptom Validity Assessment
- ▶ Word Memory Test

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Computerized Axial Tomography

► Computed Tomography

Concentration

► Attention

Concept Learning

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Synonyms

Hypotheses; Rules

Definition

An abstract or general idea that is inferred or derived from specific instances.

Current Knowledge

A *concept* is a special combination of ideas that has a particular meaning. *Rules* are concepts that direct behavior because there are consequences attached to them. A *hypothesis* relates two or more concepts with a prediction (Parente & Herrmann, 2010). Hypothesis testing, rule learning, concept learning, and problem solving are all interrelated skills. Concept learning is how humans learn to divide their environment into examples and nonexamples of things they understand. Rule learning is the process of associating concepts with consequences. Hypothesis testing is a person's ability to try out several possible concepts, solutions, or rules in a systematic way to determine which is the most useful. Cognitive psychologists define concept learning as the development of *prototypes, schemas, attributions, or exemplars* (Lamberts & Shanks, 1997). Prototypes are learned typical representations of the concept. For example, a compact car is small, gets good gas mileage, and is a Toyota. Schemas are scripts that are learnt for behaving or for evaluating behavior. For example, an introduction usually involves a greeting, a smile, and a handshake. Attributions are collections of behavior adjectives and adverbs that define the notion of a person, place, or behavior. For example, Republicans are white, rich, and drive Hummers. *Exemplars* are collections of examples of concepts that are stored in memory. For example, observing people at the mall defines the concept of an everyday person. Humans use several different strategies to form concepts. *Conservative focus* involves gradual manipulation of one aspect of a complex concept at a time until the concept clarifies. *Focus gambling* tests several possible hypotheses at the same time instead of one at a time. *Scanning* involves forming global hypotheses about a concept, then modifying them in accordance with rewards and punishments. *Trial and error* is the tendency to "just keep trying until something works." Aspects of the task that improve concept acquisition include an orderly arrangement of the items that the person must use to form the concept. A compact display in which all the aspects of the concept are present also improves acquisition. Sequential problems that present a consistent concept also improve concept acquisition. The saliency, novelty, and complexity of the concept, and whether feedback is provided affect how quickly a person learns it (Hunt & Ellis, 2003).

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Conceptual Knowledge

► Semantic Memory

Concordance

► Inter-rater Reliability

Concurrent Validity

► Test Validity

Concussion

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Synonyms

Mild traumatic brain injury; Sport-related concussion

Definition

A concussion refers to a trauma-induced alteration in neurological function. Giza and Hovda (2001) and Hovda et al. (1990) discuss the abnormal neurometabolic processes associated with concussion. Early clinical signs of concussion include alteration of mental status or behavior, and can be accompanied by other symptoms outlined below.

Diagnosis

Diagnosis of concussion is made based on identifying an event with adequate biomechanical force to cause a concussive injury and by examining acute injury severity indicators. Frequent events associated with a direct trauma or blow to the head to cause concussion include a fall, motor vehicle accident, or sports collision. In addition to a direct blow to the head, a concussion can result from rapid acceleration, deceleration, rotational or percussive forces that affect brain tissue. Injury severity indicators frequently used to diagnose concussion include alteration of consciousness (confusion), loss of consciousness (LOC), post-traumatic anterograde amnesia (PTA), retrograde amnesia, and Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974). Standard neurological examination and imaging studies are frequently normal following concussion (i.e. have limited sensitivity) so should not be relied on to diagnose concussion. Among clinical and scientific experts, a concussion is synonymous with mild traumatic brain injury (MTBI). Please see MTBI for a more detailed discussion of issues related to diagnosis, management, recovery, and outcome post concussion.

Current Knowledge

Epidemiology

Current epidemiology statistics likely grossly underestimate the true incidence and prevalence of concussion because of imprecise surveillance systems (e.g., many individuals with concussive injury never seek medical care and those who do seek care are frequently not hospitalized), and because current estimates are clouded by variable research methodologies and inconsistent definitions of concussion (McCrea, 2008; National Center for Injury Prevention and Control, 2003). As discussed in greater detail under MTBI entry, best estimates would suggest an incidence of at least 100/100,000 and perhaps as high as 500/100,0000 population depending on study methodologies (McCrea, 2008).

Recovery and Outcome Post Concussion

Concussions fall under the general rubric of MTBI, and are frequently graded by severity (e.g., grade 1–3 concussion). More significant concussive injury is associated with greater length of altered consciousness, LOC, or amnesia, although concussive injury often occurs without observed LOC or measurable amnesia. Generally speaking, acute neurologic, behavioral, or cognitive symptoms begin acutely and resolve within 7–10 days, with a small percentage of individuals exhibiting symptoms beyond 3 months (Belanger & Vanderploeg, 2005; Iverson, 2005; McCrea, 2008). Providers should expect a relatively rapid and full recovery for patients following a single concussion. There is no apparent difference in outcome across the various grades of concussion, though there are reports of longer recovery periods for "complicated" cases, such as those with intracranial abnormalities identified via imaging (Williams, Levin & Eisenberg, 1990).

Evaluation and Management Post Concussion

Early evaluation of symptoms and neurological status is essential following concussion to diagnose severity of injury and to rule out complications such as intracranial abnormalities or other comorbid injuries. Computed tomography (CT) imaging of the brain is frequently ordered by medical specialists because it continues to be the most sensitive procedure for detecting acute intracranial abnormalities post injury.

While spontaneous improvement over days to weeks is the hallmark of recovery following concussion, it is generally accepted that behavioral management (e.g., education about MTBI, recovery and outcome; teaching cognitive–behavioral strategies to manage symptoms) may minimize the likelihood of a suboptimal outcome and may contribute to improved quality of life for patients, perhaps because of greater understanding of one's injury and greater appreciation for the expected recovery following injury. Pharmacologic intervention for certain symptoms, such as pain, sleep abnormalities, or mood disturbance, may also be indicated to help with early adjustment and quality of life issues following concussion. Post-acute evaluation and monitoring is crucial for tracking a person's recovery following injury. This will help providers determine when it is appropriate for patients to resume prior activities, such as collision sports, academics, or work. Individuals recovering from concussion appear to be more vulnerable for re-injury or catastrophic injury. See Kelly and Rosenberg (1997) for an excellent discussion of issues involved in management of sports-related concussion, principles of which can be applied to management of concussions resulting from any mechanism.

Neuropsychological consultation is particularly helpful in the evaluation and management of patients following concussion. Neurocognitive testing is sensitive to early, post-acute, and subtle symptoms associated with concussion, objectively tracks cognitive recovery over time, and provides valuable information to inform return to activity decision-making. Without neuropsychological data, medical specialists must rely solely on a person's subjective report about residual neurocognitive symptoms. Education and cognitive behavioral therapy techniques are important treatment tools frequently employed by neuropsychologists or other behavioral management specialists to help prevent or treat persisting postconcussive symptoms (Mittenberg, Canyock, Condit, & Patton, 2001; Mittenberg, Tremont, Zielinski, Fichera, & Rayls, 1996).

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Conditioned Stimulus

► Stimulus Control

Conduction Aphasia

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Synonyms

Associative aphasia

Short Description

Conduction aphasia is a subtype of fluent aphasia that is characterized by fluent speech and relatively intact

language comprehension, but significantly impaired repetition. Utterance length is normal or increased, and speech has normal prosody and grammar and is produced with normal effort. There is a reduction in content words, paraphasic errors are common, and oral reading is impaired.

Categorization

Conduction aphasia is differentiated from other types of fluent aphasia (Wernicke's aphasia, transcortical sensory aphasia, and anomic aphasia) by the disproportionate impairment in repetition relative to comprehension and spontaneous production. It is differentiated from Wernicke's aphasia in particular by the patient's awareness of his or her paraphasic errors.

Natural History, Prognostic Factors, Outcomes

The prognosis for recovery of functional communication in individual with conduction aphasia depends on the underlying cause of the aphasia as well as factors such as the size of lesion and the patient's age, premorbid language skills, and comorbid health conditions. Individuals who initially present with conduction aphasia often evolve to a clinical profile of anomic aphasia, with relatively good auditory and reading comprehension, and deficits primarily in word-finding and the comprehension and production of complex syntax.

Neuropsychology and Psychology of Conduction Aphasia

Conduction aphasia historically has been attributed to the interruption of white matter pathways, notably the arcuate fasciculus (part of the superior longitudinal fasciculus), that connect posterior and anterior cortical structures involved in language comprehension and production. Thus, conduction aphasia historically was referred to as a "disconnection syndrome" (Geschwind, 1965). Lesion and imaging studies have revealed, however, that conduction aphasia may result from lesions in a variety of locations, and that arcuate fasciculus lesions are instead implicated in the production of repetitive, stereotypic utterances (stereotypies). This is consistent with the finding that the arcuate fasciculus is spared in many patients with conduction aphasia (Quigg et al., 2006). By contrast to the localization or modular view of Geschwind and others, researchers in connectionist theory have conceptualized language as the product of a distributed cortical network that can be disrupted in various ways to produce similar phenotypes. The advent of functional imaging resurrected the notion of disconnection syndromes and more modular notions of language (Catani & Ffytche, 2005), and the current view is somewhere in between a modular and connectionist perspective.

Evaluation

The signs and symptoms of conduction aphasia will be revealed only with testing of auditory and reading comprehension, spontaneous language (e.g., asking the patient to describe a picture), and repetition, so that the relative performance in these modalities can be compared.

Treatment

There is no specific treatment for conduction aphasia, beyond therapies that are appropriate for aphasia in general (▶ Aphasia).

Cross References

▶ Aphasia
▶ Nonfluent Aphasia

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Confabulation

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Description

Korsakoff initially described "pseudo-reminiscences" in alcoholic patients with amnesia who made up fictitious stories about events that did not occur. In their translation of Kosakoff's original work, Victor and Yakovlev note that Korsakoff first identified patients with a "psychic disorder in conjunction with multiple neuritis" who presented with "a derangement of memory and of the association of ideas" along with other symptoms of the now well-known Korsakoff syndrome (Korsakoff, 1955). Later, the term confabulation was introduced and defined as the "falsification of memory occurring in clear consciousness in association with an organically derived amnesia" (Berlyne, 1972). It has also been referred to as "true memories that have been misplaced in both time and place" (Kopelman, 1987) as well as the "spontaneous narrative reports of events that never happened." More recently, confabulation has been defined as "statements or actions that involve distortions of memories" (Metcalf, Langdon & Coltheart, 2007).

Confabulation, as a symptom, has been described in a number of disorders including strokes (particularly anterior communicating artery), traumatic brain injuries, dementia, metabolic disorders, and psychiatric disorders. Confabulations are mentioned as a probable symptom in most of the descriptions of Korsakoff's syndrome despite ambiguous clinical diagnostic guidelines (Borsutzky, Fujiwara, Brand, & Markowitsch, 2008). For instance, the ICD-10 mentions confabulations as a probable but not obligatory symptom for diagnosing an "Alcohol-Induced Amnesic Syndrome" while the DSM-IV (American Psychiatric Association, 1994) does not include confabulations in its guidelines for diagnosing "Alcohol-Induced Amnesic Syndrome."

Categorization

There are different descriptions of confabulation used by several authors. *Momentary confabulations* have been described as brief, partially accurate responses often inaccurately localized in time (Victor & Ropper, 2001). *Fantastic or productive confabulations* have been described as more elaborate fictitious stories (Berlyne, 1972). Further distinctions have been made between confabulations that are *provoked* by direct questions, often in the context of memory testing, or *spontaneous* confabulations occurring without any external trigger (Kopelman, 1987). The boundaries between provoked/spontaneous and momentary/fantastic confabulations are often blurred, with many authors mixing these characteristics when describing the symptoms. Some have argued against such categorizations and instead suggested that confabulation should be regarded as a continuous variable, ranging from minor distortions to more dramatic fantasies (Metcalf et al., 2007).

Neuropsychology of Confabulation

Studies using neuropsychological test performance to examine mechanisms of confabulation have not yielded consistent results. Observations of perseverative responses in individuals who confabulate have implicated frontal executive dysfunction (e.g., Kopelman, Stanhope, & Kingsley, 1997). Others have argued for more prominence of memory impairments rather than executive dysfunction (e.g., Dalla Barba, 1993). A combination of executive and memory deficits may be involved. Memory impairment may explain the presence or absence of confabulation, but the severity of confabulation may be influenced by the extent of executive dysfunction (Fischer, Alexander, D'Esposito, & Otto, 1995). Clinical lesion studies have implicated the medial prefrontal cortex in spontaneous confabulations, particularly the orbitofrontal region (Schnider, 2003). Damage to limbic structures involved in memory is also likely to be required for confabulations to occur (Fischer et al., 1995). The literature on anterior communicating artery aneurysms (ACoA) has implicated frontal structures as a necessary site of neuropathology in confabulation, although cases of nonamnestic ACoA patients with frontal lesions who do not confabulate have suggested that both frontal lobe and basal forebrain structures may be needed for spontaneous confabulations (DeLuca & Diamond, 1995).

Four types of cognitive models have been proposed to account for confabulations:

1. *Source monitoring theory.* This view was initially promoted by Johnson, Hashtroudi, and Lindsay (1993) who argued that individuals who confabulate

are unable to accurately monitor the source of their memories which results in errors and confusion. According to this view, patients who confabulate cannot accurately identify where or when events occur. They may also be unable to differentiate between whether a representation is "real" or not due to impairments in being able to access qualitative characteristics of the representation such as details about perceptual, contextual, affective, and semantic information. Furthermore, secondary evaluation deficits may also lead to the over-inclusive acceptance of memories.

2. *Temporal context theory.* Dalla Barba, Mantovan, Cappelletti, and Denes (1998) have suggested that the primary impairment in confabulation is a difficulty with "temporal consciousness." According to this view, patients who confabulate are confused about the temporal order of information retrieved from memory. While knowledge of time is preserved and patients are aware of a past, present, and future, they are unable to make correct temporal judgments about their memories, resulting in sequencing errors. Schnider (2003) has also argued that there is confusion between presently relevant and irrelevant memories, resulting in a failure to suppress activated but presently irrelevant memory traces. Neuroanatomically, Schnider et al. have linked confabulation with the posterior orbitofrontal cortex and the anterior limbic structures directly connected with it. Schnider argues that the adaptation of thought and behavior to ongoing reality is mediated by the anterior limbic system which acts by suppressing activated memory traces that do not pertain to ongoing reality. In patients who confabulate, this monitoring of ongoing reality in thought goes awry and appears to be related to the brain's reward system.
3. *Retrieval theory.* This view has been primarily promoted by Moscovitch and Melo (1997) who argue that deficient strategic retrieval processes and monitoring deficits influence confabulation. Specifically, confabulation is thought to be the result of a faulty "strategic retrieval" search and inaccurate ordering and placement of memories in context. According to this view, strategic retrieval is dependent upon executive processes and is self-initiated and goal-directed, while associative retrieval is automatic and independent of executive processes. Confabulators are thought to show deficiencies in strategic retrieval, influenced by deficiencies in the neocortical/prefrontal/hippocampal network hypothesized to be involved in strategic retrieval.
4. *Three-factor cognitive-neuropsychological theory.* Metcalf et al. (2007) propose a 3-factor combined model that hypothesizes interplay between an executive control retrieval deficit, an evaluation deficit, and a person's individual emotional/motivational biases. Specifically, according to this view confabulatory symptoms and content are the result of a failure of executive control that causes an impaired search and selection of appropriate memories from the autobiographical episodic memory store. Confabulators are purportedly unable to critically evaluate material from their autobiographical or general semantic store and accept confabulations as real memories. Further, personal biases may be involved in the preferential selection of memories that are emotionally biased.

Evaluation

Since Korsakoff's initial description in 1955 of "pseudoreminiscences" in alcoholic patients with amnesia who made up fictitious stories about events that did not occur, there has been considerable variability in how confabulation is defined, identified, assessed, and understood. For instance, it remains debated whether confabulations are readily differentiated from delusions. Some have argued that confabulations are not different from delusions since both are held firmly over time. Others have viewed confabulations, delusions, hallucinations, and insight as part of a continuum. Still others have argued that delusions and confabulations differ since confabulations tend to be associated with partially valid memories that can be traced to real events. Further debate ensues about whether confabulations are deliberate attempts to compensate for "memory gaps" due to embarrassment or whether they involve source monitoring, temporal context, or retrieval difficulties that result in an implausible or fictional output. Confabulation, particularly following ACoA aneurysms has also been viewed as representing differences in degree and not kind. Other important variables identified in the understanding of confabulation have included making the distinction between an "unaware" or "aware" process, premorbid personality factors, the need for coexistence of amnesia, differentiation from an acute confusional state, disconnection syndrome, and whether indifference/apathy or deceit/lying is involved (DeLuca, 2000). There has been further variability in decisions on how to label an individual as a "confabulator." Some have used the term based on clinical observations of spontaneous or provoked production of fabricated stories following brain injury. Other researchers

have labeled individuals as confabulators based on the number, or quality of intrusions/confabulations produced during standardized neuropsychological tests or experimental tasks. Overall, the concept of confabulation remains elusive and its underlying mechanisms remain an area requiring further understanding.

Treatment

Treatment is determined in part by etiology. Some have proposed that rehabilitation efforts should avoid memory training and repeated questions about orientation. For instance, according to Schnider (2003) it may be easier for a confabulating patient to "accept that her baby has already received food than to convince her that her baby is over 30 years old." Instead, it may be more beneficial to promote the engagement of patients in common everyday activities and accept their false interpretation of reality until spontaneous confabulations resolve (Schnider). Many spontaneous confabulations eventually resolve. In a study of eight spontaneous confabulators, almost all stopped confabulating 18 months later and regained correct orientation in time and place, as well as the ability to refer thinking and acting to ongoing reality (Schnider, Ptak, von Daniken, & Remonda, 2000). Specifically, "temporal context confusion" based on an inability to suppress intrusions of currently irrelevant memory traces into ongoing thinking paralleled the course of spontaneous confabulations in this study. Patients with isolated, less extensive, orbitofrontal lesions stopped confabulating within a few weeks and had the best neuropsychological outcomes. Patients with additional basal forebrain lesions continued to confabulate for several months and remained amnestic. One patient with extensive orbitofrontal damage and perirhinal cortex damage continued to confabulate after more than 3 years. Interdisciplinary approaches for the rehabilitation of confabulation in the treatment of patients with ACoA aneurysms have also been suggested, including a three-tiered approach focused on improving (1) intellectual awareness, the ability to understand that one has an impairment, (2) emergent awareness, the ability to recognize a problem when it is occurring, and (3) the ability to anticipate that a problem (confabulation) is going to occur (DeLuca, 1992). In one case, improvements in executive functioning using this rehabilitation approach removed obstacles to vocational activity and social functioning by improving the patient's awareness of his difficulties and enabling utilization of compensatory strategies (DeLuca & Locker, 1996).

Cross References

- ▶ Capgras Syndrome
- ▶ Delusion
- ▶ Intrusion Errors
- ▶ Reduplicative Paramnesia
- ▶ Wernicke–Korsakoff Syndrome

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Confidentiality

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Definition

Confidentiality has been defined as "containing information whose unauthorized disclosure could be prejudicial to the national interest." In psychology, it is one of the most important components of the Ethical Principles of Psychologists and Code of Conduct (2002) and the Specialty Guidelines for Forensic Psychologists (1991). The boundaries of confidentiality vary based on the setting, that is, whether it is in the clinical versus forensic realm and whether it is in the civil versus criminal realm. In treatment settings, clinicians consider confidentiality of paramount importance and they are reluctant to disclose information obtained from a client even when there are explicit legal or countervailing ethical mandates to do so (such as when a patient may harm another). Such a position is unrealistic in the forensic context because the results of forensic evaluations (be it civil or criminal) are routinely disclosed to third parties. In both clinical and forensic contexts, psychologists provide examinees with adequate "informed consent" prior to engaging in the examination. In the forensic context, it is the duty of forensic psychologists to inform clients of their legal rights regarding the purpose of an evaluation, the anticipated forensic service, the nature of procedures to be utilized, the limits of confidentiality, who has retained the examiner for the evaluation, who the report may be directed to, and that anything which is said during the examination could be included in a report or come up during deposition or trial testimony. Some jurisdictions do not provide for confidentiality of mental health evaluations. For example, when an evaluation is conducted as a result of direct court order, confidentiality is not provided.

Cross References

► Privilege

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Confirmatory Bias

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Definition

Confirmatory bias occurs when a clinician preferentially accepts or seeks evidence that confirms an initial hypothesis at the expense of thorough consideration of emerging contradictory evidence (Garb, 2003). For example, a clinician may formulate an initial impression that a client has dementia based on a referral question. During the clinical interview, the clinician may focus on questions relating to memory and change in activities of daily living while failing to ask questions that specifically pertain to differential diagnoses, such as major depressive disorder. In neuropsychological settings, this generally unintentional bias may impair judgment, hamper decision-making, produce false reports, and negatively impact the assessment approach and interpretation of findings. To address this bias, the clinician should systematically review all available data within a hypothesis-testing framework in an effort to remain as impartial as possible (Weiner & Greene, 2007).

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Conflict

► Contraindication

Conformal Techniques

► Involved Field Radiotherapy

Confound

► Confounding Variables

Confounding Variables

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Synonyms

Confound; Extraneous variable; Lurking variable

Definition

A confounding variable is an extraneous variable that obscures the true relation between two other variables or groups of interest. In experimental research designs, a confounding variable often presents as an unintended or undesirable systematic difference between groups (the independent variable) that is also systematically related to the outcome of interest (dependent variable). It hinders the ability to infer a causal relation between the variables and can lead to misattributing a causal effect to the independent variable (a threat to internal validity). Potential confounding variables are most effectively addressed during study design (e.g., via random assignment, case control matching), but may be addressed to some extent during statistical analysis (e.g., handled as a covariate).

Cross References

► Analysis of Covariance (ANCOVA)
► Error, Sources of

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Confrontation Naming

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Synonyms

Word finding

Definition

Confrontation naming involves the selection of a specific label corresponding to a viewed stimulus, usually a picture, of a viewed object or action. *Confrontation naming also refers to a type of task used in assessment when problems with naming are of concern.*

Current Knowledge

Confrontation naming tasks often are incorporated as part of clinical language testing to detect impairments of word-finding abilities in individuals with various types of neurologic impairments typically affecting the left hemisphere of the brain (Spreen & Risser, 2003). Although word finding takes place during the course of sentence generation in conversational speech, it is most often tested clinically in confrontation naming tasks where the vocabulary tested is constrained to known, identified target words. Therefore, word-finding functions are at times referred to as naming abilities (e.g., Chialant, Costa, & Caramazza, 2002).

The most common published test of confrontation naming is the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 2001); other published confrontation naming tests also exist for use in children and adults with word-finding difficulties (An Object and Action Naming Battery, Druks, & Masterson, 2000; Test of Adolescent/Adult Word Finding, German, 1990). Confrontation naming tests assess the ability to retrieve different types of words, including pictures of objects to test noun retrieval, or pictures of actions to test verb retrieval. Although most tests of confrontation naming test stimuli from a variety of semantic categories, some tests can differentiate pictures according to the semantic category to which they belong, including pictures from a variety of natural categories such as animals, fruits, and vegetables, and man-made categories such as furniture,

clothing, tools, and transportation (e.g., Goodglass, Kaplan, & Barresi, 2001).

Cross References

► Anomia
► Anomic Aphasia

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Congenital Defects

► Anencephaly

Congenital Hypothryroidism

► Hypothyroidism

Congestive Heart Failure

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Synonyms

Heart failure

Definition

Congestive heart failure (CHF) or heart failure is a condition in which the heart muscle is unable to pump sufficient blood to adequately supply the body's organs.

Current Knowledge

Common causes of CHF include coronary artery disease, myocardial infarction, hypertension, cardiac valve disease, cardiomyopathy (heart muscle disease), congenital heart disease, and endocarditis (heart infection). In CHF, the heart continues to pump, but exercise and activity cause shortness of breath ("dyspnea on exertion" or DOE), fatigue, weakness, light-headedness, or syncope. Because the heart is not pumping the blood completely and effectively, the blood "backs up" into the heart chambers, and ultimately into the venous system. This causes congestion of the tissues, and edema in the legs and internal organs, including the lungs ("pulmonary edema"), often resulting in shortness of breath, especially when lying supine ("orthopnea"). Electrocardiogram and certain blood tests are usually abnormal, and chest x-ray shows congestion of the lungs. Echocardiography is noninvasive and highly revealing, and has the potential to provide a quantitative measure of severity of heart failure as indicated by the proportion of blood in the left ventricle that can be pumped out by the muscle ("ejection fraction").

CHF is a chronic condition with acute exacerbations during which patients may experience moderate or significant distress. Chronic congestive heart failure can be managed using a combination of interventions that help the patient to "compensate" for the problems caused by CHF. Acute pulmonary edema is a medical emergency. Treatment of acute CHF episodes usually consists of rest, salt reduction, identification, and removal of the precipitating factor (such as infection, cardiac arrhythmia, or other cause), and the use of select medications that help to improve the pumping ability ("contractility") of the heart muscle and the capacity of the blood vessels to provide blood supply to other organs. These medications include angiotensin-converting enzyme inhibitors, beta-blockers, digoxin, diuretics (water pills), and vasodilators. At times, valve disease warrants valve replacement surgery, and extremely severe heart damage may require cardiac transplantation to prevent death. Usually, mild to moderate congestive heart failure can be treated with medications, adjustments of exercise and activity levels, and medical supervision.

Cross References

► Coronary Disease
► Echocardiogram

References and Readings

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Conjugate Gaze

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Synonyms

Eye movements; Versional movements

Definition

Conjugate gaze is the ability of the eyes to work together or in unison. It refers to the motion of both eyes in the *same* direction at the *same* time. The eyes can look laterally (left/right), upward, or downward. Disorders in conjugate gaze refer to the inability to look in a certain direction with both eyes.

Current Knowledge

Conjugate gaze is mediated in the brain stem by the medial longitudinal fasciculus, which is a nerve tract that connects the abducens, trochlear, and oculomotor nuclei. These nuclei, in turn, are responsible for the muscles that control eye movements. The left pontine center connects with the right frontal center for conjugate gaze to the left, and the right pontine center connects with the left frontal center for conjugate gaze to the right. If extraocular muscles are not working properly, dysconjugate gaze can result, which can then cause diplopia. The mechanisms for horizontal eye movements are better understood than vertical eye movements. As individuals age, the ability to look upward tends to decline. Cerebral structures control when and where the eyes move, and the brain stem structures control how they move.

The centers for lateral conjugate gaze are in the frontal and occipital cortices. In the frontal lobe, this area is in the posterior aspect of the frontal lobes, referred to as the frontal eye fields. This area is close to the motor strip. The function of the frontal centers is to control voluntary conjugate eye movements to the opposite side. The frontal eye fields (FEF) receive inputs from peristriate, parietal and superior temporal cortex, medial pulvinar, and the dorsomedial nucleus of the thalamus. Stimulation of FEF results in contralateral saccades. In strokes that affect this area, one may see an eye deviation toward the side of the lesion and away from the paralyzed limb. This usually occurs only in the acute phase of an infarct. At some point, the patient may be unable to move the eyes away from the lesion on command. However, they may be able to follow an object to the opposite side if the occipital lobe center is not damaged. The occipital lobe centers for lateral conjugate gaze control eye movement when an individual is following an object to the opposite side. Lesions of the occipital lobe that control lateral conjugate gaze are less common than lesions of the frontal centers.

Conjugate gaze can be disrupted by stroke or trauma, depending on the location of the damage. For instance, an intracerebral hemorrhage in the caudate nucleus or putamen will cause conjugate deviation of eye movements to the side of the lesion. Pineal tumors, which can press upon the midbrain, can cause paralysis of upward gaze. Deeper damage can affect downward saccades. Given the extensive anatomy of control of the visual system, damage or dysfunction along any of the nuclei or tracts integral to eye movements can result in abnormalities. Eye movements utilize the basal ganglia and cerebellum in their planning and coordination. Patients with basal ganglia disorders may have involuntary, small, or slow eye movements. Patients with parkinsonian symptoms may have a restriction of upgaze. In patients with progressive supranuclear palsy, a cardinal feature is the restriction of upgaze, and these patients initially complain of difficulties with reading. The flocculus of the cerebellum is important for suppression of vestibular reflex and for smooth pursuit movements. Parts of the vermis help to coordinate saccades, and damage can result in dysmetric saccades. Patients with long-standing frontal lobe lesions cannot inhibit inappropriate saccades from a fixation to a peripheral, visually attractive stimulus that appears suddenly. Frontal seizures with a focus in or

near one eye field may manifest by turning of the eye and head away from the side of the lesion.

Evoked eye movements (doll's eye test or oculocephalic reflex) and the caloric response can be used in the examination of an unresponsive patient. However, if a patient is in a comatose state due to drug intoxication or hypothermia, these tests may show no response. The assessment is as follows:

Doll's eye test – This test can be used in either the comatose or conscious patient. Hold the patient's eyes open and rapidly move/rotate the head to one side and hold it there. If the brain stem reflexes are intact, the eyes will move conjugately in the direction opposite to the head rotation. If the injury is in the brain stem, the eyes do not move. If a patient is conscious and can follow commands, have them fixate on an object. This test should not be used in a patient who has possible cervical injuries.

Caloric test – This test can be done in the comatose patient. The patient's ear canal is irrigated with 20 ml of ice-cold water. The eyes should move toward the ear that is irrigated. If the patient's eyes do not move, the lesion is in the brain stem. This test should not be used in a patient who has possible cervical injuries, or who has blood in the ear canal or a perforated eardrum.

Conjugate eye movements allow the eyes to get an image onto the fovea and keep it there. Fast movements or saccades allow images onto the fovea and slower movements keep them there. Smooth pursuit movements compensate for target movement. There are several ocular motor systems:

Saccadic system moves the eyes rapidly (up to 800°/s) in order to fixate on new targets in the visual fields. These can be voluntary or a response to a verbal command. Reflex saccades can also occur to stimuli that are threatening or to a sound in the periphery.

Pursuit system enables the eyes to track slowly moving targets (approximately 70°/s) so the image is stable on the fovea.

Vestibular eye movement subsystem maintains a stable image on the retina during head movements. The vestibulo ocular reflex maintains the eyes in the same direction in space during head movements. This is controlled by the semicircular canals, which respond to rotational acceleration of the head.

Optokinetic system complements the vestibular eye movement system. It uses reference points in the environment to maintain orientation. This system uses fixation and pursuit in humans.

Vergence system enables the eyes to move dysconjugately (converge and diverge) in the horizontal axis to maintain fixation on a moving target toward or away from the individual. These are critical for binocular single vision and depth perception.

Cross References

- ▶ Dysconjugate Gaze
- ▶ Lateral Gaze Palsy
- ▶ Saccadic Eye Movements

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Connectionist Model of Aphasia

▶ Wernicke–Lichtheim Model of Aphasia

Connective Tissue Disease

▶ Collagen Vascular Disease

Conners 3rd Edition (Conners 3; Conners 2008)

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Description

The Conners 3rd Edition (Conners 3; Conners, 2008), the latest version of the Conners Rating Scales, is a thorough assessment of attention deficit/hyperactivity disorder

(ADHD) and its most commonly associated problems and disorders in school-aged youth. The Conners 3 is a multi-informant assessment with forms for parents, teachers, and youth. Parent and teacher ratings can be obtained about youth aged 6–18 years, and youth aged 8–18 years can complete the self-report.

The assessment features multiple content scales that assess ADHD-related concerns such as inattention and hyperactivity as well as related problems in executive functioning, learning, aggression, and peer/family relations. Symptoms of distinct diagnoses can be assessed with scales that link directly to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; American Psychiatric Association, 2000) symptomatic criteria for ADHD, conduct disorder (CD), and oppositional defiant disorder (ODD). Anxiety and Depression Screener items are also included in order to provide coverage of two internalizing problems frequently associated with ADHD. The Conners 3 also provides two index scores: the Conners 3 ADHD Index and the Conners 3 Global Index. The assessment also includes validity scales (positive impression, negative impression, and inconsistency index), severe conduct critical items, impairment items (home, school, and social settings), and open-ended additional questions (additional concerns, strengths).

The Conners 3 offers full-length, short, and index form options. The full-length forms convey the most detailed information of all the forms. The short forms are useful when the administration of the full-length version is not possible or practical (e.g., when multiple administrations over time are required). The index forms are useful in screening and treatment monitoring situations. Administration requires approximately 20 min for the full-length forms, 10 min for the short forms, and approximately 5 min for the index forms. Raw scores are converted to age- and gender-based standard scores (including *T*-scores and percentiles). Results from the DSM-IV-TR symptom scales are also reported in terms of symptom counts, that is, whether or not the minimum symptom requirements set by the DSM-IV-TR have been met. The Conners 3 ADHD Index produces a probability score, which indicates whether the youth's scores are more like youth with ADHD, or with those from the general population.

The Conners 3 normative sample consists of 3,400 assessments (1,200 parent, 1,200 teacher, and 1,000 self-report assessments) including 50 boys and 50 girls from each age (6–18 years for the parent and teacher report, 8–18 years for the self-report). The racial/ethnic distribution of this sample closely matches the US population. Approximately 2,100 clinical cases were also collected from youth with following diagnoses: ADHD (including all three ADHD subtypes: ADHD predominantly inattentive type, ADHD predominantly hyperactive-impulsive type, and ADHD combined type), disruptive behavior disorders, learning disorders, anxiety disorders, major depressive disorder, bipolar disorder, and pervasive developmental disorders.

Historical Background

The first versions of the Conners Parent and Teacher Rating Scales were developed at the Harriet Lane Clinic of the John Hopkins Hospital in the 1960s during Dr. Leon Eisenberg's controlled studies of psychotherapy and medications. The purpose of the original scales was to serve as the basis for a detailed interview about a child's problems. The first version of the parent scale contained items grouped in terms of problems (e.g., sleep, eating, temper, friendships, school). The teacher scale included items related to functioning within the classroom setting (e.g., group participation, attitude towards authority). The earliest research studies on the scales supported the research properties of the scales. For example, the very first study on the teacher scales (Conners, 1969) demonstrated adequate test-retest reliability. In addition, good sensitivity to drug treatment and nondrug therapy effects were established.

The scales were first formally published in 1989 as the Conners Rating Scales (CRS™, Conners, 1989, 1990) and offered a standard format with normative data, detailed information about the psychometric properties and the proper use of the scales, and the hand-scorable "QuikScore™" form allowing for easy administration and scoring. The CRS was later revised (CRS-R; Conners, 1997) to offer a self-report component and scales linked to the *Diagnostic and Statistical Manual of Mental Disorders* – Fourth Edition (American Psychiatric Association, 1994) criteria for ADHD. The Conners 3, the latest revision of the Conners Rating Scales, incorporates the key features of its predecessors with updated normative data and psychometric properties, an age range specific to the assessment of school-aged children, and an increased focus on the assessment of ADHD, associated features, and the disorders most commonly comorbid with ADHD.

Psychometric Data

Results of reliability analyses revealed that the Conners 3 forms have high levels of internal consistency, with

Cronbach's alpha ranging from 0.77 to 0.97 (mean Cronbach'a alpha = 0.90), and excellent temporal stability, with test-retest correlations ranging from 0.71 to 0.98 (mean $r = 0.83$, all correlations, $p < 0.001$). There is also a great deal of consistency between multiple parent and/or teacher ratings of the same youth, with inter-rater reliability coefficients ranging from 0.52 to 0.94 (mean $r = 0.77$, all correlations, $p < 0.001$).

The Conners 3 Manual reports a variety of studies demonstrating convergent/divergent validity through correlations of Conners 3 scales scores with other related measures of childhood psychopathology. Overall, scales that assess similar constructs tended to be moderately to strongly intercorrelated, while scales that did not assess similar constructs tended to have smaller correlations. Table 1 provides highlights from these analyses.

Results from discriminative validity analyses indicated that the Conners 3 scores accurately discriminate between relevant groups. Results from a series of multivariate analysis of covariance revealed that, for all scales, the means for the target clinical groups were significantly higher than the means for the general population and other clinical groups (e.g., youth diagnosed with ADHD, predominantly hyperactive-impulsive type scored significantly higher on the hyperactivity/impulsivity and ADHD hyperactive-impulsive scales than did youth without a clinical diagnoses, as well as youth diagnosed with other disorders). The sizes of the group membership effects (as determined with partial η^2) were moderate to large, on average, accounting for 19.1% of the variance in scores. In terms of the classification accuracy of the scores (as determined by a series of discriminant function analyses), the mean overall correct classification rate was 75.6% across all forms.

Clinical Uses

The Conners 3 can be used as an assessment tool as well as in planning and monitoring treatment plans. Standardized scores allow for the objective comparison of an individual with age- and gender-based expectations. Correspondence of items with DSM-IV-TR symptomatic criteria for ADHD, CD, and ODD in combination with information about associated features and level of impairment facilitates differential diagnosis decisions in clinical practice. The Conners 3 offers a scoring feature, which links the assessment results to areas of eligibility under the Individuals with Disabilities Improvement Act of 2004 (IDEA 2004) making the assessment useful for identification of appropriate educational classification and/or services for students in the public school system. The Conners 3 can also be used as an assessment tool in screening and research contexts.

In addition to being an assessment tool, the Conners 3 forms can be employed as a tool for planning, monitoring, and evaluating treatment plans. Elevated scores from the Conners 3 suggest areas to target in treatment, with individual item responses can be used to guide decisions about specific behaviors requiring intervention. The assessment can also be used in treatment monitoring situations, for example to monitor the effectiveness of an individual's response to treatment or to evaluate an intervention program.

The Conners 3, at the time of writing, is too recent to have generated research literature, but empirical studies with earlier versions of the forms have demonstrated the utility of the assessment in treatment outcome studies, epidemiological and etiological studies of ADHD and other behavior problems, construct and discriminative validity studies, correlational studies, and cross-cultural studies.

Conners 3rd Edition (Conners 3; Conners 2008). Table 1 Overview of Correlations between the Conners 3 and Other Measures

Conners 3 scales	Other measures	*r* (range)
Inattention, ADHD inattentive	BASC-2: Attention problems	0.52–0.89
	ASEBA: Attention problems	0.72–0.96
Hyperactivity/Impulsivity, ADHD hyperactive-impulsive	BASC-2: Hyperactivity	0.41–0.91
	BRIEF: Inhibit	0.76–0.92
Executive functioning	BASC-2: Executive functioning	0.43–0.68
	BRIEF: Plan/Organize	0.70–0.87
Aggression, conduct disorder, oppositional defiant disorder	BASC-2:Aggression	0.59–0.95
	ASEBA: Aggressive behavior	0.58–0.93
Peer relations	ASEBA: Social problems	0.72–0.84

Note. All *rs* significant, $p < .05$. BASC-2 = Behavior Assessment System for Children, Second Edition; ASEBA = Achenbach System of Empirically Based Assessment; BRIEF = Behavior Rating Inventory of Executive Function.

Cross References

- ▶ Attention Deficit Hyperactivity Disorder
- ▶ Conners Comprehensive Behavior Rating Scales
- ▶ Conners Continuous Performance Test-II
- ▶ Individuals with Disabilities Education Act

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Conners Comprehensive Behavior Rating Scale

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Description

The Conners Comprehensive Behavior Rating Scales (Conners CBRS; Conners, 2008) is a comprehensive assessment tool, which can be used to assess a wide range of behavioral, emotional, social, and academic issues in school-aged youth. The Conners CBRS is a multi-informant assessment with forms for parents, teachers, and youth. Parent and teacher ratings can be obtained about youth aged 6–18 years, and youth aged 8–18 years can complete the self-report. The assessment features multiple Content scales that assess emotional distress, aggressive behaviors, academic difficulties, hyperactivity/impulsivity, social problems, separation fears, perfectionistic and compulsive behaviors, violence potential, and physical symptoms. Scales are also included to assess the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association; APA, 2000) diagnostic criteria for symptoms of generalized anxiety disorder, separation anxiety disorder, social phobia, obsessive compulsive disorder, major depressive episode, manic episode autistic disorder, Asperger's Disorder, attention deficit/hyperactivity disorder (ADHD), conduct disorder, and oppositional defiant disorder. Other clinical indicators are also included for other potential problem areas, including bullying, enuresis/encopresis, panic attack, pervasive developmental disorder, pica, post-traumatic stress disorder, specific phobia, substance use, tics, and trichotillomania. The assessment also includes the Conners Clinical Index (Conners CI), Severe Conduct and Self Harm Critical items, Validity scales (Positive Impression, Negative Impression, and Inconsistency Index), Impairment items (home, school, and social settings), and open-ended Additional Questions (additional concerns, strengths).

In addition to the Conners CBRS form, the assessment also offers the Conners CI as a standalone form. The Conners CBRS forms provide a comprehensive view of a youth's behavioral, social, emotional, and academic functioning. The Conners CI is a brief 24-item index with items from the Conners CBRS form that best differentiate youth with a clinical diagnosis from youth in the general population.

Administration requires approximately 25 min for the Conners CBRS forms and 10 min for the Conners Clinical Index form. Raw scores are converted to age- and gender-based standard scores (including *T*-scores and percentiles). Results from the DSM-IV-TR symptom scales are also reported in terms of symptom counts, that is, whether or not the minimum symptom requirements set by the DSM-IV-TR have been met. The Conners CI produces a probability score, which indicates whether the youth's scores are more like youth with a clinical diagnosis (disruptive behavior disorder, learning/language disorder, mood disorder, anxiety disorder, or ADHD) or with those from the general population.

The Conners CBRS normative sample consists of 3,400 assessments (1,200 parent, 1,200 teacher, and 1,000 self-report assessments) including 50 boys and 50 girls from each age (6–18 years for the parent and teacher report, 8–18 years for the self-report). The racial/ethnic distribution of this sample closely matches the US population. Approximately 2,000 clinical cases were also collected from youth with following diagnoses: ADHD, disruptive behavior disorders, learning disorders, anxiety disorders, major depressive disorder, bipolar disorder, and pervasive developmental disorders.

Historical Background

The Conners CBRS is a new assessment tool, which is built on the foundation of the Conners Rating Scales. The Conners Rating Scales are a widely used and well validated assessment of ADHD and related issues. When updating and revising the latest version of the scales (Conners 3rd Edition; Conners, 2008), the need for a comprehensive assessment of behavioral, social, emotional, and academic concerns became evident. The Conners CBRS was therefore developed to provide clinicians with an assessment and treatment planning/monitoring tool, which would address a broad range of clinical issues in school-aged youth and which could be linked to diagnostic and intervention systems such as the DSM-IV-TR (APA, 2000) and Individuals with Disabilities Education Improvement Act of 2004.

Psychometric Data

Results of reliability analyses revealed that the Conners CBRS assessments have high levels of internal consistency, with Cronbach's alpha ranging from 0.69 to 0.97 (mean Cronbach'a alpha = 0.86), and excellent temporal stability, with test-retest correlations ranging from 0.56 to 0.96 (mean $r = 0.81$, all correlations, $p < 0.001$). There is also a great deal of consistency between multiple parent and/or teacher ratings of the same youth, with inter-rater reliability coefficients ranging from 0.53 to 0.89 (mean $r = 0.74$, all correlations, $p < 0.001$).

The Conners CBRS Manual reports a variety of studies demonstrating convergent/divergent validity through correlations of Conners CBRS scales scores with other related measures of childhood psychopathology. Overall, scales that assess similar constructs tended to be moderately to strongly intercorrelated, while scales that did not assess similar constructs tended to have smaller correlations. Table 1 provides highlights from these analyses.

Results from discriminative validity analyses indicated that the Conners CBRS scores accurately discriminate between relevant groups. Results from a series of multivariate analysis of covariance revealed that, for all scales, the means for the target clinical groups were significantly higher than the means for the general population and other clinical groups (e.g., youth diagnosed with a disruptive behavior disorder scored significantly higher on the Aggressive Behavior and Violence Potential scales than did youth without a clinical diagnoses, as well as youth diagnosed with other disorders). The sizes of the group

Conners Comprehensive Behavior Rating Scale. Table 1 Overview of correlations between the Conners CBRS and other measures

Conners CBRS Scale		Other measures	*r*
Conners CBRS Content Scales	Emotional distress	BASC-2: Anxiety	0.47–0.87
		ASEBA: Anxious/Depressed	0.53–0.85
	Academic difficulties	BASC-2: Learning problems	0.78–0.93
	Aggressive behaviors, violence potential	ASEBA: Aggressive behavior	0.60–0.96
	Physical symptoms	BASC-2 somatization	0.59–0.78
DSM-IV-TR Symptom Scales	ADHD inattentive	ASEBA: Attention problems	0.72–0.91
	ADHD hyperactive-impulsive	BRIEF: Inhibit	0.74–0.89
	Major depressive episode	BASC-2: Depression	0.38–0.71
		ASEBA: Anxious/Depressed	0.43–0.83
	Generalized anxiety disorder	BASC-2: Anxiety	0.46–0.67
		ASEBA: Anxious/Depressed	0.51–0.83
	Social Phobia	MASC: Social anxiety	0.62–0.68
	Separation anxiety disorder	MASC: Separation/Panic Scale	0.42–0.53
	Autistic disorder, Asperger's disorder	BASC-2: Developmental social disorders	0.43–0.69
		ASEBA: Social problems	0.64–0.80

Note. All *rs* significant, $p < .05$. BASC-2 = Behavior Assessment System for Children, Second Edition; ASEBA = Achenbach System of Empirically Based Assessment; BRIEF = Behavior Rating Inventory of Executive Function; MASC = Multidimensional Anxiety Scale for Children.

membership effects (as determined with partial η^2) were moderate to large, on average, accounting for 14.5% of the variance in scores. In terms of the classification accuracy of the scores (as determined by a series of discriminant function analyses), the mean overall correct classification rate was 78% across all forms.

Clinical Uses

The Conners CBRS can be used as an assessment tool as well as in planning and monitoring treatment plans. Standardized scores allow for the objective comparison of an individual with age- and gender-based expectations. Correspondence of items with DSM-IV-TR, symptomatic criteria for a number of disorders in combination with information about the youth's level of impairment, facilitates differential diagnosis decisions in clinical practice. The Conners CBRS offers a scoring feature, which links the assessment results to areas of eligibility under the Individuals with Disabilities Improvement Act of 2004 (IDEA, 2004), making the assessment useful for identification of appropriate educational classification and/or services for students in the public school system. The Conners CBRS forms can also be used as an assessment tool in screening and research contexts.

In addition to being an assessment tool, the Conners CBRS can be employed as a tool for planning, monitoring, and evaluating treatment plans. Elevated scores from the Conners CBRS suggest areas to target in treatment, with individual item responses can be used to guide decisions about specific behaviors requiring intervention. The assessment can also be used in treatment monitoring situations, for example to monitor the effectiveness of an individual's response to treatment or to evaluate an intervention program.

Cross References

- ▶ Conners 3rd Edition
- ▶ Conners Continuous Performance Test
- ▶ Differential Diagnosis
- ▶ Individuals with Disabilities Education Act

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Conners Rating Scales

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Synonyms

CPRS; CRS

Description

The Conners Rating Scales are used as a general screening tool for the detection of problematic behaviors, and Attention Deficit Hyperactivity Disorder (ADHD) symptoms in particular, in children and, more recently, in adults.

Historical Background

Rating scales and symptom checklists provide an effective, quick, and standard approach to the measurement of problematic behaviors observed in children. The Conners rating scales were initially developed as comprehensive checklists of basic presenting problems of children. A re-standardization took place in the late 1990s, which was designed to provide a stronger empirical base and a more narrowed focus of common behavioral problems in childhood. The revised scales were developed with norms derived from a large, representative sample of North American children, using confirmatory factor analyses to develop a definitive factor structure. The revised scales focused on behaviors directly related to ADHD and its associated behaviors. The item content was updated to reflect the recent knowledge and developments concerning ADHD. The revised scale's content contained fewer items, yet the authors claimed that with greater focus on ADHD-related behaviors and concordance between scale items and current conceptualizations of ADHD, the CPRS-R provided better discriminatory power for detecting ADHD children than previous versions.

Psychometric Data

The authors report that the psychometric properties of the revised scale are adequate as demonstrated by good internal reliability coefficients, high test–retest reliability, effective discriminatory power, and predictive and structural validity. Various researchers have suggested three main areas for the application of Conners scales: as a general screening tool for the detection of problematic behaviors in children, as a complimentary tool for clarifying a specific diagnosis, and as a measurement tool for the assessment of treatment results.

Clinical Uses

The Conners scales are designed to assess symptoms as reported by various informants (e.g., parents, teachers, self-report for adolescents) using corresponding factor structures. There are also short and long forms that vary from approximately 20 to 80 items. The Conners ratings have also been compared across cultures, including (for example) Italian, British, Chinese, Brazilian, and New Zealand cultures and, consequently, translated into a number of different languages, including Sudanese Arabic, Turkish, and Hindi.

The Conners Adult ADHD Rating Scale (CAARS) was more recently developed to assess ADHD symptomatology in adults. The rating scale was designed to be completed by respondents about themselves or about a significant other. Factor analyses used to select items and determine the structure of the CAARS scale were based on data from a derivation sample consisting of 839 normal adults and from a clinical sample consisting of 167 adult. Four major dimensions were reported: three corresponded to the core features of ADHD seen in children and the fourth, a secondary consequence of ADHD: (1) Inattention/Executive Functioning, (2) Hyperactivity/Restlessness, (3) Impulsivity/Emotional Lability, and (4) Problems with Self-Concept. Internal reliability coefficients for the CAARS scales for four different age groups (18 years plus) were reported to be "excellent" (coefficient alphas ranging from 0.86 to 0.92 for males and females) and test–retest reliability over a month was felt to be strong (0.80–0.91).

Cross References

► Conners 3rd Edition (Conners 3; Conners 2008)
► Conners Comprehensive Behavior Rating Scales

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Conners' Continuous Performance Test (CPT)

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Description

The Conners Continuous Performance Test - 2 (CPT-2; Conners, 2000) is a computer administered test that is designed to assess problems with attention. The Conners CPT-2 presents 360 stimuli trials (i.e., individual letters) on the screen, with 1, 2, or 4 s between the presentation of letters (ISI: Inter-Stimulus Interval). The 360 trials are divided into 18 blocks of 20 trials each. The ISIs are counterbalanced across these blocks. Respondents are instructed to press the spacebar or the appropriate key on the mouse for any letter that appears, except the letter "X." The CPT takes fourteen min to administer excluding the recommended practice test. Over the duration of the test, 324 non-X stimuli (i.e., targets) appear, and the letter "X" (nontarget) appears 36 times. One of the primary advantages of this paradigm is the high proportion of targets that appear, yielding a larger pool of responses for generating the statistical output. Many statistics are computed including omission errors (i.e., failing to respond to a target letter), commission errors (i.e., responding to a nontarget), hit reaction time (the average speed of

correct presses to target letters), hit reaction time standard error (which assesses the consistency of responses to targets), detectability (the ability to discriminate between targets and nontargets), response style (assesses speed vs accuracy response tendencies), perseverations (reaction times less than 100 ms), hit reaction time by block (change in reaction time as the test progresses), standard error by block (change in response consistency as the test progresses), reaction time by ISI (change in mean reaction time across ISI), and standard error by ISI (change in response consistency across ISI). These statistics are converted to *T*-scores and can be interpreted in terms of various aspects of attention including inattention, impulsivity, and vigilance. The CPT-2 also provides a confidence index probability (expressed as a percentage out of 100) of the results being from a clinical as opposed to a nonclinical case.

T-scores are computed using General Population (not pre-identified with a clinical condition) norms (Conners, 2000) obtained for children and adults aged 6 years and above. The test is based on a total of 1,920 normative cases, out of which 52.8% of the cases were female, 47.0% were White, 27.0% Black, and 21.4% of other ethnic origin. Demographic information for age is shown in Table 1. T-scores are computed based on the age and gender of the respondent. Data was also collected for 378 ADHD cases, and 223 neurologically impaired cases (e.g., post-concussive, other organic brain syndrome, dementia).

Historical Background

The acronym "CPT" was first introduced by Rosvold, Mirsky, Sarason, Bransome, and Beck (1956) who used a particular version to detect attention lapses in subjects with petit mal epilepsy. In early CPTs such as the one they used, subjects were required to press a key when a target letter (e.g., "X") appeared or when the target letter appeared preceded by another letter (e.g., "AX"). While the CPT was being explored in clinical settings, Mackworth (1957) initiated basic vigilance studies. By changing basic test parameters, different CPT paradigms could be constructed. The CPT-2 paradigm was initially released as a DOS application (Conners CPT 3.0; Conners, 1994), and later renormed and converted into a windows application (Conners, 2000).

Psychometric Data

Reliability. Split-half reliability results were computed for the original standardization sample (Conners, 1994, 2000). The mean split-half reliability was 0.83 (range 0.66 to 0.95). Test-retest reliability is reported in Conners (2000) and is based on a small sample (n = 23). Test-retest correlation was high for the confidence index (r = 0.89), and moderate to high for omissions, commissions, Hit Reaction Time, Hit Reaction Time Standard Error, Variability, Detectability, and Response Style (range r = 0.43 to r = 0.84). The Block change and ISI change statistics were less consistent across test administrations (r = 0.05 to r = 0.51).

Validity. Validation of the CPT-2 focused on its two primary clinical uses: to help identify clinical problems with attention or potential neurological impairments and to monitor treatment effects. Conners (2000) compares means from the General Population normative data (n = 1483), and ADHD cases (n = 271) in an ANCOVA controlling for age and gender for 6–17 year olds. Performance of the ADHD group was significantly worse on the CPT-2 for all of the measures except commissions where no significant difference was found. Discriminant Function Analysis was also used to examine classification accuracy for the CPT-2. For 6–17 year olds, sensitivity is reported at 83% and specificity at 82%. Similar analyses were conducted on the adult samples. In addition to General Population data (n = 437), ADHD data (n = 107), neurologically impaired data was also available (n = 223). Performance of the ADHD group and neurologically impaired group was significantly worse than the General Population group for all of the CPT-2 measures. The neurologically impaired group also scored significantly worse than the ADHD group on several measures. They made significantly more omission errors, had significantly slower reaction times, had more variable reaction times, and their standard error was affected more by letter presentation speed (ISI).

Conners' Continuous Performance Test (CPT). Table 1 Age distribution of CPT-2 Normative Sample

Age	6–7	8–9	10–11	12–13	14–15	16–17	18–34	35–54	55+
n	88	283	369	258	261	224	237	146	54

Discriminant Function Analyses comparing ADHD to General Population cases yielded 87% specificity and 88% sensitivity. When comparing neurologically impaired cases to General Population cases, specificity was 92% and sensitivity was 85%.

In terms of treatment sensitivity, Conners, Casat, Gualtieri, and Weller (1996) examined the efficacy of bupropion in the treatment of children with ADHD and used the Conners' CPT as one of the efficacy assessment tools. A double-blind procedure was used with 72 ADHD children and 32 ADHD control children aged 6–12. Subjects were randomized to receive 3–6 mg/kg of buproprion per day or placebo twice daily. Significant treatment effects were found. Epstein et al. (2006) examined mean differences between 190 children who took a stimulant medication on the day of neurological testing and 126 children who took no stimulant medication of the day of testing. Children who were not on medication made significantly more omission errors and commission errors, had significantly slower reaction times, and were significantly more variable in their response speed. Boonstra, Kooij, Oosterlaan, Seargeant, and Buitelaar (2005) tested 43 adults with ADHD in a double-blind, cross-over, and placebo controlled study. The CPT-2 was administered during the third week of titrated treatment with methylphenidate and during the third week of treatment with placebo. Large medication effects were observed for commission errors, standard error of hit reaction time, and inattentiveness. A moderate effect was also observed for hit reaction time.

Clinical Uses

Although the CPT-2 cannot be used alone to make a diagnosis, it provides performance based information that aids in the diagnostic process. The performance based information from the CPT-2 complement informant reports commonly obtained from parents, teachers, or by self-report. Response patterns on the CPT-2 enable practitioners to better understand the type of deficits that might be present (e.g., Epstein, Erkanli, Conners, Klaric, Costello, & Angold, 2003; Hervey et al., 2006). For example, some response patterns might suggest inattentiveness or impulsivity, while other response patterns may indicate activation/arousal problems or difficulties maintaining vigilance. The CPT-2 has also commonly been used as a way of assessing treatment effectiveness. Careful charting of CPT scores while testing different drug dosages can often be helpful in determining optimal dosage levels.

Cross References

- ▶ Attention Deficit, Hyperactivity Disorder
- ▶ Conners 3rd Edition (Conners 3; Conners 2008)
- ▶ Conners Comprehensive Behavioral Rating Scale

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Consciousness

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Synonyms

Alertness; Awareness; Mindfulness; Self-awareness; Wakefulness

Definition

Consciousness comes from the Latin word "conscientia" which means "knowledge-within", or knowledge that is shared. Today the term is used to describe the experience of "self" as distinct from the external environment. It is characterized by experiences of alertness, self-awareness, and attention of oneself relative to the environment relative to the self; i.e., identify, which in turn involves awareness of one's own perceptions, associations, emotional experience, and the cognitive interpretation of these experiences. More narrowly, consciousness is often defined level of arousal, wakefulness, alertness, responsiveness, and adaptability in contrast to states of coma or sleep.

However, consciousness has defied unitary definition, perhaps because it is intrinsically bound to subjective experience.

Historical Background

The nature of human consciousness has been a primary topic of philosophical inquiry throughout the ages. The ancient Greek philosophers contemplated questions regarding the relationship between external and objective reality. For example, Plato recognized that humans can think and speak, and yet not be fully aware of their larger realm and whether their observations were valid with respect to external reality. His cave allegory was an effort to describe this problem. For medieval religious philosophers (e.g., Aquinas), it referred to the act of applying moral knowledge to one's actions. Descartes extended the concept of "conscientia" to include the idea that it involves a psychological state of mind, and used separate terms to explain this level of experience from moral knowledge, though he did not formally employ the modern concept of consciousness in his dualism of mind–body interaction. John Locke later wrote "I may be held morally responsible for the act of which I am conscious of having achieved; and my personal identity; my-self goes as far as my consciousness extends itself", its first modern usage associated with the idea of "self" and personal identity. Interestingly, Locke still linked consciousness to the ideas of morality and responsibility. The nature of consciousness was a recurrent theme of renaissance philosophers.

The quest to better understand the nature of consciousness dominated the new science of psychology at it evolved in the late nineteenth century. Wilhelm Wundt believed that identifying the constituents of human consciousness was a primary task for psychology. He postulated that "apperception" represented the core activity of the mind and was the interface between internal and external experience. Notably, Wundt's primary method for exploring these processes was introspection, which essentially is the process of looking inward or self-reflection. These ideas evolved into structuralism, the first dominant theoretical school of psychology.

A dramatic shift away from consideration of consciousness occurred during much of the twentieth century as behaviorism became the dominant perspective, as consciousness was viewed as metaphysical and beyond empirical observation. Even cognitive scientists tended to steer away from tackling this construct, probably because of the difficulties associated with experimentally studying it. Yet, for clinicians working with the subjective experiences of their neurological and psychiatric patients, consciousness was difficult to avoid. When the neurologist Babinski (1914) first described patients who were unaware of their own deficits (i.e., anosognosia) he was essentially addressing the issue of consciousness and self-awareness. Initially, the problem of consciousness was handled by narrowing the meaning of the term to refer to the state of being awake, alert and aware, in contrast to coma, stupor, or sleep, which in turn led to the idea of level of consciousness, which remains a standard part of mental status examinations.

Current Neuropsychological Knowledge

Over the past 2 decades there has been a major resurgence of interest and research effort directed at the nature of consciousness, along with growing recognition that the construct is a necessary one, as consciousness describes an important element of human experience, the sense of self. A number of scientific developments set the stage for this shift in zeitgeist. The evolution of the field of cognitive science provided empirical methods for studying covert "mental" processes. Also as cognitive processes such as attention and memory were better understood, foundations were established for studying phenomena like intention and self-awareness. Computational neuroscience was also influential in this regard. It is quite difficult to operationalize or conceptualize consciousness as a single node in a top-down modular cognitive framework. This is less of a problem for computational theories that take a bottom-up approach, such as the parallel distributed processing (PDP) models proposed by Rummelhart and McClelland (1986), as consciousness can be conceptualized as an emergent byproduct of associative networks acting together with feedback or

feed-forward organization. Another major impetus for consciousness becoming an acceptable topic for scientific inquiry was advances in the field of neuroscience itself. Functional imaging methods have made it easier to measure brain responses associated with subjective cognitive states, providing evidence that these states have valid neurobiological underpinnings. Furthermore, growing recognition of the brain's complexity has played an important role. There is now general consensus that cognition involves interactions among large networks of cortical neurons, which communicate across larger functional systems in an integrated manner. This perspective enabled cognitive neuroscientists to shift away from having to view consciousness as a unitary process sitting on top of the pyramid of cognitive functions.

Several lines of research and clinical evidence have had particular bearing on current knowledge regarding the neuropsychology of consciousness. The effect of various types of drugs, most notably hallucinogens, opiates, and alcohol on conscious experience provides one of the most clear cut illustrations of the fact that even among healthy people consciousness is not a static phenomenon. One's sense of reality, identify and self-awareness can be dramatically altered by drugs such as LSD, presumably by altering the flow of information processing. Case studies have consistently shown that disruption of self-awareness as a function of damage to specific brain regions. Furthermore, clinicians working with patients experiencing neurodegenerative dementias have long been aware of dramatic impoverishment in the quality of conscious experience as the disease progresses, corresponding with the extent of cortical atrophy. Among patients with Alzheimer's disease (AD) and other neuronal dementias, a loss of "self" is one of the hallmark features of the end-stage condition. In fact, it is when AD patients lose their ability to respond to people who are familiar and meaningful to them, and when they lose awareness of their own identify, their personal memories, and the nature of their cognitive problems that families typically experience greatest despair. In fact, it is often this change, along with an inability for self-care that leads to nursing home placement rather than the amnestic disturbance per se. The loss of identity in AD patients usually corresponds with personality changes and profound impoverishment in abstraction and associative ability. While other cognitive functions like language are often also severely impaired at this point, it may be difficult to fully appreciate these changes, though the quality of emptiness and loss of self that may ultimately be the characteristic that best defines severe dementia rather than impairment in a particular cognitive domain.

In contrast to AD, patients with certain other degenerative conditions often show general preservation of consciousness despite having marked impairments of other functions. For example, patients with severe Parkinson's disease typically have major motor symptoms, cognitive control problems, including executive and attention dysfunction. They may also show blunting of emotional response and altered emotional functioning. Yet, they are often painfully aware of their deficits, and have greater preservation of sense of self than the AD patient. Similarly, patients with Multiple Sclerosis (MS) sometimes exhibit pseudobulbar affect. They are affectively labile with dramatic shift in the outward expression of emotion, but yet they report not feeling the emotion that corresponds to this expression. Despite these problems with affective and behavioral control, patients with MS typically are very aware of their deficits and may feel locked in because of their symptoms.

Alterations in self-awareness and consciousness also occur as a result of damage to specific brain regions. This fact is significant since it suggests that certain functional anatomic systems play an important role in the experience of consciousness, though probably not as a function of any one of these systems alone. Subcortical lesions affecting the mid-brain, brain stem and thalamus often cause dramatic alterations in level of consciousness. The reticular system which generates ascending activation to cortical areas plays a well established role in this phenomenon, with damage to this system contributing to problems with arousal and alertness. Damage to these areas frequently underlies coma, and the electrical activity measured by EEG is amplified by generators in these systems. Persistent vegetative state is a clinical illustration of this, as these unfortunate patients exhibit impaired higher cortical functions, have persevered circadian sleep-wake cycles and autonomic function. Compared to healthy people who are awake, patients with this syndrome have impaired connectivity between brainstem and thalamic areas and the cortex. This results in a reduced cortical activation, with generalized slowing consistent with coma. These subcortical areas are clearly essential for maintaining normal consciousness, since disconnecting them from cortical areas impairs alertness, attention, and other cognitive functions linked to consciousness. The activation and arousal associated with this functional anatomic system makes an essential contribution to is a necessary part of the maintaining consciousness, thought this activation is not sufficient to account for the full dynamic of consciousness (i.e., self-awareness and sense of identity).

The thalamus appears to play an important role in consciousness, but as a convergence site of reticular

activation from lower brain systems and because of its role in integrating this activation with cortical input. Bilateral ablation of the central medial zone of the intralaminar nucleus of the thalamus produces coma and persistent vegetative states. This thalamic area is affected by general anaesthetics and drugs that cause sedation, suggesting that it is necessary for anti-psychotic drugs. This evidence suggests that a functioning thalamus is necessary, but not sufficient, for human consciousness, though again this area alone is not sufficient to account for all aspects of consciousness. Cortical and limbic functions are necessary elements of normal consciousness. Patients with bilateral occipital damage who exhibit blind sight are clearly experiencing some alteration in consciousness and self-awareness. Yet, this disturbance may be relative focal and limited to vision. For these individuals, altered consciousness has not affected all aspects of self-awareness and identity. Damage to other cortical areas may also produce specific impairments of awareness and self-consciousness. For example, the neglect syndrome commonly associated with right inferior parietal damage often results in anosognosia and impaired awareness of what is being neglected. For some of these patients, the alteration in awareness and consciousness extends beyond the limits of the spatial disturbance that is observed. Yet, typically patients with these lesions do not lose their sense of self completely. They may show diminished ability for self-reflect, but usually show abilities in this regard as long as not tied to the types of spatial processing that is affected.

The frontal cortex seems to have be the cortical area with greatest influence on consciousness, self-regulation and self-referential processes. The famous case of Phineus Gage, the railroad worker who suffered severe frontal damage when a spike penetrated his brain provided historical illustration of this. Following his injury Gage became indifferent to social consequences and showed little self-regard for his own behavior (Harlow, 1848). While it is impossible to accurately evaluate the nature of change in his subjective sense of consciousness and self-awareness based on the historical record, it seems clear from clinical descriptions that his self-awareness was dramatically altered. In modern case study, Eslinger and his colleagues (1986) have demonstrated impaired empathy associated with frontal lobe disturbance, which affects social reasoning, judgment and perspective taking. Interestingly, this disorder of empathy also involves problems with moral reasoning, a notion consistent with early philosophical ideas regarding consciousness, which as discussed earlier conceptualized consciousness as tied to man's capacity for moral knowledge.

There is strong evidence from studies of neglect syndrome that medial frontal and anterior cingulate cortical areas play a major role in "intention" (Cohen, 1993). The intent and plan for action seems to be an important element of consciousness, as this capacity is linked to the experience of drive and spontaneity. An illustration of this is seen in patients who undergo cingulotomy, as their primary long-term neurocognitive deficit involves diminished performance on tests requiring intention, and also reduced spontaneity, initiative, and creative impulse. It is also noteworthy that syndromes that result in alterations of consciousness that involve delusions and a fractured sense of reality, such as reduplicative para-amnesia and Capgrass syndrome, tend to occur among patients with frontal lobe damage, often involving the non-dominant hemisphere. In cases of reduplicative paramnesia, patients may feel with certainty that while the house in which they are living looks identical to their real home, that it is in fact a replica of their home. When queried these patients usually acknowledge the logical implausibility of their belief, but state that they are certain because of some feeling they have, suggesting a relationship between emotional response and the processing of these frontal systems. Primate studies support the role of the frontal cortex in this regard, and led Crick and Koch (1995) to propose that consciousness is largely dependent on the resonance of thalamo-cortical systems acting in a recursive manner.

The role of memory and its underlying functional neuroanatomic systems raises a number of interesting issues for consciousness. Patients like HM, who have suffered bilateral damage to hippocampal or paralimbic areas, lose their ability to form new episodic memories. Yet, they typically retain retrograde memory for events that happened earlier in their life, which enables them to retain some sense of personal identity based on these recollections. Yet, they have lost the ability to update these memories or to adapt these memories to new circumstances. Clearly this results in an alteration of consciousness, though they may still report knowing who they are or what they think about certain topics.

In addition to the contribution of specific cortical and subcortical areas to the experience of consciousness, there is a rich history of research on brain laterality, specifically the effects of disconnection between the left and right hemisphere of the brain. The corpus callosum provides the white matter linkage between the hemispheres. Roger Sperry observed remarkable alterations in consciousness and awareness among patients who underwent surgical severing of the corpus callosum (split brain) for treatment of intractable seizures. When the two hemispheres are no longer able to communicate,

each processes and interprets information presented to it in different ways. For example, the left hemisphere is typically more involved in language processing, and therefore tended to be aware of verbal elements of information during tasks compared to the right hemisphere. Because of a lack of integration between the cortical hemispheres, it is possible to dissociate elements of consciousness tied to each side of the brain. Yet, following this procedure, it was only under experimentally controlled conditions that major dissociations occurred. Post-surgically, people still experienced a single identity in everyday experience, suggesting that splitting the two hemispheres does not completely disrupt the sense of a unified consciousness.

Neuropsychological Models of Consciousness

Consideration of the neuropsychological literature to date has established the foundations for the cognitive neuroscience of consciousness. Over the past 15 years, theoretical models have been developed to explain how the brain creates "mind" and "consciousness". These models include contributions from philosophers (Dennett, 1991; Churchland, 2005), neuropsychologists/behavioral neurologists (Cohen, 1993; Damasio, 1995, 2000) and cognitive neuroscientists (Shallice, 1978; Eccles, 1994; Crick and Koch, 1995; McClelland et al., 1997; Grossberg, 1999). It is beyond the current scope to review each of their models. However, there are several features common to each, as well as a few fundamental distinctions between these models, as summarized below.

Most current models of consciousness take the perspective that consciousness is not localized to a single brain region, but rather is an emergent function of multiple systems acting in an integrated fashion. (1) Almost all researchers and theorists agree that lower brain areas (e.g., reticular system) that energize the brain and lead to altering levels of activation are a necessary requirement for consciousness, but not sufficient to explain the phenomenon. (2) The thalamus seems to play and essential role, particularly as a nexus for this activation and for integration and gating of cortical information. (3) Frontal regions appear to play several important roles, including the generation and sustaining of intention, switching and gating of information, and the enabling of feedback, and feed-forward mechanisms that enable reprocessing of certain associative information and feedback control for gating of this information as it flows from posterior cortical systems. (4) Limbic areas, such as the amygdala, likely play a significant role, by linking emotional valence to particular associative information. In interaction with cortical areas, these limbic areas enable amplification of affective signals, and also their regulation, providing for the intensity and richness of emotional affective experience. (5) Some type of mechanism that enables temporal binding of associative experiences in an integrated way likely occurs that creates the feeling of a unified conscious experience and sense of single identity, "I".

Some of the distinctions that exist between theories of consciousness relate to the emphasis that is placed on particular cognitive functions. Some recent investigators have emphasized the role of posterior brain systems involved in visual processing to a greater extent. A number of theories emphasize the role of frontal brain systems. For example, one of the first neuroscientific models of consciousness proposed by Shalice (1978) viewed consciousness as a byproduct of dominant action systems that govern the stream of thought processes. As discussed previously, many models have focused on frontal systems with varying degrees of emphasis on particular processes. Damasio's model of consciousness emphasizes the critical role of emotional processing systems, as well as the somatic or visceral activity of the body as a whole.

Perhaps the greatest distinction between theories of consciousness stems from the extent to which it is viewed as distinct from the physical processes that comprise, or essentially indistinguishable from these physical processes, as Dennett argues. Furthermore, some of these models take more of a top-down approach, whereas others view consciousness from a bottom-up perspective, as an emergent byproduct of all of the more basic associative processes that underlie cognition. The PDP approach of McClelland and Rummelhart (1986, 1997), and other neurocomputational theories (e.g., Grossberg, 1999) take a more bottom-up approach and tend to view consciousness as the sum total of these distributed processes. Despite these differences, there seems to be little disagreement that consciousness is a complex cognitive phenomenon that results from the interaction of multiple brain systems and processes, which in an integrated coherent manner provides for the sense of self, universally experienced by healthy humans.

Future Directions

The cognitive and neurobiological bases of consciousness remain major frontiers in neuropsychology. Clinicians evaluating patients following acute brain injury routinely assess level of impaired consciousness is (i.e., the degree of

coma, stupor or lethargy), though this level of analysis does not fully capture the richness of normal conscious experience. None the less, clinical research is likely to yield greater precision in the assessment of "levels" of consciousness.

Some progress has been made in understanding how consciousness arises as a by-product of other cognitive functions, most notably emotional experience, attention and executive functioning. Computation neuroscience has offered a perspective for explaining consciousness as an emergent property of complex neural systems. Yet, the measurement of the qualitative experience that comprises consciousness is still far from becoming part of mainstream neuropsychological assessment, or from being fully operationalized as a cognitive construct or neuropsychological domain. Greatest progress has been made with respect to the study of anosognosia, and recent efforts to better understand the relationship between subjective experience of cognitive impairments and also cognitive and emotional experience and neuropsychological performance. This seems to be a fertile area of continued investigation. Functional imaging approaches have provided considerable insight into the neural bases of consciousness and likely will continue to be on the cutting edge of these efforts, since they provide a means for observing brain response associated with subjective experience. Evidence of the attentional default network that emerged from functional imaging research illustrates the type of insight about the neural bases of consciousness that can be achieved from these approaches.

Cross References

- ▶ Anosognosia
- ▶ Attention
- ▶ Capgras Syndrome
- ▶ Minimally Conscious State
- ▶ Parallel Distributed Processing
- ▶ Reduplicative Paramnesia
- ▶ Reticular Activating System
- ▶ Split-Brain

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Consolidation

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Synonyms

Memory consolidation

Definition

Consolidation is the process of converting short-term memory to permanent long-term memory through the formation of new synapses.

Current Knowledge

History

The first written evidence of consolidation was by Marcus Fabius Quintilian, who stated "curious fact, of which the reason is not obvious, that the interval of a single night will greatly increase the strength of the memory" and "the power of recollection, which is the most important element of memory, undergoes a process of ripening and maturing during the time which intervenes." The term "consolidation" was coined by Georg Elias Müller and Alfons Pilzecker, whose conducted research between 1892 and 1900 and found that memory takes time to "consolidirung." Published in their article "Experimentelle Beiträge zur Lehre vom Gedächtnis," Müller and Pilzecker found that it takes time to form memories, and that these new memories can be disrupted for some time after formation.

Process of Consolidation

The process of consolidation occurs in the neocortex of the brain, with memory information sent from the hippocampus. Two specific processes are involved in consolidation based on the period of time needed to create long-term memories from the short-term memories. Synaptic consolidation occurs the quickest, consolidating memories within the first few hours of learning. System consolidation, on the other hand, takes between weeks to years for hippocampus-dependent memories to become independent of the hippocampus. Consolidation can occur more rapidly if new information is incorporated with an already existent schema. In addition, the largest amount of consolidation happens during rapid eye movement (REM) sleep.

Long-Term Memory

Studies in anterograde amnesia, such as with Patient H. M., have shown that lesions on the medial temporal lobe prevent long-term memory formation. Long-term memory is information that has been consolidated and is permanently stored in the brain. While consolidation is initiated in the limbic system, specifically in the hippocampus, long-term memories are stored throughout the cortex. When the memory is retrieved, it can be modified with recently acquired memories, thus strengthening the original memory.

Assessment and Measurement

Assessment of consolidation is done through testing short-term memory with material modality tests, then measuring long-term memory after a delay or a distraction. The participants will be given a list of words, for example, and asked to repeat them back to the examiner. After a period of time has passed (delay), the participants will be prompted to repeat the words again. The examiner may also measure consolidation by using a distraction to prevent the participants from rehearsing the information. To effectively measure consolidation, conditions must be controlled when testing both short-term memory and long-term memory.

Neuroimaging

Research by Takashima et al. tracked the brain's activity during the process of consolidation. As the brain consolidates short-term memories into long-term memories, the functional magnetic resonance imaging (fMRI) shows an increase in activity in the hippocampus. Once the brain switches from consolidation to memory retrieval, the hippocampal activity decreases and the ventral medial prefrontal region increases. Sterpenich et al. used fMRI to examine consolidation during sleep. The activation in the hippocampus decreased over time (3 days compared to 6 months), indicating that the memories were being stored in the neocortex and no longer dependent on the hippocampus. After 6 months, activity increased in the ventral medial prefrontal region.

Cross References

- ▶ Memory
- ▶ Short-Term Memory

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Consortium to Establish a Registry on Alzheimer's Disease

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Synonyms

CERAD

Description

The Consortium to Establish a Registry for Alzheimer's Disease (CERAD) neuropsychological battery was designed to provide an efficient, standardized method to evaluate cognitive functioning in individuals with Alzheimer's disease (AD). Originally employed in a large-scale longitudinal research program, tests were selected to provide coverage of the primary cognitive deficits associated with AD (e.g., memory, language, praxis, and intellectual status) and to have a range of difficulty appropriate for use through much of the course of the disease (Morris et al., 1989). Individual tests within the battery include well-known, commonly used measures such as the Mini-Mental State Exam (MMSE; Folstein, Folstein, & McHugh, 1975), a 15-item modified Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1978), and an Animal Naming verbal fluency test (Isaacs & Kennie, 1973). Also included are a ten-item word list memory test, with free recall (immediate and delayed) and recognition (delayed) conditions, and a constructional praxis test that requires examinees to copy four line drawings of increasing complexity (Rosen, Mohs, & Davis, 1984).

Administration time for the battery is 20–30 min for persons with AD; persons without AD may complete the battery in less time (Morris et al., 1989). As may be expected given the brevity of the battery and the range of cognitive ability of the target populations, floor and ceiling effects have been noted for individuals with later stage AD and for individuals without AD, respectively (Morris et al., 1989). Normative data are available based on the CERAD program control sample (Welsh et al., 1994), as well as from studies of specific demographic groups (see Clinical Uses).

Historical Background

The Consortium to Establish a Registry for Alzheimer's Disease (CERAD) was first funded by the National Institute on Aging in 1986 in response to a US Congressional mandate to collect epidemiological data concerning the incidence of AD. A nationwide consortium of 24 university medical centers (the CERAD program) developed, standardized, and evaluated methods for clinical, neuropsychological, neuropathologic, and neuroimaging assessments of AD. Data from the study population, including 1,094 patients with AD and 463 controls evaluated annually between 1987 and 1996, are available on CD-ROM for research purposes (CERAD, Box 3203, Duke University Medical Center, Durham, NC 27710). As the bulk of the CERAD data collection took place before the widespread use of cholinesterase inhibitor treatment for AD, these data provide a rich source of information as to the natural history of AD. A special issue of *Neurology* (49, suppl. 3) published in 1997 commemorated the tenth anniversary of the CERAD program and reviewed study design, implementation, and key findings from wide-ranging investigations of AD.

Psychometric Data

Psychometric data for the CERAD neuropsychological battery reported for participants enrolled over the first 19 months of the study indicated good interrater reliability across all tests, with intraclass correlation coefficients ranging from 0.92 to 1.0 (Morris et al., 1989). One month test–retest data showed variable reliability

(*r*'s from 0.36 to 0.90), with lower correlation coefficients for measures whose variance may have been constrained by ceiling effects (Morris et al., 1989). The efficacy of CERAD battery measures (individually and in combination) for detecting AD and for staging dementia severity has been evaluated using discriminant function models (Welsh, Butters, Hughes, Mohs, & Heyman, 1991; Welsh, Butters, Hughes, Mohs, & Heyman, 1992). The word list delayed recall measure was shown to most effectively differentiate those with AD from controls (correctly identifying 94% of controls and 86% of mild AD patients), although the ability of this measure to distinguish dementia severity groups was limited due to floor effects. For those with impaired scores on delayed recall, performance on the naming measure was shown to modestly increase the discrimination of those with mild from moderate AD (improvement in overall accuracy from 65 to 71%). Verbal Fluency and Constructional Praxis performances were shown to increase discrimination of those with moderate from severe AD (improvement in overall accuracy from 66 to 81%). Within a cognitively impaired sample, the CERAD battery has also been found to distinguish those with AD from those with schizophrenia (Davidson et al., 1996). Clinical–pathological correlations have shown association between CERAD neuropsychological performance and AD pathology on autopsy (Hulette et al., 1998). Data from the CERAD program have been used to identify predictors of time to institutionalization in patients with AD, but information from the CERAD neuropsychological battery (with the exception of the MMSE) was not considered in this study (Heyman, Peterson, Fillenbaum, & Pieper, 1997).

Clinical Uses

The chief objective of the CERAD neuropsychology task force was "to develop a brief, reliable battery of neuropsychological tests that would be widely used by researchers and clinicians to assess the cognitive status of patients with AD at entry and during long-term annual follow-up" (Welsh-Bohmer & Mohs, 1997). The CERAD neuropsychological battery has been widely used in research studies, including those completed at many of the NIH-sponsored Alzheimer's Disease Centers. Normative data based on 413 CERAD control subjects aged 50–89 have been published to facilitate clinical application (Welsh et al., 1994). These data are stratified by age, education, and gender, as these factors were shown to differentially impact test performance. Normative data for patients with AD have also been reported, in addition to annual rates of change on CERAD test scores over a 4-year period (Morris et al., 1993).

The CERAD battery has been extensively translated. Normative data for the CERAD are available for a wide range of racial/cultural/demographic groups (Bertolucci et al., 2001; Fillenbaum, Heyman, Huber, Ganguli, & Unverzagt, 2001; Fillenbaum et al., 2005; Ganguli et al., 1991; Ganguli et al., 1996; Guruje et al., 1995; Whyte et al., 2005). Beeri et al. (2006) provide normative data for individuals over the age of 85.

Methods for deriving a single summary score for the CERAD battery (excluding the MMSE) and normative data for this summary score for unimpaired, mild cognitive impairment (MCI), and AD groups have been published (Chandler et al., 2005). This total score was reportedly more consistent at differentiating these three groups from one another compared to the MMSE total score, which showed limited ability to distinguish patients with MCI from unimpaired individuals, and the word list delayed recall score, which showed limited ability to distinguish MCI from AD.

Some authors have described an overreliance of the CERAD battery on using verbal modalities of testing, at the expense of other relevant information. To address this issue, a visual memory measure based on recall of the Constructional Praxis test items has been proposed. Administration instructions and psychometric properties of a delayed recall and recognition condition of this measure have been reported (Spangenberg, Henderson, & Wagner, 1997).

In sum, the brevity of the CERAD neuropsychological battery, combined with the vast associated research base, make the CERAD a useful tool for assessment of cognitive function in cases of suspected dementia where "factors such as fatigue, motivation, or incapacity prevent a more extensive neuropsychological examination of cognitive capacities" (Welsh et al., 1994). Assessment in specialized memory clinics, however, would typically employ a more extensive neuropsychological battery.

Cross References

- ▶ Alzheimer's Disease
- ▶ Blessed Dementia Screen
- ▶ Clinical Dementia Rating Scale (CDR)
- ▶ Mattis Dementia Rating Scale (DRS)
- ▶ Mini-Mental State Exam (MMSE)

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Constraint Induced Therapy

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Definition

Constraint induced therapy (CIT) typically refers to a therapeutic intervention involving restraint of a non-affected upper extremity combined with intensive practice with the affected upper extremity (e.g., Alberts, Butler, & Wolf, 2004; Charles & Gordon, 2005; Taub & Uswatte, 2005, 2006).

Historical Background

CIT has its origins in primate unilateral deafferentation studies, where it was observed that animals avoided use

of their deafferented limb unless the intact limb was restrained (e.g., Taub & Uswatte, 2005, 2006). Concepts from CIT were then applied to humans in single case studies, case series, and small group studies, primarily with people who had sustained hemiplegia following stroke (Taub et al., 1993). The Extremity Constraint Induced Treatment (EXCITE) study is a randomized clinical trial involving over 200 individuals enrolled at 3–9 months post-stroke, using uniform CIT methods and measurement at six testing sites in the USA (Nichols-Larsen, Clark, Zeringue, Greenspan, & Blanton, 2005; Winstein et al., 2003; Wolf et al., 2006).

Rationale or Underlying Theory

CIT reflects the integration of a wide variety of research fields, including neuroplasticity, motor learning, and behavioral therapy (Sterr, Szameitat, Shen, & Freivogel, 2006). Two mechanisms are typically proposed as the basis for increased use of affected extremities in CIT: overcoming learned nonuse, and induction of use-dependant cortical organization (Morris & Taub, 2001).

Learned nonuse is said to occur following brain injury because unsuccessful motor attempts with the affected extremity leads to punishment (e.g., pain, incoordination, failure to meet the goal of the movement). Compensatory behavior also occurs when use of the non-affected extremity is positively reinforced.

CIT aims to counter-condition learned nonuse. For example, in animals, the functional limb is restrained and the animals complete forced, repetitive movements with the affected limb. This results in an alteration in the contingencies of reinforcement, such that compensatory use of the non-affected limb is no longer reinforced, and use of the affected limb is reinforced.

Use-dependant cortical reorganization is a second primary mechanism thought to underlie CIT. Neuroimaging and transcranial magnetic stimulation studies of the brain prior to and following CIT have suggested cortical reorganization around the infarct site, and recruitment of a large portion of neurons adjacent to those originally involved in the control of the limb (Dong, Dobkin, Cen, Wu, & Winstein, 2006; Morris & Taub, 2001; Park, Butler, Cavalheiro, Alberts, & Wolf, 2004).

Goals and Objectives

A major goal of CIT is to increase the use of the affected limb in real world environments (Sterr et al., 2006). In contrast to rehabilitation strategies that focus on developing compensatory techniques, CIT aims to optimize and develop residual functions (Lillie & Mateer, 2006). Although CIT has been shown to result in significant gains in motor activity, it should not be expected to restore or normalize movements to pre-injury levels (Taub & Uswatte, 2005).

Treatment Participants

CIT has been applied in a variety of populations, including upper limb hemiparesis following stroke, traumatic brain injury, and children with hemiplegia (Charles & Gordon, 2005; Lillie & Mateer, 2006; Shaw et al., 2005). Research has suggested significant improvement in a number of populations, including those with "chronic TBI" (identified as $>$ 1 year post-TBI; Shaw et al., 2005) and both "subacute" and "chronic stroke" (identified as $<$ 1 year post-stroke and $>$ 1 year post-stroke, respectively; Page, Levine, & Leonard, 2005).

Adherence to treatment and level of residual motor function prior to CIT is positively related to treatment gains (Shaw et al., 2005; Taub & Uswatte, 2005). Cognitive impairment is linked to less improvement (Morris et al., 2006). An inherent assumption in CIT is that a residual level of motor function remains in the affected extremity, which enables a degree of positive reinforcement for its use (Taub & Uswatte, 2006). A minimal level of residual motor function is often specified as an inclusion criterion in research studies of CIT, as is a lack of general cognitive impairment (e.g., Nichols-Larsen et al., 2005).

Due to the higher degree of neural plasticity early in development, it has been suggested that children may benefit from CIT to an even greater extent than adults (e.g., Taub & Crago, 1995 as cited by Charles & Gordon, 2005). Specific modifications have been made to make CIT more amenable to childhood populations, including conducting CIT in the home environment and practice sessions in the context of play (DeLuca, Echols, Law, & Ramey, 2005).

Treatment Procedures

CIT is generally administered as a package treatment, incorporating restraint of the unaffected limb, and structured practice (e.g., shaping and repetitive practice) with the affected limb. A "transfer package" involving behavioral techniques used to promote transfer of gains from the laboratory to daily life is emphasized in CIT

(Taub & Uswatte, 2005). Structured practice with the affected arm involves everyday functional tasks (Alberts et al., 2004), such as eating lunch, throwing a ball, etc. (Glover, Mateer, Yoell, & Speed, 2002).

Conventional CIT treatment aims for restraint of the unaffected limb for 90% of waking hours, and structured practice 6–8 h per day (Kaplon, Prettyma, Kushi, & Winstein, 2007). The treatment period for restraint and structured practice is 5 days per week, for 2–3 consecutive weeks with a 1:1 therapist–patient ratio.

The intensive nature of CIT has prompted concerns regarding its applicability to clinical settings, due to institutional and clinical factors. For example, facilities may lack the space, therapist time, and associated financial resources to administer CIT (Levine & Page, 2004). Concerns have also been raised regarding attrition rates, compliance, and health care coverage (Levine & Page, 2004; Page & Levine, 2003).

Because these factors can impede service delivery and the aims of CIT, modifications of treatment protocols have been introduced. For example, group delivered CIT (Brogardh, 2006), automatized delivery of CIT with less therapist involvement (AutoCITE; Lum, Uswatte, Taub, Hardin, & Mark, 2006; Taub, Lum, Hardin, Mark, & Uswatte, 2005), and fewer hours of restraint and shorter practice sessions over longer periods (Levine & Page, 2004; Page & Levine, 2003; Page, Sisto, Johnston, & Levine, 2002) have all resulted in significant treatment gains, often comparable to the gains of conventional CIT (e.g., Levine & Page, 2004; Lum et al., 2006). Current research suggests that particularly important features are prolonged rehearsal on specific tasks involving the affected limb and shaping in effort to improve everyday activity (Pomeroy & Tallis, 2002).

Efficacy Information

CIT has been shown to significantly improve performance on laboratory tests of motor ability and functional outcome measures. The effect sizes for functional outcome measures tend to be very large in magnitude (Taub & Uswatte, 2005). Effect sizes on laboratory tests tend to be more variable (as reviewed by Lillie & Mateer, 2006).

Outcome Measurement

There can be significant discrepancies between "real world" spontaneous functional use and motor ability as measured in a laboratory setting, and it is therefore recommended that these dimensions be assessed separately (Uswatte & Taub, 2005).

The Wolf Motor Function Test is a frequently used measure of laboratory motor performance (e.g., Uswatte & Taub, 2005). Functional outcome is most often studied through use of Motor Activity Log (MAL), a semistructured interview of extremity use in real-life settings (e.g., Winstein et al., 2003).

Qualifications of Treatment Providers

Physiotherapists most frequently provide services. Occupational therapists have also been identified as providing services (e.g., Brogardh, 2006). Specialized training in CIT is recommended (DeLuca et al., 2005; Winstein et al., 2003).

Cross References

- ▶ Brain Plasticity
- ▶ Cognitive Rehabilitation
- ▶ Head Injury
- ▶ Hemiparesis
- ▶ Hemiplegia
- ▶ Physical Therapy
- ▶ Rehabilitation Psychology
- ▶ Traumatic Brain Injury

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Constructional Apraxia

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Synonyms

Constructional dyspraxia

Short Description or Definition

Constructional apraxia is an inability to reproduce patterns or join component parts into a whole. This condition is assessed through observation of a patient completing activities such as drawing, copying, or building three-dimensional objects (Lezak, Howieson, & Loring, 2004). Impairments in processing spatial forms observed in constructional apraxia can occur in the absence of apraxia of singular motor movements (Benton, 1969).

Categorization

Recent studies suggest qualitatively different types of constructional apraxia determined by the location of brain insult. In general, patients with right hemisphere impairment make more coordinate type errors (e.g., distance and angular distortions), whereas those with left hemispheric impairment tend to commit categorical errors (e.g., position exchanges and pattern reversals) (Laeng, 2006). Patients with right hemispheric impairment generally experience difficulties with the overall gestalt of the constructional task. Their approach to the task may appear to be more fragmented and disjointed, not coming

together as a whole. Constructions created by patients with right hemisphere impairment may make drawing that are sparse or may be significantly distorted with regard to perspective or proportion. Some patients with right hemisphere injury may create overdetailed or repetitive drawings, while others may draw elaborate pictures that are missing important components. Additionally, patients with right hemispheric lesions frequently demonstrate left-sided visual inattention and include more details on construction tasks.

In contrast, those with left hemisphere impairment may be able to construct the overall concept and proportions accurately, and their constructions may also be symmetrical. However, they often tend to generate drawings with fewer details (McFie & Zangwill, 1960). Patients with left hemisphere impairment may perform construction tasks better when given a model rather than working on command. They tend to focus on overall shape rather than specific details. Overall, construction deficits tend to be more common when the lesion is located in the posterior portion of the brain rather than the anterior areas. Furthermore, while patients with cortical lesions make the same types of constructional errors as patients with subcortical lesions, those with subcortical impairment appear to have more significant impairment.

Epidemiology

A review of literature revealed no known reports of prevalence of constructional apraxia. In fact, constructional apraxia is generally included as a subcategory of apraxia, which is listed as a "rare disease" by the office of rare diseases (ORD) of the National Institutes of Health (NIH). Therefore, apraxia is known to affect less than 200,000 people in the population of the USA. Thus, as a subcategory of apraxia, constructional apraxia affects even fewer individuals. Common causes of constructional apraxia include dementia (e.g., Alzheimer's Disease) and stroke, which are two of the most frequent neurological diseases.

Natural History, Prognostic Factors, and Outcomes

Constructional apraxia is not in itself a disease but a symptom of another neurological illness. Therefore, prognosis of the condition is related to the prognosis of the underlying neurological condition. Prognosis for individuals with constructional apraxia varies and has not been well studied. Some patients may improve via continued treatment, while others may have symptoms spontaneously diminish. For other patients, symptoms may be permanent. Further research for prognostic factors of constructional apraxia is warranted.

Neuropsychology and Psychology of Constructional Apraxia

Symptoms associated with constructional apraxia frequently indicate deficits associated with right parietal dysfunction, or the non-dominant hemisphere and global cognitive processing impairment. However, recent research has shown involvement of both hemispheres, namely the parietal lobes, in constructional apraxia. Furthermore, neuroimaging research has implicated the ventral premotor area, posterior part of the inferior temporal sulcus, and occipital cortex in constructional deficits (Makuuchi, Kaminaga, & Sugishita, 2003; Nielson, Cummings, & Cotman, 1996). Constructional apraxia should not be conceptualized as a unitary syndrome caused by discrete lesions (Guérin, Ska, & Belleville, 1999), but as resulting from impairment in lateralized perceptual processing of spatial abilities (Laeng, 2006). Constructional apraxia manifests more frequently in patients with lesions in the non-dominant hemisphere (De Renzi, 1997). Consequently, constructional apraxia often co-occurs with deficits in visual-spatial perception (Benton, 1982), although both conditions may exist independently. Therefore, the presentation of constructional apraxia differs among patients; some struggle with copying figures, whereas others demonstrate difficulty constructing designs with blocks (Lezak et al., 2004).

Evaluation

The utility of constructional apraxia as an estimate of global cognitive functioning has a well-established history in neurology, neuropsychology, and rehabilitative settings. Tasks commonly utilized to assess constructional apraxia include copying, drawing, or constructing simple figures, such as clocks or crosses. These efficient, brief, and easy-to-administer construction tasks are attractive to clinicians for use with patients with impaired test-taking skills. The tasks used to assess constructional apraxia have proven to be remarkably sensitive to global neurological impairment because of the involvement of various cognitive processing domains necessary for completing construction

tasks. Construction tasks can require complex cognitive processes, including visual-spatial and visuoperceptual abilities, receptive language skills, numerical knowledge, graphomotor skills, and intact executive functioning (Freedman et al., 1994; Lezak et al., 2004; Shulman, 2000). The sensitivity of construction tasks to global cognitive impairment renders these tasks advantageous in the assessment of patients with known or suspected neurological impairment, including those with Alzheimer's Disease, Huntington's Disease, Parkinson's Disease, aphasia, seizures, CVAs, and TBIs. As an example, construction tasks have been shown to be useful as screening tools and markers for disease progression with Alzheimer's patients. Furthermore, the presence of constructional apraxia early in the course of Alzheimer's Disease has been shown to be a predictor of accelerated cognitive decline (Smith, Esiri, Barnetson, King, & Nagy, 2001).

Treatment

While physical and occupational therapies may modestly improve the functioning of an individual with constructional apraxia, no specific medical treatment has been found to be effective with significantly improving constructional deficits. Furthermore, medications that target the slowing of dementia do not appear to help with constructional apraxia. The most beneficial approach for treatment of individuals with constructional apraxia involves ensuring their environments are safe. For example, it is important to arrange furniture in the home in a way that the patient can navigate safely.

Cross References

- ▶ Apraxia
- ▶ Dementia
- ▶ Stroke
- ▶ Visual-Motor Function

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Constructional Dyspraxia

▶ Constructional Apraxia

Content Validity

▶ Test Validity

Content-Referenced Testing

▶ Domain Referenced Test Interpretation

Contextual Memory

► Source Memory

Contingency Table

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Synonyms

2 × 2 Table

Definition

A contingency table is generally a representation of categorical data in a tabular format, such as a 2 × 2 table, though the table can have three or more variables.

Current Knowledge

There are row variables on the horizontal axis and column variables on the vertical axis. It represents mutually exclusive variables. Good examples of contingency tables are hit rate, sensitivity, and specificity, as well as positive and negative predictive power. In the case of hit rates to assess the number of correct classification decisions that result from the use of a particular test or measure, one would enter the number of true positives, true negatives, false positives, and false negatives into a contingency table like the one below.

Contingency Table. Table 1

	Cond. +	*Cond. –*	
Test +	**True + (TP)**	**False + (FP)**	*Total + Decisions*
Test –	**False – (FN)**	**True – (TN)**	*Total – Decisions*
	Base Rate	*Cond. Absent*	*Total of all decisions*

Cond. = Condition

Contingency Table. Table 2

	TBI	*No TBI*	
Test +	**35**	**15**	**50**
Test –	**5**	**45**	**50**
	40	**60**	**100**

Cond. = Condition

In this example, hit rate would be calculated by adding the number of true negatives and false negatives and then dividing them by the number of total decisions ((TP + FN)/total decisions). This allows for a relatively simple way to assess the relationships between categorical variables.

A concrete example would be a group of 100 patients, 40 of whom have a true diagnosis of traumatic brain injury (TBI) who are administered a brief test to diagnose the presence of the disorder. By comparing the obtained scores to a cutting score, 55 patients are tagged as having the disorder and 45 patients as not having the disorder. Using this information, a contingency table to find hit rate can quickly be defined, sensitivity, and specificity, as well as positive and negative predictive power.

One can then calculate hit rate ((TP + FN)/total decisions) or (35 + 5)/100 = .4, sensitivity (TP/(TP + FN)) or 35/(35 + 5) = .875, specificity (TN/(TN + FP)) or 45/(45 + 15) = .75, positive predictive power (TP/(TP + FP)) or 35/(35 + 15) = .70, and negative predictive power (TN/(TN + FN)) or 45/(45 + 5) = .90. In verbal form, hit rate is a proportion of the number of correctly identified TBI patients and incorrectly ruled out TBI patients divided by the total number of patients (40% in this case) or sensitivity is computed by the percentage of people who actually have the disorder who were appropriately detected by the test (87.5% in this case).

More complex contingency tables can be used, such as 3 × 2, 3 × 3, or more, though relationships between variables are less clear owing to difficulties with interaction effects. More complex contingency tables are often analyzed with more complex statistical procedures, such as log-linear analysis.

Cross References

► Negative Predictive Power
► Positive Predictive Power
► Sensitivity
► Specificity

Continuous Performance Tests

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Synonyms

CPT

Description

An attention paradigm that has evolved into a class of neuropsychological tests used to assess sustained attention. There is not a single continuous performance test (CPT) test, as a number of commercially available and research CPT tasks exist and have been published in the neuropsychological literature. The common characteristic of all CPT tests is that they involve sequential presentation of stimuli, usually letters or numbers, over an extended period of time. The task demand is to attend and respond to particular target stimuli, while ignoring other stimuli that serve as nontarget distractors.

Historical Background

Early efforts by psychologists to assess attention in the context of intellectual or other cognitive testing typically relied on tests such as digit span, which provided a useful measure of attentional focus and span, but did not address other important elements of attention, such as the patients' ability to selectively attend to information or to sustain attention (Cohen, 1993). Sustained attention was particularly difficult to assess using traditional paper-and-pencil tests, as it required the measurement of signal detection performance over extended periods of time. Psychologists typically relied on behavioral observation or analysis of patterns of inconsistency in test performance over time to derive evidence of sustained attention problems. The development of the tachistoscope for rapid presentation of visual stimuli with controlled timing provided a means of circumventing this problem. Mirsky et al. (1956) described the continuous performance paradigm and provided research data supporting its sensitivity in detecting brain damage. The advent and widespread availability of computer technology in the decades that followed led to more widespread experimentation with this paradigm and it eventually being more commonly used in standard neuropsychological evaluations. In fact, the CPT was one of the first neuropsychological paradigms widely adapted for computerized assessment. Subsequently, impairments on this paradigm were demonstrated across various disorders, most notably schizophrenia (Nuechterlein, 1983) and attention deficit disorder (Epstein et al., 2003).

Psychometric Data

In its original form, the CPT provided measures of accuracy and response bias over the course of a fixed time period using signal detection methodology. Although the tests may vary in terms of length and type of stimulus used, the underlying paradigm is the same across versions of the test. Patients are presented with series of letters (typically) or other stimuli on a screen, and are told to push a response key only when they see the "target" stimulus. They are instructed not to respond when they see any other stimuli. The letter "x" has often been used as the target in CPT paradigms, with the task to respond to this letter while ignoring other letters that flash before them. Several common variations of the standard CPT paradigm exist, in particular, a conditional task, in which the patient must only respond to the target letter when another stimulus occurs immediately before it (e.g., A-X), which increases the difficulty of the task.

In recent years, a variety of tests based on computerized CPT paradigms have been developed, with varying degree of sensitivity and specificity to clinical disorders of attention. At the present time, the most widely used commercially available CPT tests are versions by Conners, Epstein, Angold, and Klaric (2003) and the Test of Variables of Attention (TOVA), though a variety of others exist, such as VIGIL (1990) and the d2 Test of Attention (Brickenkamp, 1992). Typically, a CPT is included as part of a more comprehensive battery of tests of attention and executive functioning.

Given that the CPT paradigm is based on signal detection theory and method, there are certain indices common to all versions of the test; correct responses, errors of omission (misses), errors of commission (false positives), and response time (RT). Correct responses are based on the sum of the number of times the patient correctly responds to the target and correctly avoids responding to distractors. Omission errors (misses) are errors involving a failure to respond to the target, while commission errors (false positives) are errors involving a response to nontargets. Errors of commission reflect a failure to inhibit responding. High omission rates indicate that either the patient is not

focusing adequately on the stimuli or that their processing speed is slow and they are unable to respond rapidly enough. CPT tests usually also provide a measure of mean response time (RT), which reflects the processing speed of the patient during the task. Higher rates of correct detections indicate better attention performance.

From these primary measures, signal detection indices are usually derived based on a comparison of error types. Based on the total number of errors of commission and omission on the CPT, a discrimination index (d′) is calculated which provides a measure of accuracy based on standardized scores (z-scores) from normative samples. A measure of response bias (β) is also usually derived based on the difference in standard scores for each type of error. Response bias indices provide a better way of interpreting tendencies to make one type of error or the other because they account for the total number of errors of each type. Receiver operator characteristics (ROC) metrics can also be derived, which provide a way of interpreting the tendency to make errors of each type as a function of the signal detection parameters of the task, such as percentage of targets to nontargets. General equations for d′ and β are shown below.

$$d' = z(\text{misses}) + z(\text{false positives})$$

$$\beta = z(\text{misses}) - z(\text{false positives})/z\ (\text{total errors})$$

In the past, many versions of the CPT provided only these basic indices, which limited their usefulness in characterizing problems with sustained attention. While d′ and β provide excellent measures of detection accuracy, response deposition, and overall attention performance, they do not directly provide measures of performance over time. Recent versions of the Conner's and other CPT tests tend to now include a measure of temporal variability in performance. While there are a variety of ways that the temporal nature of performance can be determined, two general types of measures exist: (1) performance decrement and (2) performance inconsistency. Performance decrement provides a measure of the change in performance between the beginning and end of the test. In theory, if a person is failing to sustain attention, their performance should worsen the longer they stay on task. However, often this measure is not impaired except when there is a severe problem with sustained attention and fatigue after several minutes of effort. Performance inconsistency indices provide an alternative temporal measure based on variance across time, as opposed to linear decrement.

A primary problem in interpreting the meaning and clinical significance of CPT findings stems from the lack of consistency across versions of the tests, particularly with respect to basic parameters such as the duration of the interstimulus interval (ISI), the total duration of the task, and the task demand. For example, a task requiring detection of a single letter is much easier than a conditional paradigm requiring detection of a letter sequence (A-X). Furthermore, the duration of a typical CPT test can range from several minutes to over 20 min. Versions with a long ISI tend to be relatively easy to perform, but are tedious, with behavioral challenge arising from the monotony of the task, which over a long test period is really a test of vigilance. In contrast, versions that have a short ISI with conditional task demands or other characteristics that increase difficulty tend to be more sensitive to sustained attention in the context of greater information processing demand and requirement for focused attention. Adaptive-rate continuous performance methods provide a means of circumventing this issue. For example, the adaptive-rate continuous performance test (ARCPT; Cohen, 1993) adjusts its ISI over the course of the test based on accuracy of response to compensate for the slow speed of processing. The final ISI that is maintained by the end of the test provides a strong measure of capacity limitations related to processing speed deficits. Also the ARCPT provides separate vigilance decrement and inconsistency indices to enable assessment of the temporal dynamics of performance over 10 blocks of time over the duration of the test. The ARCPT measures attentional performance on a rapid and challenging task which is less subject to boredom. Therefore, the ARCPT provides enables the assessment of sustained attention during a cognitively demanding task compared to standard CPT paradigms. Also, because the ISI adjusts to shorter durations when a patient is performing well, the test can typically be completed in about 10 min. The ARCPT also provides several metrics that enable assessment of temporal inconsistencies in performance.

Despite the fact that CPT tests vary in a variety of ways, many of the commonly used measures (e.g., Conner's CPT) report good test–retest reliability in healthy adults. CPT performance tends to correlate well with performance on other information processing-based measures and performance on speeded tests of attention and executive function, such as the Stroop and trail-making test. Validation studies conducted with the ARCPT indicate strong reliability (r = .95) for accuracy of performance (d′) across samples and strong validity as indicated by the sensitivity of CPT indices to measures of structural and functional brain and systemic physiological disturbances as discussed in greater detail below.

Clinical Uses

CPT tests should be included in all comprehensive neuropsychological batteries designed to provide a thorough assessment attention, executive functioning, and processing speed. There is a well-established research and clinical literature demonstrating that this paradigm is highly sensitive to brain dysfunction. The fact that the CPT yields measures of information processing characteristics based on a task with controlled timing and consistency of stimulus presentation makes it a valuable addition to standard paper-and-pencil tests of cognitive function.

Performance deficits on the CPT are evident in patients with various forms of brain dysfunction (Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). The most obvious use for the CPT is in the assessment of attention deficit disorder (ADD), for which the objective assessment of sustained attention is central to the diagnosis. However, there is now an extensive research literature demonstrating deficits or outright impairments of CPT performance among patients with a wide range of neurological and psychiatric conditions. For this reason, impaired CPT performance should be considered a highly sensitive measure of functional attention impairment and brain dysfunction, though not necessarily specific to one type of neurological or psychiatric condition.

Impairments of CPT are evident in patients with neurodegenerative disorders like Alzheimer's disease, as well as disorders affecting subcortical and white matter brain systems, such as multiple sclerosis, HIV, and cerebrovascular disease. In fact, patients with cardiovascular disease show a relationship between cardiac output and CPT performance (Jerskey et al., 2009). Among patients with severe affective disorders, CPT performance is often impaired and associated with problems with effort (Cohen, Lohr, Paul, & Boland, 2001). CPT has been shown to be sensitive to sustained attention and information processing deficits associated with neurotoxic exposure to lead, solvents, drugs, or other substances. An important clinical determination that needs to be made when examining performance on the CPT is whether there is evidence of an actual problem with sustained performance, or whether a broader problem with focused and selective attention exists that occurs regardless of the time spend on task.

Cross References

► Attention Deficit Disorder (ADD)
► Signal Detection
► Sustained Attention
► Vigilance

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Contraindication

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Synonyms

Conflict; Counterindicant

Definition

A contraindication is a circumstance, condition, symptom, or factor that increases the risk associated with a medical procedure, drug, or treatment. A contraindication refers to

any intervention considered inappropriate or inadvisable based upon unique factors of the situation such as potential harmful interactions between drugs or medical conditions that renders an individual vulnerable if implemented.

A contraindication may be absolute or relative. Absolute contraindications are those which are inadvisable without exception or qualification. They are either permanently recommended against, or temporarily until the disqualifying condition is remediated. The use of the atypical antipsychotic medication, clozapine carries a risk of agranulocytosis, a sever low white blood cell disorder condition. Clozopine would be an absolute contraindication in an individual with a history of bone marrow suppression

Relative contraindications refer to circumstances in which procedures or treatments are considered in comparative terms. They are contingent upon a risk/benefit analysis of the relevant factors. The proportionate value of an intervention is compared with its potential for negative consequences. An example of a relative contraindication would be the use of an anti-convulsant/mood stabilizing medication in the ongoing treatment of a pregnant woman with severe bipolar mania and suicidality, but whose has been asymptomatic only while on lithium.

Cross References

- ▶ Extrapyramidal Side Effects
- ▶ Iatrogenic
- ▶ Signs

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Contrecoup Injury

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Definition

Contrecoup injury is an injury to the brain tissue directly beneath the skull, opposite to the point of impact. It results from acceleration–deceleration events during which the force impacting the head causes the brain to slam into the skull on the opposite side of the blow. Motor vehicle accidents, falls, sports injuries, and physical assaults with blunt objects frequently result in contrecoup injuries. Skull characteristics make the most probable sites of injury in the frontal and temporal lobes, as tips of the skull can more easily be forced into the underlying brain tissue in the frontal and temporal lobes. Neuropsychological evaluation can help to identify cognitive impairments arising from both the primary and secondary sites of injury.

Cross References

- ▶ Acceleration Injury
- ▶ Biomechanics of Injury
- ▶ Cortical Contusion
- ▶ Traumatic Brain Injury

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Controlled Attention

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Synonyms

Central executive; Cognitive control; Supervisory attentional system

Definition

Higher-order cognitive processes that influence the regulation of behavioral responses and the selection of information to be attended to are based on task-related goals.

Current Knowledge

Neuroanatomical

From a neuroanatomical perspective, brain networks other than those related to frontal lobe functions can be involved in controlled aspects of attention. For example, parietal lobe structures appear to be important for response intention and sensory selective attention. However, control behaviors such as response planning, selection, and execution are associated with networks of cortical and subcortical regions linked to the frontal lobes. It is these functions that are most commonly refer to when discussing control functions of the brain. Although there is not a clear anatomical mapping, studies suggest that different regions of the frontal lobe (and associated networks) relate to different aspects of control, such that there is a heterogeneity of functioning that would argue against any unitary conceptualization of attentional control (Cohen, 1993).

Cognitive

Similarly, cognitive researchers have made an effort to move away from a homunculus view of control functions, pointing out that terms such as *central executive* (Baddely & Hitch, 1974), *supervisory attentional system* (Shallice, 1988), and *executive control* (e.g., Anderson & Green, 2001) all take on the role of a poorly defined black box and give the impression of a unitary concept. These terms may have been useful as placeholders, while cognitive researchers worked out the details of other related systems, such as the slave systems of Baddely and Hitch's (1974) model of working memory. However, with an emerging focus on attentional control and with new tools for the direct study of phenomena such as task switching, response inhibition, goal formation, and planning in the form of functional neuroimaging (e.g., Shallice, Picton, Alexander, & Gillingham, 2008), computational modeling (O'Reilly, Braver, & Cohen, 1999), and statistical modeling techniques (Miyake et al., 2000), there is a growing body of information that specifies the subprocesses involved in these selection functions with increasing clarity (see also Monsell & Driver, 2000 for a review).

Cross References

- ▶ Divided Attention
- ▶ Focused Attention
- ▶ Frontal Lobe
- ▶ Vigilance

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Controlled Oral Word Association Test

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Synonyms

Category fluency; CFL test; COWA; COWAT; F-A-S test; Letter fluency; Phonemic fluency; Verbal fluency

Description

The *Controlled Oral Word Association Test* (COWAT) is a measure of verbal fluency and is a subtest of the *Multilingual Aphasia Examination* (MAE; Benton, Hamsher, & Sivan, 1994). The COWAT uses the three letter set of C, F, and L to assess phonemic fluency. Individuals are given 1 min to name as many words as possible beginning with one of the letters. The procedure is then repeated for the remaining two letters (see Strauss, Sherman, & Spreen, 2006 and Benton, Hamsher, Rey, & Sivan, 1994 for

specific administration instructions). Several tests of phonemic fluency exist, some of which are part of larger test batteries (e.g., the MAE or the *Neurosensory Center Comprehensive Examination for Aphasia;* Spreen & Benton, 1977) and others that can be administered independently (e.g., the F-A-S Test).

Verbal fluency is a cognitive function that facilitates information retrieval from memory. Successful retrieval requires executive control over cognitive process such as selective attention, mental set shifting, internal response generation, and self-monitoring. Tests of verbal fluency evaluate an individual's ability to retrieve specific information within restricted search parameters (Lezak, Howieson, Loring, Hannay, & Fischer, 2004). The two most common parameters are semantic fluency and phonemic fluency.

Historical Background

Borkowski, Benton, and Spreen (1967) were early proponents of systematically examining word fluency in persons with brain damage. They recognized that so-called word fluency tasks had been used in neuropsychological investigation of patients with brain damage and undertook two studies of the task. The first established the relations between word fluency and various English letters; the second examined word fluency data for persons with brain damage and control patients. They presented normative data for the first study and comparative data for the second, clearly supporting their hypotheses of the utility of word fluency assessment.

Since publication of these data, word fluency tasks, and the COWAT in particular, have been investigated in detail. Spreen and Risser (2003) suggest that this assessment tool in its various forms has been one of the most frequently and thoroughly investigated neuropsychological assessment measures for unimpaired and neurologically impaired persons. A search of electronic databases confirms this suggestion.

Psychometric Data

Psychometric data for the COWAT and other phonemic fluency tests, as well as other verbal fluency tasks (e.g., semantic fluency) are readily available. Norms have been published for children and adults of varying ages, levels of education, ethnic diversity, and geographical diversity (Loonstra, Tarlow, & Sellers, 2001; Strauss et al., 2006). Some differences have been noted between test forms, most notably, between the COWAT and F-A-S Test (Barry, Bates, & Labouvie, 2008); the CFL form appeared more difficult, while results for the F-A-S form appeared more variable. In addition, COWAT scores have been correlated with neuropsychological measures such as reading tests and IQ tests.

Clinical Uses

Scoring for the COWAT and other verbal fluency tests is straightforward. The examiner writes each word as it is produced by the individual. The transcript is reviewed and inadmissible words (i.e., repetitions, proper names, or slang) are eliminated. The test score is the total number of different words produced for all three letters (see Strauss et al., 2006 and Benton et al., 1994 for specific administration instructions).

Supplementary scoring measures for the COWAT and other phonemic fluency tests provide additional information in clinical diagnosis and treatment. Supplementary scorning measures are error analysis, and cluster and switching analysis (see Table 1). In error analysis, the examiner notes any observable pattern of production of errors that suggests a loosening of executive control over cognitive processes that would result in errors. For example, a pattern of multiple repetitions of previous responses suggests perseveration and inefficient self-monitoring. Error patterns provide qualitative performance data and may appear as common patterns such as repetition of a word, or idiosyncratic patterns. Clustering and switching analyses evaluate the depth of the searchable knowledge base (clusters) and the cognitive flexibility within and across categories (switching) (Troyer, Moscovitcvh, & Winocur, 1977). An example of an efficient search strategy would be identifying a cluster or subcategory within the category (e.g., words that begin with "cr" in the COWAT task of naming words that begin with C) and producing as many items in that category as possible and then switching clusters (e.g., to words beginning with "cl"). Clusters are scored by counting the number of clusters and calculating the mean cluster size; switches are scored by counting the number of transitions between clusters. Rules for scoring cluster size and number of switches appear in Troyer et al. (1997) and normative data for healthy adults appear in Troyer (2000).

Scores from the COWAT are useful in evaluation of persons with neurogenic communication disorders, such as aphasia following stroke, traumatic brain injury, and dementia. Studies have included COWAT in the diagnostic batteries given to several patient populations and also in treatment studies as measures of behavioral change. The utility of the COWAT in identifying the nature

Controlled Oral Word Association Test. Table 1 An example of F-A-S Test results and cluster and switch scoring for a person with aphasia

	Responses	Clusters and Switches
FAS-Test: "F"	fast, fun, fickle, fuschsia, finger of fate, four, fifty-four, forty, fornicate Words = 9	Clusters = 8 (mean size = 1.1) fast fun fickle fuchsia finger of fate four fifty-four forty, fornicate *Switches = 7*
FAS-Test: "A"	apple, aardvark, alpaca, ammonia, arsenic Words = 5	Clusters = 5 (mean size = 1) apple aardvark alpaca ammonia arsenic *Switches = 4*
FAS-Test: "S"	substantial, sum, subtraction, stuck, structure, symbol, sympathy, stroke, sixty Words = 9	Clusters = 5 (mean size = 1.8) substantial, sum, subtraction stuck, structure symbol, sympathy stroke sixty *Switches = 4*
Total	Words = 23	Clusters = 18 Mean size = 1.27 *Switches = 15*

and severity of performance deficits in clinical populations has been supported; however, conflicting findings have been reported. Typically, the total number of acceptable responses is the reported test result; however, increasingly cluster and switching scores are reported as well. The COWAT is valuable in detecting cognitive dysfunction, but it requires further study before definitive conclusions are possible with regard to performance patterns that can be linked with specific neurogenic behavioral deficits.

Cross References

- ▶ Aphasia
- ▶ Aphasia Tests
- ▶ Benton, Arthur
- ▶ Boston Diagnostic Aphasia Examination
- ▶ Circumlocution
- ▶ Clustering
- ▶ Cognitive-Communication Disorder
- ▶ Cognitive Functioning
- ▶ Cognitive Processing
- ▶ Cue
- ▶ Cued Recall
- ▶ Error Recognition and Correction
- ▶ Free Recall
- ▶ Mayos Older American Normative Studies (MOANS)
- ▶ National Adult Reading Test
- ▶ Phonemic Cue
- ▶ Semantic Cue
- ▶ Semantic Fluency
- ▶ Verbal Mediation
- ▶ Western Aphasia Battery

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Contusion (Cerebral)

► Cortical Contusion

Conventional Antipsychotics

► Antipsychotics

Conversation Analysis

► Discourse Assessment

Conversion Disorder

► Cogniform Disorder
► Somatization

Convulsion

► Grand Mal Seizure

Convulsive Disorder

► Epilepsy

COPD

► Chronic Obstructive Pulmonary Disease

Coping

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Synonyms

Stress management

Definition

Coping is responding to environmental stimuli, events, and circumstances for the purpose of minimizing or managing stress, solving problems, and modulating physiological and emotional responses. Coping is often paired with stress (as the latter elicits the former) in what has become the stress and coping literature associated with Richard Lazarus and Susan Folkman. Stress responses typically involve appraising the stimulus or event, which begins the process of assigning value (e.g., distressing) and determining responses (e.g., fight, flight, freeze). Coping is a process that follows stress appraisals, and coping responses seek to manage stress with cognitive, physiological, and behavioral responses. Various coping strategies have been categorized, such as appraisal-focused, problem-focused, or emotion-focused coping.

Cross References

► Self-Regulation
► Stages of Adjustment

► Stress
► Stress Management

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Core Battery

► Flexible Battery

Coronary Angioplasty

► Angioplasty

Coronary Artery Bypass Graft

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Synonyms

CABG

Definition

Coronary artery bypass surgery (CABG) is a surgical procedure for coronary artery disease in which arteries or veins from other parts of the body are grafted from the aorta to the coronary arteries in order to bypass the blocked portions. Indications for surgery include disease of the left main coronary artery and/or disease of all three coronary arteries and abnormal ventricular function. It may be performed in patients with severe angina which is unresponsive to medical management.

Patients undergo general anesthesia and a midline incision (median sternotomy) allows the surgeon to visualize the heart and vessels. The artery or vein grafts are then harvested. Frequently used vessels include the internal thoracic arteries, radial arteries, and saphenous veins. The heart is then stopped using a special mixture of chemicals and the patient is placed on cardiopulmonary bypass where the blood flow returning to the heart is diverted through a heart–lung machine that provides extracorporeal circulation and oxygenation. The graft is then sewn into place and the heart is restarted. The sternum is then closed with wires, and the incision is sutured.

Current Knowledge

In the early years of cardiac surgery several investigators noted transitive postoperative delirium, whereas others reported immediate postoperative neurological abnormalities, however, no well-conducted or controlled studies were available. More recent studies have shown a short-term neuropsychological decline in the 7–14 days after CABG, including deficits in psychomotor speed, attention, verbal learning, and memory. Studies have reported an 11–75% incidence of postoperative cognitive decline (POCD) after cardiac surgery. Age is considered to be the strongest predictive factor of postoperative cognitive dysfunction. It is hypothesized that "off-pump" surgery causes less cognitive decline as it avoids extracorporeal circulation, but studies have not shown this to be true. Other potential causes of neurocognitive decline include intraoperative cerebral oxygen desaturation or microemboli.

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Coronary Artery Disease

► Coronary Disease

Coronary Disease

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Synonyms

Atherosclerotic heart disease; Coronary artery disease; Coronary heart disease; Ischemic heart disease

Definition

Coronary disease, or coronary artery disease (CAD), results from atherosclerosis, or narrowing of the arteries that provide blood supply to the heart muscle ("myocardium").

Current Knowledge

Atherosclerosis, which is the process by which cholesterol and fat deposits adhere to the inside walls of blood vessels, creates "plaques" that block the blood supply through blood vessels. When this narrowing or occlusive process blocks the coronary artery, the accompanying obstruction to blood flow reduces the supply of oxygen and nutrients to the heart muscle, creating ischemia that causes chest pain ("angina pectoris"), or death of the cells of the heart ("myocardial infarction"). At times, it can cause other problems such as arrhythmias (abnormal heart rhythm) or congestive heart failure, in which the heart is unable to pump sufficient blood to the remainder of the body. CAD is the most common type of heart disease and the leading cause of death in the USA. Risk factors for CAD have been well studied; these include smoking, diabetes, hypertension, hyperlipidemia, obesity, sedentary lifestyle, excessive alcohol intake, and family history. Although chest pain is the most common symptom, dyspnea, palpitations, diaphoresis, nausea, radiation of the pain to the neck and left arm, and no symptoms at all, also are possible. The diagnosis relies on electrocardiogram, blood levels of cardiac enzymes, echocardiography, select cardiac stress tests, and direct visualization on coronary angiography or other imaging studies. The treatment includes the use of beta-blockers, calcium channel blockers, angiotensin converting enzyme inhibitors, nitroglycerine, aspirin, or other medications; antihypertensive and lipid-lowering agents; diet, exercise, and lifestyle changes; thrombolysis; angioplasty with coronary stent placement; and coronary bypass graft surgery.

Cross References

► Angioplasty
► Anticoagulation
► Antiplatelet Therapy
► Atherosclerosis
► Cholesterol
► Congestive Heart Failure
► Infarction
► Ischemia
► Myocardial Infarction
► Stent
► Thrombosis
► Tissue Plasminogen Activator
► Vasospasm

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Coronary Heart Disease

► Coronary Disease

Corporis Callosi

► Corpus Callosum

Corpus Callosum

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Synonyms

Commissural magna; Corporis callosi; Interhemispheric commissure

Definition

Corpus callosum is the largest axonal tract of the adult brain that provides symmetrical connections between the two hemispheres.

Current Knowledge

The corpus callosum is the largest commissure of the adult brain that provides a bridge for the passing of information from one cerebral hemisphere to the other by 200–300 million myelinated and unmyelinated axons. The size of the corpus callosum varies greatly but is generally larger in females than in males. In the human, the corpus callosum begins development around the 11th week of gestation and continues through adolescence. Initially, the corpus callosum is composed of astrocytic processes, which serve as conduits for growing axons extending to the contralateral hemisphere. This interhemispheric commissure lies beneath the cortex at the bottom of the cerebral longitudinal fissure. It forms much of the roof of the lateral ventricles and is composed of four parts: the rostrum, the genu, the body (also known as the trunk), and the splenium. Each portion of the corpus callosum is responsible for connecting symmetrical regions of the two hemispheres with the rostrum and genu connecting portions of the prefrontal and premotor cotices, the body interconnecting the premotor, motor, supplementary motor, and the posterior parietal cortices, while the splenium connects portions of the temporal, occipital, and parietal lobes. Although being the largest white matter tract of the brain, the corpus callosum is not essential for life. The complete or partial absence of the corpus callosum is known as agenesis of the corpus callosum. This condition is rare, and estimates of incidence vary widely but are generally reported to range between 0.3 and 0.7% in the general population and as high as 3% in individuals with development disabilities. Agenesis of the corpus callosum results in symptoms ranging from asymptomatic with normal intelligence to intellectual retardation, seizures, hydrocephalus, and spasticity. In individuals suffering from severe epilepsy, a surgical procedure known as corpus callostomy may be performed, which involves the partial or complete transaction of the corpus callosum. This procedure is only performed in patients who are at risk of accidental injury resulting from severe seizures. The layman's term for this condition is split brain due to the loss of interhemispheric communication. Much of the original research detailing the consequence of corpus callostomy was conducted by Dr. Roger Wolcott Sperry.

Cross References

▶ Ganglion
▶ Gazzaniga, M. S. (1939–)
▶ Sperry, Roger Wolcott (1913–1994)
▶ Split-brain

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Corpus Mamillare

▶ Mammillary Bodies

Correction Factor

▶ K Scale

Correlation Coefficients

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Synonyms

Pearson *r*, *r*, rho

Definition

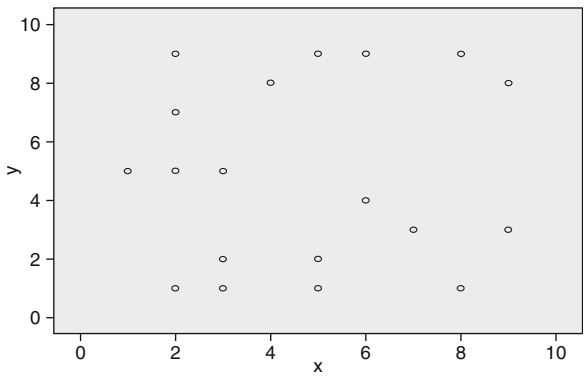
A measure of the strength of the relationship between two variables, *x* and *y*.

Historical Background

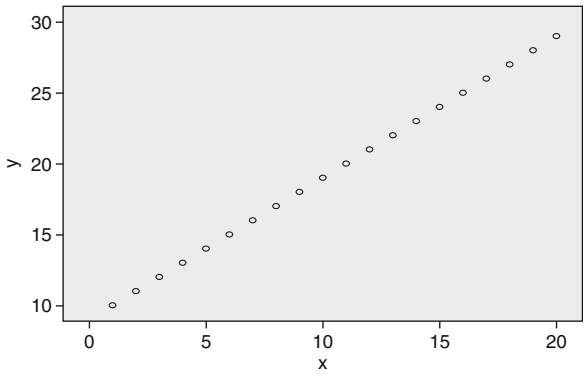
The correlation, or co-relation, coefficient is typically attributed to Karl Pearson who developed the formalized idea of correlation during the mid- to late 1800s. However, the beginning of the idea of correlation may have come from Sir Francis Galton, cousin of Charles Darwin. Galton worked on genetic heritability of sweet peas. Through his work on heritability, he developed the beginnings of regression and correlation. Pearson, who worked in Galton's lab and was later his biographer, attributed the development of the regression slope to Galton. Pearson then generalized Galton's work into the Pearson Product Moment Correlation (PPMC), or "moment" meaning the average of a set of products. The PPMC is often identified as "*r*," which Galton originally used to denote "regression" and Pearson later used as notation for correlation. In 1896, Pearson published an article in which he credited Bravais for developing the rudiments of the correlation formula. In this work published in the *Philosophical Transactions of the Royal Society of London*, Pearson showed that the optimum values for the regression line and correlation could be derived from the product moment or $\Sigma\ xy/n$, where *x* and *y* are deviations from their respective means, usually expressed as standard *z* scores and *n* is the number of pairs.

Current Knowledge

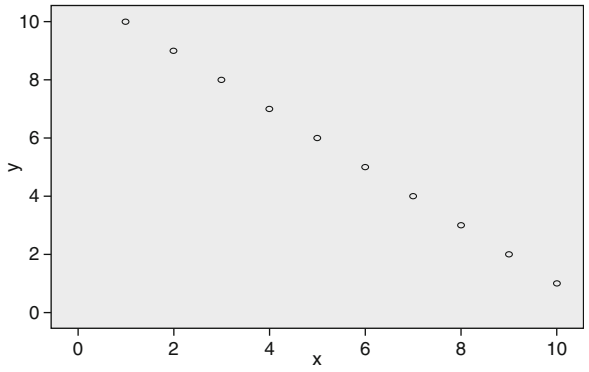
A correlation, or co-relation, of two variables can be visually scanned initially by simply looking at a scatter plot of the data. The values of a correlation can range between +1 and −1 with these two extremes representing perfect relationships, which rarely if ever occur in research, and a correlation coefficient of 0 would represent no relationship, given certain assumptions are made. A line can be drawn through data that are related that generally approximates a regression line, which is how Galton identified the first regression line in his heritability studies with peas. Correlation is a measure of the strength of the relationship between two variables. Thus, if data in a scatter plot are randomly scattered, such as in the example below, then there is no relationship, as no single line adequately represents the data and the correlation would be roughly 0.



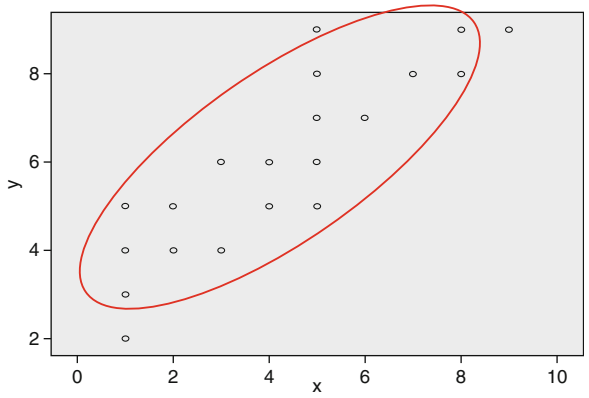
If data are positively related, such that as one variable increases in value so does the other, then one would be able to draw a line that would approximately represent the data. The below example illustrates an ideal relationship that rarely occurs in research of $a + 1$ correlation such that for every one unit that *x* goes up, *y* goes up one unit.



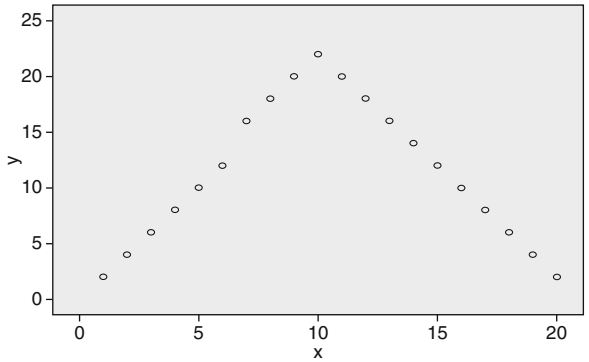
There may also be negative associations, such that for every unit that *x* goes up, *y* goes down, or $a - 1$ relationship, such as the below idealized example.



However, typically a scatter plot will not be as obvious and the data will look more like an ellipse such as in the example below, which still illustrates a strong correlation of over .8.



The above examples show linear relationships between variables, which is an assumption of most correlation coefficients. An examination of scatter plots can also show nonlinear relationships, which are not well represented by standard correlation coefficients, such as the PPMC, but can be shown with other methods, such as eta. The below example has a near 0 Pearson *r*, but the variables are clearly related nonlinearly.



The correlation coefficient is therefore a measure of the extent to the relation of pairs of observed data points can be expressed as a linear function. The greater the summed square of differences between the observed data and the values described by the linear function, the lower the correlation coefficient. The closer the observed data points lie to the line of best fit, the higher the correlation coefficient.

There are several forms of correlation that need to be addressed including those for continuous variables, categorical variables, and correlations that are better descriptions of nonlinear and skewed relationships. The scope of this article is to cover the basic foundations of correlation coefficients. The use of correlation ranges far and wide and a thorough examination of this subject is the subject of chapters and entire texts referenced below. The object of this entry is to cover foundational issues related to correlation coefficients. There are more correlation coefficients than those presented below, though several different forms of correlation and their properties are considered.

It is important to note that correlation, even a strong correlation, does not infer causation. In other words, simply because two variables are related does not mean that one causes the other. For example, you need light to read this text. Simply because the light is on while you are reading does not mean that light is causing you to read, they are certainly related, as there will always be some light source when you read, but one does not cause the other. A determination of causation requires adequate experimental design.

There are certain important components that underlie most correlations. Correlations are most meaningful when certain criteria are met, and different correlation coefficients are affected differently by failure to meet different assumptions. The Pearson product moment correlation coefficient has the most stringent set of assumptions. These include:

1. The data are interval: The level of measurement must at least be interval as opposed to nominal or ordinal, though this can be managed if it is violated.
2. The data must have a linear relationship: Most correlations assume that the data are linearly related, as opposed to nonlinear. To the degree which the data are better explained by a nonlinear relationship correlation coefficients will underestimate the relationship. Prior to completing a correlation analysis this can be examined by looking at a scatter plot, as discussed above.
3. The variables have similar and normal underlying distributions (more formally in a bivariate correlation, such as the Pearson *r*): To the degree that the

distributions of the different variables differ, the correlation will attenuate the relationship between them. For example, if correlating a variable that is normally distributed, like height, with one that is highly skewed like ability to walk. This is done when correlating a continuous variable with a dichotomous variable or when correlating interval data with ordinal. If this assumption is violated to a significant degree then rank order correlation may be used, such as the Spearman's rho or other nonparametric correlations.

4. The data must have homoscedasticity: The error, or random variance in *X* and *Y*, contained in the data, is assumed to be equal along the entire distribution. If, for example, one portion of the distribution has a much larger random variance, then the correlation may overrepresent the relationship because it would underrepresent the amount of variance that is attributable to error. If data are heteroscedastic, then the distribution will not be normal and the correlation may misrepresent the data.
5. The data has no or limited outliers: Correlation is sensitive to outliers. Some forms of correlation are more robust to a violation of this assumption than others, but normally correlation is a representation of deviations of mean values. Thus, to the degree that the mean is affected by outliers then the correlation will be also. If there are many outliers, or few that are large, this problem can be managed through use of a Spearman rho, which ranks data.
6. There is limited measurement error present in the sample: It is assumed that the measurement is relatively free from or has limited error. In other words, if one has an invalid test that only measures error then the correlation will be near zero, as the data will be random and show little relationship. It is rare in behavioral research to be free from error, but limiting it is an important aspect of experimental control.
7. The data have adequate variance: In order to adequately assess relationships among variables there must be variance among the variables. In general, the more variance the higher the correlation will be.

To the extent that these criteria are violated, which they often are, the correlation coefficient is more or less impacted depending on the type of correlation.

There are several different methods calculating basic correlation coefficients depending on the type and characteristics of the data being analyzed. There are several different types of correlation that depend on the level of measurement (nominal, ordinal, interval, ratio) being made. Most are special cases of the Pearson *r*. There are also other methods that are more robust to violations of the above assumptions, such as Spearman's rho or eta in the case of a nonlinear relationship. The correlation coefficient is considered a measure of *effect size*, which is a measure of how different two distributions are. By convention a correlation of .1 is small, .3 moderate and .5 large (Cohen, 1988), at least in the behavioral sciences that can expect weaker relationships in general due to the complexity of the subject matter. For example, the correlation between IQ and academic achievement is approximately .55 (Griffenstein & Baker, 2003), but one would expect no effect for a correlation between IQ and height. The correlation coefficient can also be squared to give the coefficient of determination, which is the amount of variance in one variable that is accounted for by another. For example, if one had a correlation of .55 between IQ and academic achievement then the coefficient of determination (r^2) would be .30, which means that approximately 30% of the variance in academic achievement is accounted for by IQ.

1. PPMC or *r*: The PPMS is an association of two continuous variables that shows the degree of *linear* relationship between them. It can be calculated using raw scores, deviation scores, or by using a covariance formula. A common method is to calculate the standard score (or *z* score) and sum the products of the two variables and divide them by the degrees of freedom $(n - 1)$ or $\Sigma\ xy/n\ \Sigma\ 1$.
2. Point-biserial or r_{pb}: This is a special case of the PPMC for use when there is a continuous variable, such as height, and a dichotomous nominal variable, such as gender. The point-biserial correlation is for naturally dichotomous variables, such as gender, not artificially *dichotomized* variables, such as taking a naturally continuous distribution, such as intelligence, and making it into high and low intelligence.
3. Phi: This is a special case of the PPMC for use when both variables are dichotomous and nominal. Note that as with r_{pb} this is for naturally dichotomous variables, such as gender, not artificially *dichotomized* variables.
4. Biserial or r_b: This is for use when there is one continuous variable, such as height, and a dichotomized variable, such as high and low intelligence. So, the biserial correlation measures the relationship between *X* and *Y* as if *Y* were not artificially dichotomized. This is similar to the point-biserial, but the formula is designed to replace some of the variance that is lost. One of the important components listed above is that the data have adequate variance. However, when a variable is dichotomized much of the variance is lost.

Thus, the biserial formula replaces some of that variance, which always yields a higher correlation than a point-biserial correlation.

5. Tetrachoric: This is similar to the phi, except that it is assumed that there is a continuous distribution underlying the dichotomized variables and it thusly replaces some of that variance, as opposed to phi that assumes that the two variables are naturally dichotomous, such as gender and employment status.
6. Spearman's rho or r_s or ρ: Spearman's rho is a nonparametric statistic. In other words, it makes no assumptions about the underlying distribution of the variables. One of the primary components of the typical PPMC is that there is a normal or bell-shaped distribution underlying the variable. However, in many cases, distributions are highly skewed. For example, if one were to look at a distribution of people who live into adulthood in the USA, the distribution would likely be very skewed to the left, meaning that the vast majority of people live at least until they are of adult age. Relate that distribution to the number of people who vote, which would be positively skewed, meaning that few people of adult age vote proportionally and a PPMC would not yield an inaccurate representation, as this distribution significantly violates the assumptions of the PPMC. Thus, one could convert raw scores into *ranks* and complete the correlation. This is more robust to violations of some of the underlying components of correlation.
7. Measures of nonlinear relationships: Eta is a measure of association that can be used if the data are nonlinear, as in the final scatter plot above. It is a measure of the strength of the effect and is always positive. Eta is always higher than |r| and is therefore a biased estimate of the effect.

In practical usage, many of the different correlation coefficients are calculated using the same method, such as the PPMC and the point-biserial, given the ubiquity of computer statistical packages. What is important to note with any correlation being used are the number and degree of the components that are violated and what impact that has on the relationship between the variables. For example, if one is using a PPMC to assess highly skewed data then the relationship may be very low, where actually, if a nonparametric method is used, such as the Spearman's rho, then the relationship may be significant. Conversely, if a PPMC is used on data that have many large outliers the relationship may look strong and be significant, but in reality it is the impact of those large outliers that are falsely raising the strength of the associations.

Cross References

▶ Statistical Significance
▶ *z* Score

References and Readings

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Cortex

▶ Cerebral Cortex

Cortical Activation

▶ Arousal

Cortical Arousal

▶ Arousal

Cortical Blindness

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Synonyms

Anton's syndrome; Blindsight

Short Description or Definition

Cortical blindness refers to severe visual loss produced by bilateral damage to the geniculostriate visual

pathways. The underlying pathophysiological mechanism involves direct destruction and/or de-afferentation of primary visual cortex (striate cortex, Brodmann area 17, or V1). The term Anton's syndrome is applied to patients with cortical blindness who demonstrate explicit denial or unawareness (anosognosia) of their visual impairment.

Categorization

Although no universally agreed upon classification system exists, patients with cortical blindness differ in terms of the severity of the visual loss, the presence/absence of spared visual abilities (conscious or unconscious), the degree of awareness of the deficit, and the extent of functional recovery. There are also variations across cases with respect to the capacity to generate internal visual representations using mental imagery and the susceptibility to experience abnormal visual sensations/hallucinations. The precise neurobiological mechanisms underlying these individual differences in clinical presentation remain to be determined.

Epidemiology

Cortical blindness is a relatively rare condition, typically caused by bilateral occipital strokes in the territory of the posterior cerebral arteries. Other less common etiologies include anoxic brain damage, carbon monoxide poisoning, head trauma, and occipital lobe tumors. Transient cortical blindness can be seen in the context of the reversible posterior leukoencephalopathy syndrome (RPLS) related to hypertensive encephalopathy, the use of chemotherapeutic and immunosuppressant drugs, or the injection of radiological contrast agents during cerebral angiography.

Natural History, Prognostic Factors, Outcomes

Although some recovery of visual function is observed in most cases of cortical blindness, patients with neuroimaging evidence of extensive structural damage to occipital cortex typically do not regain full normal vision. Residual visual field defects are common, and cortical blindness may evolve into visual agnosia characterized by a persistent inability to recognize objects, faces, or words despite the return of more elementary visual functions. It is currently unclear whether recovery of visual capacity in cortical blindness is mediated by spared neural tissue within primary visual cortex, or whether it reflects the strengthening and increasing utilization of alternative visual pathways to extrastriate cortical regions that bypass the damaged geniculostriate system, or both. Anosognosia for visual loss in patients with Anton's syndrome also tends to diminish over time, often parallel to the resolution of the visual impairment, but partial defects of awareness are not uncommon. In cases of transient cortical blindness due to RPLS, full recovery of vision can occur within a few days along with complete resolution of the characteristic neuroradiological abnormalities.

Neuropsychology and Psychology of Cortical Blindness

In severe cases of cortical blindness, all forms of conscious visual perception may be abolished. However, this type of total visual loss is relatively uncommon and many patients retain at least some rudimentary visual awareness of motion or light. Visual form discrimination is profoundly impaired and objects, faces, or written words cannot be identified. Visuospatial orientation is also severely compromised and patients may repeatedly bump into objects when attempting to navigate in their environment.

Although individuals with cortical blindness do not have normal conscious awareness of visual events, they can sometimes demonstrate surprising ability to respond to stimuli presented within the blind portions of their visual fields. Specifically, patients may be able to detect, locate, and discriminate visual stimuli that they claim not to see. Residual visual capacity within the cortically blind field in the absence of conscious awareness has been referred to as "blindsight" (Weiskrantz, 1986; Stoerig & Cowey, 1997, 2007; Stoerig, 2006). The fact that blindsight can be observed in patients with extensive destruction or de-afferentation of primary visual cortex suggests that the preserved visual abilities of these individuals are mediated by neural pathways from retina to extrastriate visual cortex that bypass the damaged geniculostriate system. There are in fact a number of alternative non-geniculostriate pathways capable of transmitting visual information to a variety of temporo-parietal extrastriate cortical areas via subcortical relay nuclei in the midbrain and diencephalon (Weiskrantz, 1986; Stoerig & Cowey, 1997, 2007; Stoerig, 2006). The specific visual functions retained in blindsight may depend on which of these multiple parallel visual pathways are available to patients. For instance, the

residual capacity to detect motion and to locate and manually grasp targets presented in the blind field may depend on projections from the retina to the superior colliculus, with additional connections to the pulvinar and to middle temporal (MT/V5) and dorsal parietal cortex (Danckert & Rossetti, 2005). Other alternative pathways from retina to ventral extrastriate cortex or amygdala might be involved in mediating unconscious visual form discrimination (Trevethan, Sahraie, & Weiskrantz, 2007) and implicit recognition of emotional facial expressions (Morris, DeGelder, Weiskrantz, & Dolan, 2001; Pegna, Khateb, Lazeyras, & Seghier, 2005). Functional imaging studies in patients with blindsight are consistent with the notion that unconscious processing of visual information depends on non-geniculostriate visual pathways. Specifically, these investigations have demonstrated activation in midbrain/thalamic nuclei, extrastriate cortical areas, and the amygdala in the absence of concomitant activity in the lesioned primary visual cortex during visual stimulation of the blind field (Sahraie, Weiskrantz, Barbur, Simmons, Williams, & Brammer, 1997; Stoerig, 2006; Morris et al., 2001; Pegna et al., 2005).

Patients with cortical blindness may demonstrate unawareness of their profound visual impairment. This striking clinical condition is referred to as Anton's syndrome. Anosognosia for visual loss can take several different forms. In extreme cases, patients emphatically deny being blind and produce confabulatory responses when questioned about their visual abilities. Other patients acknowledge a change in their vision but they typically offer a variety of excuses to explain the difficulty, attributing it to poor lighting conditions or to a problem with their eyeglasses. A number of theories have been advanced to explain anosognosia for blindness (Bisiach & Geminiani, 1991; Heilman, 1991; Celesia, Brigell, & Vaphiades, 1997; Adair, Schwartz, & Barrett, 2003). For instance, it has been proposed that normal visual awareness depends on a hypothetical monitor located in extrastriate visual association areas that receives afferent input from primary visual cortex (Heilman, 1991). In addition to evaluating activity within the visual areas of the brain, the monitor also sends efferent information to cortical language areas enabling subjects to verbally comment on their visual experiences. Lesions that disrupt the input and/or output connections of the visual monitor, or produce damage to the monitor itself, result in anosognosia for visual impairment that may include verbal denial of the deficit and the production of confabulatory responses by the disconnected language areas. It has also been suggested that in cases of cortical blindness, internally generated visual experiences in the form of hallucinations or visual imagery may provide faulty input to an otherwise intact monitor. The loss of normal visual input following damage to primary visual cortex may in fact give rise to frequent "release" hallucinations in patients with cortical blindness due to increased cortical excitability in de-afferented extrastriate areas. Furthermore, since visual perception and imagery are mutually inhibitory under normal circumstances, the absence of bottom-up activation by geniculostriate afferent signals may be accompanied by a relative enhancement of visual imagery mediated by unopposed top-down activation of sensory representations in extrastriate cortex. Patients may deny blindness because they continue to experience internally generated visual sensations and may mistake these for veridical perceptions resulting from retinal stimulation by external visual events.

To understand anosognosia for visual loss, it is useful to briefly consider what is currently known about the neural correlates of normal visual awareness. In this context, it is important to emphasize that conscious awareness of visual events normally entails the capacity to acknowledge, describe, reflect upon, and make appropriate cognitive judgments about ongoing visual experiences (Weiskrantz, 1997; Block, 2005; Dehaene, Changeux, Naccache, Sackur, & Sergent, 2006). Thus, awareness and the ability to provide introspective report or commentary about the quality, content, and veridicality of visual perceptions are closely related functions. Although localized neural activity in cortical visual areas (striate and extrastriate cortex) is obviously necessary for visual information to reach this "commentary stage" of conscious awareness, it is by itself not sufficient (Weiskrantz, 1997; Rees, Kreiman, & Koch, 2002; Dehaene et al., 2006). In particular, evidence from recent neuroimaging studies suggests that conscious vision requires the additional recruitment of dorsolateral prefrontal and parietal cortical regions implicated in visual attention and working memory (Rees, Kreiman, & Koch, 2002; Dehaene et al., 2006). Prefrontal cortex is also involved in mediating strategic cognitive operations necessary for critically evaluating and interpreting the meaning and significance of visual experiences in order to determine the most appropriate behavioral response.

Activation of frontal and parietal regions by input from cortical visual areas is also likely to play an essential role in awareness of abnormal visual function. Specifically, damage to primary visual cortex and extrastriate areas may give rise to visual field defects and/or domain-specific impairments in processing distinct visual stimulus attributes (e.g., form, color, motion). If the same lesions also interfere with the bottom-up activation of the fronto-parietal cortical network that normally enables conscious

awareness and introspective report about visual experiences, anosognosia for the visual impairment ensues. Defective awareness of visual function may also result from direct damage to the putative fronto-parietal network that provides the critical top-down attentional amplification required for perceptual information processed within visual cortical modules to enter consciousness, or it may be produced by lesions that disrupt feedforward/feedback connections between fronto-parietal cortex and visual processing areas. In summary, conscious awareness of both normal and abnormal vision requires dynamic reciprocal interactions between cortical visual areas and specialized regions within frontal and parietal cortex. Lesions that disrupt the spatial distribution, intensity, or timing of activation across the different functional components of this large-scale neural system may give rise to a variety of clinical conditions characterized by unawareness of preserved or impaired visual processing, including blindsight, visual neglect, and anosognosia for blindness.

Evaluation

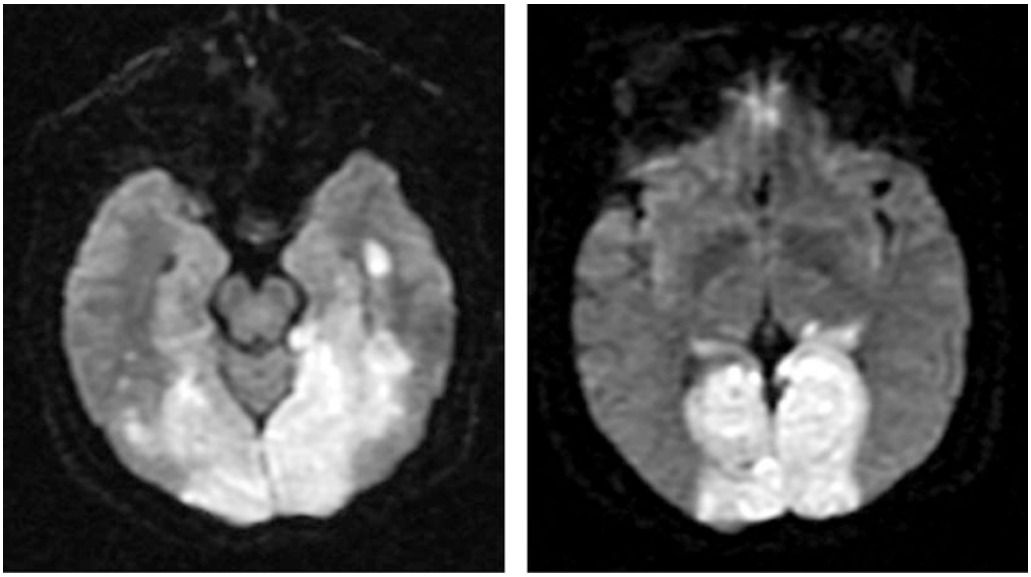
Neuro-ophthalmological examination in cortical blindness confirms the normal fundoscopic appearance of the retinas and optic nerves. The pupillary light response is characteristically preserved, as the retinal fibers that mediate this reflex leave the optic tract prior to the origin of the geniculostriate pathways. Reflexive blinking to threatening visual gestures is usually absent, and in the majority of cases optokinetic nystagmus cannot be elicited by moving a striped object in front of the patient's eyes. The dissociation between preserved pupillary light response and absent optokinetic nystagmus can have diagnostic utility in distinguishing patients with cortical blindness from patients with severe peripheral visual loss due to bilateral eye or optic nerve pathology in whom both responses are lost, and also from individuals with psychogenic blindness in whom both responses are retained.

Clinical evaluation of visual function in patients with cortical blindness should include tests of visual acuity, determination of light and motion perception in different sectors of the visual field, tests of spatial localization, as well as assessments of color perception, object and face recognition, and reading ability. Studying blindsight usually requires the use of specialized testing equipment in an experimental laboratory environment. However, in the clinical setting blindsight can be tested by requiring patients to guess the presence/absence, location, movement, or identity of objects presented in their blind field using a forced-choice response method (e.g., was it a key or a coin?). Better than chance performance on these types of tasks is taken to be indicative of blindsight. Alternatively, patients can be asked to try to reach for and grasp objects in their blind field. Regardless of the testing method used, it is important to establish after each trial whether the patient had any conscious awareness of the visual stimulus. Visual imagery can be tested by asking the subjects to answer questions about the visual attributes of familiar objects or animals (e.g., do polar bears have long or short tails?). Patients should be questioned about abnormal visual perceptions and hallucinations. Unawareness of deficit may be revealed by spontaneous comments or behavior, but patients should also be specifically asked to describe the quality and content of their visual experiences. The severity of anosognosia can be quantified by simple rating scales (Bisiach & Geminiani, 1991; Celesia, Brigell, & Vaphiades, 1997).

Structural neuroimaging (CT/MRI) studies in patients with cortical blindness due to stroke typically reveal extensive bilateral infarctions involving primary visual cortex and underlying white matter, often with evidence of lesion extension into adjacent extrastriate visual association areas (Brodmann areas 18/19, 37) (Figure 1). SPECT/PET scans frequently demonstrate blood flow/metabolic abnormalities that extend beyond the boundaries of the lesions seen on CT/MRI, providing evidence that the cortical visual areas that appear spared by structural imaging studies are in fact functionally compromised. In patients with transient cortical blindness due to RPLS, neuroimaging studies have demonstrated reversible bilateral subcortical white matter abnormalities in posterior occipito-temporo-parietal regions attributable to vasogenic edema.

Treatment

A number of behavioral approaches have been tried with varying degrees of success to restore visual function in the cortically blind field and/or help patients learn alternative strategies to compensate for their visual impairment (Kerkhoff, 2000). Attempts to increase awareness of the visual deficit in patients with anosognosia constitute an important component of the treatment program. It has been shown that blindsight performance can be improved by training, and the use of these techniques may aid the recovery of vision in individuals with cortical blindness (Stoerig, 2006; Sahraie et al., 2006).



Cortical Blindness. Figure 1 Diffusion-weighted MRI scan in a patient with cortical blindness following bilateral posterior cerebral artery strokes. Note massive destruction of primary visual cortex (Brodmann area 17), with lesion extension into ventral extrastriate visual association areas (Brodmann areas 18,19,37). This individual demonstrated complete denial of blindness (anosognosia), consistent with Anton's syndrome

Cross References

- ▶ Anosognosia
- ▶ Visual Hallucinations

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Cortical Color Blindness

► Achromatopsia

Cortical Contusion

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Synonyms

Bruise; Contusion (cerebral)

Definition

Cortical contusions are bruises on the brain tissue that form from the small blood vessel leaks (veins and arteries covering the parenchymal tissue), or a series of microhemorrhages following trauma. Trauma is usually the result of physical blows to the head such as those sustained in a motor vehicle accident, direct blow to the head from assault, or significant sports-related injuries. Veins and arteries on the surface of the brain are damaged, which results in bleeding and bruising. When the blood vessel is torn, blood escapes from the vessel at a rate that is faster than the blood that can be absorbed by the brain. Consequently, cortical contusions commonly result in edema and increased intracranial pressure.

Current Knowledge

Second to diffuse axonal injury, cortical contusion is the most common type of intra-axial lesion following brain trauma. By radiologic definition, a cortical contusion must involve some portion of the superficial gray matter. Because gray matter has more vasculature than white matter, most cortical contusions are hemorrhagic, whereas diffuse axonal injuries are rarely hemorrhagic. The frontal and temporal lobes are the most common sites of cortical contusions. When present, cortical contusions are usually found bilaterally. Compared to diffuse axonal injury lesions, cortical contusions are much less likely to involve an initial presentation of coma or altered loss of consciousness.

Cross References

► Contrecoup Injury
► Edema
► Focal Lesion, Contusion
► Intracranial Pressure

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Cortical Deafness

► Pure Word Deafness

Cortical Lewy Body Disease (CLBD)

► Dementia with Lewy Bodies

Cortical Magnification

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Definition

Cortical magnification refers to the fact that the number of neurons in the visual cortex responsible for processing the visual stimulus of a given size varies as a function of the location of the stimulus in the visual field. Stimuli occurring in the center of the visual field that have been detected in the fovea of retina are processed by a very large number of neurons in the primary visual cortex of the occipital lobe, though these neurons handle only a very small region of the central visual field. Conversely,

stimuli detected in the peripheral visual field tend to be processed by a much smaller number of neurons in the primary visual cortex.

Current Knowledge

Cortical magnification reflects an important concept in the field of cognitive neuroscience; the cortical volume, and ultimately the number of neurons allocated to a particular function, typically varies as a function of the significance of the function. For example, since the sense of touch is particularly important for the hands, there are many more nerve receptors in the finger tips than in the trunk of the body. Similarly, the volume of motor cortex dedicated to controlling the hands and mouth in humans is much greater than the volume dedicated to large muscle groups with more limited action. Given the critical role that vision plays in human cognition, tremendous magnification of neurons is dedicated to visual processing, with this magnification occurring at various processing stages along the visual pathways beginning in the retina. The extent of cortical magnification is often expressed as a ratio of millimeters of cortical surface per degree of visual angle. This ratio varies across visual areas. Among primates, neurons devoted to processing foveal input from the retina are about 100 times more prevalent than neurons devoted to peripheral stimuli in the primary visual cortex (Daniel & Whitteridge, 1961).

The principle of cortical magnification indicates an important relationship between the number of neurons dedicated to big or small visual angles and the receptive field of those neurons. When a large number of neurons are involved in a small visual angle, there is inherently a large processing capacity being assigned to a smaller area of visual focus. Conversely, a smaller number of neurons handling a visual angle are indicative of a larger receptive field, as each neuron must be sensitive to changes occurring across a larger area of space. This creates an inherent relationship between the spatial frequency of the visual information being processed and the size of the receptive field that is essential for considering how neurons across different visual cortical areas are tuned to respond to the featural and spatial characteristics of visual input. A consequence of this organization for the primary visual cortex is that visual acuity and the ability to detect small features of stimuli are best in the center of the visual field and poorest in the periphery. Yet, broad spatial changes with movement are easily detected at the periphery. Since visual cortical areas differ in their emphasis on specific informational characteristics (e.g., shape, color, texture, position, movement), neurons with greatest sensitivity to these dimensions vary as a function of cortical magnification factors and a tradeoff of size of the receptive field and the number of neurons dedicated to each part of the field.

Cross References

- ▶ Feature Detection
- ▶ Magnocellular Neurons
- ▶ Parvocellular Neurons
- ▶ Spatial Frequencies
- ▶ Spatial Processing

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Cortical Malformation

- ▶ Heterotopia

Cortical Mapping

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Synonyms

Direct stimulation mapping; Electrical stimulation mapping (ESM); Functional mapping

Description

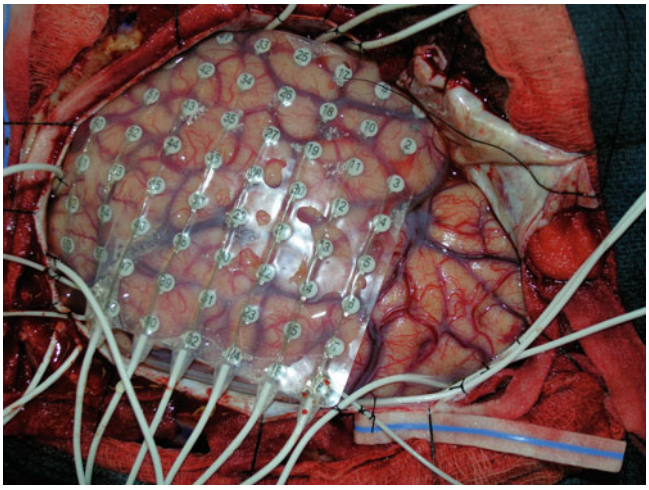
Cortical mapping is an invasive procedure in which electrical stimulation is applied briefly to the cortical

surface for the purpose of identifying areas critical for sensory, motor or language function. This procedure is utilized when brain surgery involves the removal or disruption of potentially functional cortical areas. Sites identified via cortical mapping are typically spared from resection, with the goal of preserving function postoperatively.

Stimulation is applied using a bipolar stimulator, usually via pairs of adjacent subdural electrodes. The procedure can be conducted intra-operatively in a conscious patient before resection of brain tissue, or extra-operatively, if subdural electrodes have been implanted, most commonly in pharmacologically resistant epilepsy patients who require intracranial EEG monitoring to delineate the region of seizure onset (Fig. 1). The identification of sensory and motor sites is based on stimulation-evoked "positive" responses, such as a subjective sensation (e.g., tingling) or an observable movement (e.g., muscle twitch). In contrast, stimulation of language cortex elicits no experiential or observable response in an inactive patient. Rather, cortical language mapping relies on "negative" responses, in that the patient must be engaged in a language task and stimulation of language cortex will disrupt task performance. Thus, for language mapping, stimulation produces a discrete, reversible lesion, theoretically enabling the examiner to observe the functional consequences of damage to the site(s) stimulated. Stimulation of frontal language cortex typically produces speech arrest, whereas stimulation of posterior (temporal/parietal) language cortex typically elicits comprehension, naming or reading difficulties. Because cortical language mapping is based on negative responses, thorough mapping of language cortex requires administration of tasks assessing a range of language functions.

Depending on the location of the area identified and the nature of the response elicited by stimulation, it is generally held that removal of a positive sensory, motor, or language site identified by stimulation will result in impaired function postoperatively. Of the few clinical series published, results suggest that postoperative function is best preserved when the resection margin exceeds 1 cm from the functional site. On the other hand, there is some evidence that certain sites identified by stimulation can be removed without concern of postoperative decline. These include motor sites identified in the supplementary motor area, tongue, and lower face areas (due to their bilateral representation), and possibly, language sites identified in the basal temporal region, although this is somewhat controversial.

For motor and sensory mapping, the duration of electrical stimulation is typically 2 s, whereas language mapping typically requires 4–8 s of stimulation, depending on the particular task under assessment. The level of stimulation administered ranges from 1 mA to a maximum of 17 mA, with motor and sensory cortex typically utilizing <10 mA and language cortex typically requiring >10 mA. Under normal circumstances, cortical stimulation causes neither pain nor discomfort. One risk of stimulation, however, is the evocation of a seizure, particularly in patients with epilepsy, due to a likely lower seizure threshold in epileptogenic areas. To minimize the probability of a stimulation induced seizure, EEG is monitored on an ongoing



Cortical Mapping. Figure 1 Implanted subdural electrode grid used for EEG recording and electrical stimulation mapping

basis, and the stimulation intensity is lowered when abnormal discharges are associated with stimulation. Nevertheless, benzodiazepines are typically kept close at hand for IV administration for instances when a seizure occurs and fails to resolve rapidly on its own.

Historical Background

Alteration of function via cortical stimulation in both animals and humans dates back to the mid 1800s. The procedure came into clinical use in the early twentieth century in association with surgical resection of epileptogenic cortex in patients with pharmacologically refractory epilepsy. Initially used to identify sensory and motor cortex, Wilder Penfield and colleagues pioneered the technique in the 1950s for use in the identification of language cortex. Stimulation-based language mapping was further refined by George Ojemann and colleagues who essentially established the current clinical standards. In addition to its clinical utility, investigators have used the opportunity provided by clinical stimulation to investigate structure-function relations. These studies build upon the lesion model, contributing more precise information regarding functional localization due to both the controlled setting, and the smaller, more discrete "lesions" induced temporarily by stimulation than that typically found with naturally occurring lesions.

Psychometric Data

Cortical mapping procedures remain unstandardized. Due to its highly invasive nature, data from cortical mapping are based on clinical rather than normal populations, and therefore, classic psychometric data are unavailable. It has also been difficult to assess reliability, as cortical mapping is rarely performed more than once in the same patient. Nevertheless, patients with indwelling subdural grids who undergo mapping over 2 or more days, and patients who require a second surgery involving adjacent brain regions provide opportunities for repeat mapping, although these circumstances are relatively infrequent. In the absence of published studies addressing this issue, anecdotal reports suggest a reasonable level of consistency in the location of simulation-identified sites within individuals. Across individuals, consistency is relatively high for the location of motor and sensory cortex. Frontal language cortex is slightly more variable, with most positive sites clustered in the frontal opercular region, anterior to the tongue area. The location of posterior temporal and parietal language sites appears to vary more among individuals, although this might merely reflect alterations in the distribution of language sites in patients with epileptogenic cortex in the temporal region.

Clinical Uses

One of the main challenges with brain surgery involving cortical regions is to remove a sufficient amount of pathological tissue without removing areas critical for function. Cortical mapping is used to identify cortical regions critical for function, in order to spare these areas from resection or protect them from damage during surgical procedures. Cortical mapping is typically performed when there is concern that resective brain surgery (e.g., tumor resection, epilepsy surgery) might impinge upon, or possibly overlap with cortical regions necessary for function.

Cross References

- ▶ Epilepsy
- ▶ Functional Imaging
- ▶ Localization

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Cortical Motor Pathways

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Synonyms

Corticospinal tract; Pyramidal tract; Voluntary motor tract

Definition

The cortical motor pathway consists of four regions of the cerebral cortex (primary motor cortex, posterior parietal cortex, premotor cortex, and supplementary motor cortex) whose neuronal cell bodies are located in layer V (five) and whose projections are involved with the execution of muscle contraction largely on the contralateral side of the body.

Current Knowledge

The cortical motor pathway describes a trajectory of fibers whose cells of origin are located in layer V of the cerebral motor cortex. The cerebral motor cortex is a term that describes the four main areas of the cerebral cortex that contribute to the planning, control, and execution of voluntary motor fibers; the primary motor cortex (M1), secondary motor cortices, premotor cortex, and the supplementary motor area (SMA).

Cortical Location and Overall Function

The primary motor cortex (M1) is located in the frontal lobe of the brain and the cells of origin, in layer V, are found within the precentral gyrus. These cells generate neural impulses that control the execution of movement directed to skeletal muscles on the contralateral side of the body. Other regions of the cortex that contribute to the cortical motor pathway – termed secondary motor cortices – include the posterior parietal area (PMA), the premotor cortex (PMC) and the supplementary motor area (SMA). The posterior parietal cortex is responsible for transforming visual information into motor commands relayed via the premotor and supplementary motor area. The premotor cortex is involved in sensory guidance of movement and control of proximal and trunk muscles of the body. The supplementary motor area is involved in the planning and coordination of complex movements, such as those requiring coordination of two-handed movement.

Multiple pathways arise from the efferent projections of the motor cortices. Neuronal cell bodies located in layer V of the M1, SMA, and premotor cortex send vast projections that collectively give rise to the largest single pathway, the pyramidal or corticospinal tract. The tract descends through the internal capsule and forms the pyramids of the medulla, crossing midline at the pyramidal decussation, ultimately terminating in the ventral horn of the cervical-through-lumbar spinal cord. These cortical motor pathways work in tandem with other cortical motor areas of the brain – notably the cerebellum and subcortical motor nuclei (the basal ganglia) – to execute planning and control of voluntary motor activity. Other projections from layer V of M-I include the corticostriatal fibers to the striatum (caudate and putamen), corticorubral fibers to the red nucleus, and projections that terminate in the reticular formations with the medulla and pons of the brain stem. M-I projections also modulate other motor areas within the cortex and include reciprocal connections with the supplementary motor area, the premotor and posterior parietal motor areas. These fibers are part of a vast reciprocal network that cross via the corpus callosum and are referred to as callosal connections. The SMA contributes to the corticospinal tract and has reciprocal callosal projections to contralateral areas of the motor cortex. The premotor cortex (PMC) contributes to the corticospinal tract and also has extensive reciprocal connections to both SMA and MI.

Cross References

- ▶ Cerebral Cortex
- ▶ Decerebrate Posturing
- ▶ Decorticate Posturing
- ▶ Hemiplegia
- ▶ Homunculus
- ▶ Internal Capsule
- ▶ Periventricular White Matter
- ▶ Precentral Gyrus
- ▶ Pyramidal System
- ▶ Sensorimotor Assessment
- ▶ Supplementary Motor Area

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Cortical–Subcortical Loop

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Synonyms

Basal ganglia-thalamocortical circuit; Cortico-basal ganglia loop

Definition

The cortical–subcortical loop describes a class of distinct, parallel circuits that connect specific regions of cerebral cortex with the basal ganglia and specific thalamic nuclei. The thalamic nuclei complete the loop by projecting back to the same regions of cortex from which the circuits originate.

Current Knowledge

Several distinct, anatomically segregated cortical–subcortical loops may be characterized based on the functional role of the cortical regions involved.

Motor loop. The motor loop plays a role in the preparation and execution of movement. Primary motor cortex and associated premotor areas project via the putamen to the ventral tier nuclei of the thalamus, which then complete the loop with projections back to motor cortex. Somatotopic organization is maintained through all stages of this circuit.

Oculomotor loop. The oculomotor loop is involved in the control of eye movements. The frontal eye fields (FEF) and supplementary eye fields (SEF) project via the body of the caudate nucleus to the ventral anterior (VA) and mediodorsal (MD) thalamic nuclei, which complete the loop by projecting back to FEF and SEF.

Prefrontal associative loops. The prefrontal associative loops describe two distinct components, which subserve different aspects of higher cognitive processing. The "dorsolateral prefrontal loop" plays a role in cognitive processes including spatial memory and working memory. In this circuit, the dorsolateral prefrontal cortex (PFC) projects via the dorsolateral head of the caudate to VA and MD thalamic nuclei, which then project back to dorsolateral PFC. The "lateral orbitofrontal loop" plays a role in cognitive processes including the ability to select and shift behavioral sets, and response inhibition related to social context or emotional subject matter. In this loop, the lateral orbitofrontal cortex (OFC) projects via the ventromedial caudate nucleus to VA and MD thalamic nuclei, which in turn send projections back to lateral OFC.

Affective-motivational loop. This loop is also called the "Limbic loop." This circuit plays a role in emotional and motivational behaviors. Widespread areas of "limbic cortex" including the anterior cingulate gyrus, medial OFC, and portions of the temporal lobe all send projections via the nucleus accumbens to MD thalamus. The circuit is completed by thalamocortical projections from MD to the anterior cingulate and medial OFC.

Clinical Disorders and Treatment Approaches

An understanding of the architecture of cortical–subcortical loops has given rise to a prevailing view of numerous clinical disorders as essentially circuit disorders, arising from abnormal neuronal activity at some stage of the finely tuned circuit. The best-studied examples involve the motor loop, where disturbances within the circuit can result in either hypokinetic movement disorders (e.g., Parkinson's disease) or hyperkinetic disorders (e.g., chorea, ballismus, and dystonia). In addition, abnormal activity within the non-motor loops may be associated with disorders as diverse as obsessive–compulsive disorder, schizophrenia, and Tourette syndrome. Recently, interventions that surgically remove or modify (e.g., with deep brain stimulation) the dysfunctional component of the cortical–subcortical loop have met with considerable success. These promising treatment approaches are the subject of intensive ongoing research.

Cross References

▶ Basal Ganglia
▶ Deep Brain Stimulator (Parkinsons)
▶ Parkinson's Disease

References and Readings

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Corticobasal Degeneration

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Synonyms

Corticobasal syndrome; Corticodentatonigral degeneration with neuronal achromasia

Short Description or Definition

First described as corticodentatonigral degeneration with neuronal achromasia by Rebeiz et al. in 1968, corticobasal degeneration (CBD) was long thought to be predominantly a motor disorder. Indeed, the original description of the disorder emphasized the relative preservation of mental faculties. More recently, emphasis has been placed on the neurobehavioral features of CBD, and the overlapping clinical and neuropathological features of CBD with frontotemporal lobar degenerations continue to generate debate about the classification and nosology of the disorder.

The motor presentation of CBD most often involves an asymmetric, progressive, akinetic-rigid parkinsonism of gradual onset that responds minimally if at all to levodopa and is sometimes accompanied by dystonia or myoclonus. Cortical signs that are common in CBD include asymmetric apraxia, cortical sensory signs (e.g., astereognosis, graphesthesia), and alien hand sign. The latter may involve a sense of lack of ownership of the limb in the absence of visual cues, involuntary purposeful movements, or frank interference of one limb with the other's execution of purposeful movement. These motor and cortical signs are core features of CBD.

Categorization

The neurobehavioral expression of CBD can be quite variable, and cases with confirmed CBD neuropathology have presented with features suggestive of primary progressive aphasia and frontotemporal dementia. Coupled with the recognition of CBD as a tauopathy, the occasional neurobehavioral resemblance of CBD to frontotemporal dementia and primary progressive aphasia has lead some to argue that CBD is a member of the "Pick complex" of disorders (Kertesz, 2003). Given the clinically heterogeneous presentation of CBD, and the fact that the core features of CBD can be produced by other conditions, it has been recommended that the term corticobasal syndrome (CBS) be applied to the core clinical features of CBD regardless of etiology. In contrast, the term corticobasal degeneration (CBD) should be reserved for the distinctive neuropathological condition of CBD, regardless of its clinical presentation (Lang, 2003).

From a neuropathologic standpoint, CBD, like frontotemporal lobar degeneration, has been categorized as a tauopathy. Tau is a microtubule-associated protein that promotes the polymerization of tubulin and thus, microtubule assembly. The human tau gene, containing 16 exons, is located on the long arm of chromosome 17 and encodes for the six isoforms of tau found in the central nervous system. The isoforms differ by the presence or absence of amino acid inserts encoded by exons 2, 3, and 10. Whether the transcript of exon 10 is spliced in or out of the final tau protein product determines whether the isoform has three or four repeated microtubule-binding domains (three isoforms have three repeats and three isoforms have four repeats). The four repeat isoforms of tau (4R tau) promote microtubule assembly at more than twice the rate of the three repeat (3R tau) isoforms. Although the expression of 3R and 4R tau is cell-type specific, the 3R tau expression in normal human brain is 1–1.5-fold higher than the 4R expression level. In spontaneous and genetic CBD, 4R tau represents the main pathological inclusion. Recent findings that mutations associated with parkinsonism (in LRRK2) and frontotemporal lobar degeneration (in progranulin) can be seen in some cases presenting with corticobasal syndrome further highlight the heterogeneity of corticobasal syndrome (CBS).

Autopsy in CBD cases reveals asymmetric frontal and parietal atrophy, depigmentation of the substantia nigra without Lewy bodies, and often the presence of ballooned cells in cortex. Tau-positive astrocytic plaques, oligodendroglial coiled bodies, and threadlike lesions are seen in white and gray matter, especially the superior frontal and parietal gyri and the pre- and post-central gyri, and in the striatum.

Epidemiology

Prevalence and incidence of CBD are unknown, and poor diagnostic accuracy no doubt contributes to this. Although the H1/H1 tau haplotype has been identified as heightening susceptibility to both CBD and progressive

supranuclear palsy, no clear genetic etiology has been identified. Dementia and other cognitive and behavioral abnormalities were thought to be rare in CBD until the last decade, but it is now appreciated that the frequency of neurobehavioral abnormalities observed as a presenting problem or during the course of the condition is quite high. It might be that the inconsistent incidence and prevalence estimates of cognitive impairment in CBD are a function of whether patients were drawn from movement disorder, dementia, or psychiatry clinics.

Natural History, Prognostic Factors, Outcomes

Disease onset is usually in the sixth decade of life, and mean time to death from diagnosis is about 7 years.

Neuropsychology and Psychology of Corticobasal Degeneration

CBD involves an asymmetric apraxia, most often of the ideomotor type, but ideational and limb kinetic apraxias also occur. Thus, patients most often have difficulty demonstrating the use of tools. Poor drawing (constructional apraxia) is also commonly seen. Language disturbance occurs early or during progression of the syndrome, and the aphasia is most often non-fluent (about 56% of cases) or anomic (30%). The pattern of performance on language tests in patients with the traditional CBD presentation is somewhat inconsistent across studies, but phonological impairments may be an important feature. Performance on verbal fluency tests, especially lexical or phonemic fluency tests, is usually impaired either due to the executive demands of those tasks or aphasia. Performance on semantic memory tasks such as conceptual matching and visual confrontation naming and expressive vocabulary is relatively preserved and impaired in a minority of patients, although some studies have reported considerable impairment on semantic tasks early in the disease. When naming is impaired, disproportionate benefit is derived from cuing suggesting a retrieval rather than semantic memory deficit. Comprehension is typically preserved early, but comprehension of grammatically complex material declines with disease progression.

Executive dysfunction, as indicated by poor performance on tasks such as the card sorting tasks and the Trailmaking test is common. Episodic memory impairments in CBD are relatively mild early in the course of the condition and appear to involve both encoding and retrieval deficits. Remote memory has been little studied in CBD, but the pattern of poor recall but intact recognition on remote memory tasks suggests a retrieval deficit. Visuospatial impairments have been observed.

With respect to emotional and neuropsychiatric issues, depression is common in CBD (73% of patients), though apathy (40%), irritability (20%), and agitation (20%) also occur with frequency.

Evaluation

The selection of specific neuropsychological tests in CBD, like any other condition, should be guided by the referral questions and the patient's ability to cooperate and meet task demands. However, tests of executive function (e.g., planning, abstraction, and cognitive flexibility), praxis, visuospatial functions, attention, learning and memory, and word retrieval should be employed. Symptom inventories relating to apathy, depression, and "frontal" behavior syndromes, such as the Neuropsychiatric Inventory, Hamilton depression scale, and Frontal Systems Behavior Scale can be helpful in characterizing neuropsychiatric features of CBD.

Treatment

Some cases may show transient improvement in parkinsonian features with levodopa treatment; dopamine agonists are generally even less helpful than levodopa. Tremor, if present, may be alleviated by benzodiazepines. Antidepressants with anticholinergic profiles are to be avoided given possible adverse cognitive side effects, but selective serotonin reuptake inhibitors may be helpful in treating depression. Speech therapy is helpful in treating dysphagia.

Cross References

- ▶ Basal Ganglia
- ▶ Corticobasal Degeneration
- ▶ Frontal Lobes
- ▶ Frontal Temporal Dementia
- ▶ Frontotemporal Lobar Degeneration
- ▶ Gait Disorders
- ▶ Movement Disorders
- ▶ Parkinson Plus Syndromes
- ▶ Parkinson's Disease
- ▶ Parkinsonian Movement

► Striatum
► Tauopathy

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Cortico-Basal Ganglia Loop

► Cortical–Subcortical Loop

Corticobasal Syndrome

► Corticobasal Degeneration

Corticodentatonigral Degeneration with Neuronal Achromasia

► Corticobasal Degeneration

Corticoliberin

► Corticotropin-Releasing Hormone

Corticospinal Tract

► Cortical Motor Pathways

Corticotropin-Releasing Factor (CRF)

► Corticotropin-Releasing Hormone

Corticotropin-Releasing Hormone

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Synonyms

Corticoliberin; Corticotropin-releasing factor (CRF)

Definition

Corticotropin-releasing hormone (CRH) is a hormone that is primarily produced by the hypothalamus and is involved in the stress response. It is released from the paraventricular nucleus of the hypothalamus with the primary action within the anterior lobe of the pituitary to initiate the release of adrenocorticotropic hormone (ACTH). CRH (41 amino acids long) is derived from a 191 amino acid preprohormone. Other areas of CRH synthesis include peripheral tissues, and it is highly expressed in the placenta.

Cross References

► Hormone

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Cortisteroids

► Steroids

Coumadin®

► Warfarin (Coumadin)

Counseling

► Psychotherapy

Counseling Relationship

► Therapist–Patient Relationship

Counterindicant

► Contraindication

Couples Therapy

► Cognitive Behavioral Couples Therapy

COWA

► Controlled Oral Word Association Test
► F-A-S Test
► Verbal Fluency

COWAT

► Controlled Oral Word Association Test
► F-A-S Test
► Verbal Fluency

CPM

► Raven Matrices

CPRS

► Conners Rating Scales

CPT

► Continuous Performance Tests

Craig Handicap Assessment and Reporting Technique

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Synonyms

CHART; CHART-SF

Description

The Craig Handicap Assessment and Reporting Technique (CHART) is a 32-item instrument designed to provide a simple, objective measure of the degree to which impairments and disabilities result in handicaps (societal participation limitations) for adolescents and adults (15 years and older) in the years after initial rehabilitation. The CHART includes six subscales (physical independence, cognitive independence, mobility, occupation, social integration, and economic independence), which closely reflect the disablement model developed by the World Health Organization (WHO, 1980, 2001). Each subscale contains 3–7 questions, which together quantify the extent to which individuals fulfill various social roles. CHART focuses on objective, observable criteria that are easily quantifiable and unlikely to be open to subjective interpretation. Each of the domains or subscales of the CHART has a maximum score of 100 points, which is considered to be the level of performance typical of the average nondisabled person. High subscale scores indicate less handicap or higher social and community participation.

The CHART was developed in 1992 for use with persons with spinal cord injury (SCI) and originally did not address the WHO handicap dimension described as "orientation" (Whiteneck, Charlifue, Gerhart, Overholser, & Richardson, 1992). The current CHART was revised in 1995 with the addition of a "Cognitive Independence" subscale (to assess orientation) and has proven to be an appropriate measure of societal participation that can be used with individuals having a range of physical or cognitive impairments (Mellick, Walker, Brooks, & Whiteneck, 1999). A 19-item Short Form with subscales closely approximating the subscale scores for the CHART long form is recommended for those applications or populations in which time is at a minimum (Whiteneck et al., 1998).

The CHART was designed to be administered by interview, either in person or by telephone and takes approximately 15 min to administer. Participant-proxy agreement across disability groups on the CHART has provided evidence in support of the use of proxy data for persons with various types of disabilities (Cusick, Brooks, & Whiteneck, 2001). There is no set time period for administering the CHART; however, it is recommended that multiple measurements be taken over the course of a person's lifetime to assess changes with adaptation to the disability and to gain insight into changes in participation, which may occur over time.

Historical Background

WHO describes a conceptual model of disablement that includes impairment at the organ level, disability describing functional status, and "handicap," or more recently, "participation," encompassing the roles one plays in the world and society. Despite its importance as a rehabilitation goal, handicap (absence of social participation) is the least often measured of all rehabilitation outcomes. A great deal of work has been done in developing tools to measure and document impairment and disability; however, limited attempts have focused on the measurement and assessment of long-term participation limitations (handicap), despite the fact that psychosocial adjustment is clearly regarded as the ultimate outcome of rehabilitation. The CHART was specifically developed to help fill this gap – to assess the WHO dimensions of handicap and to provide a simple, objective measure of the degree to which impairments and disabilities result in participation limitations in the years after initial rehabilitation.

The model of disablement suggested by the WHO provides useful conceptual distinctions for impairment, disability, and handicap (WHO, 1980). In practical terms, *impairment* occurs at the organ level, representing any loss or abnormality of psychological, physiological, or anatomical structure or function. *Disability* occurs at the person level, demonstrated as any restriction or lack of ability (resulting from impairment) to perform any activity in the manner or within the range considered normal for a human being. *Handicap* occurs at the societal level. It is a disadvantage for a given individual, resulting from an impairment or a disability that limits or prevents the fulfillment of a role that is normal (depending on age, sex, and social and cultural factors) for that individual. The initial disablement model, the "International Classification of Impairment, Disability and Health (ICIDH)"

(WHO, 1980), was later revised as the "International Classification of Functioning, Disability and Health (ICF)" (WHO, 2001). The domain of "handicap" was reconceptualized and changed to "participation." The migration away from the use of "handicap" toward the more widespread use of "participation" is evident in literature published since 2001. According to the WHO, handicap (participation) describes the total effects and interplay of all the consequences of disability: social, economic, cultural, and environmental.

Each CHART dimension of handicap is characterized by directly observable qualities which lend themselves to easy quantification. While an infinite number of factors might have been included, to keep the instrument to a practical length the following dimensions have been operationalized based on the WHO definitions.

Physical Independence is the individual's ability to sustain a customarily effective independent existence. The major component of this subscale is the number of hours per day someone is needed to provide routine or occasional assistance (whether paid or unpaid). Individuals are viewed as somewhat less handicapped if they take primary responsibility for instructing and directing people who are providing assistance to them.

Cognitive Independence is the individual's ability to sustain a customary level of independence without the need for supervision. The factors included in this subscale reflect the amount of hours that a person needs supervision both inside and outside the home, as well as the amount of difficulty an individual has in remembering, communicating, and managing money.

Mobility is the individual's ability to move about effectively in his/her surroundings and is demonstrated by the hours per day out of bed, days per week out of the house, nights per year spent away from home, accessibility of the home, and transportation utilization.

Occupation is the individual's ability to occupy time in the manner customary to that person's sex, age, and culture. The time spent in various activities is used to measure this dimension. The relative value society places on different activities is used to weight the time in each category. Although there was a potential for subjective bias based on value judgments in developing the scale in this dimension, priority has been give to gainful employment, schooling, and active homemaking and maintenance, and this prioritization has been supported by validity and reliability testing. Other elements documented include volunteer work, recreational pursuits, and self-improvement activities.

Social Integration is the individual's ability to participate in and maintain customary social relationships. The factors included in this subscale include household composition, romantic involvement; the number of relatives, business associates, and friends with whom regular written or oral contract is maintained, and the frequency of initiating conversations with strangers.

Economic Self-Sufficiency is the individual's ability to sustain customary socioeconomic activity and independence. This dimension is defined as the remaining disposable household family income after nonreimbursed medical expenses have been excluded.

Psychometric Data

Initial calibration of the CHART scoring system was based on administration of the instrument to 88 able-bodied individuals and 100 persons with SCI. Once the norms had been established, two studies were conducted to assess the psychometric properties. CHART showed high test–retest reliability – 0.93 for the total score and from 0.80 to 0.95 for the subscales. The correlation of subject-proxy scores was 0.83 for the total CHART score. Rasch analysis established that CHART is a well-calibrated linear scale, with a good fit of both items and persons to its data (Whiteneck et al., 1992).These studies established the CHART as a reliable and valid instrument, as well as a well-calibrated linear scale (Whiteneck et al.; Dijkers, 1991). A decade later, subsequent testing on a group of 236 persons with SCI reported similar results – test–retest correlations of 0.87 and subject-proxy correlations of 0.85 were reported (Whiteneck, Brooks, & Mellick, 1997).

The Revised CHART which included the "Cognitive Independence" subscale was tested on 1,110 persons in six impairment categories – SCI, traumatic brain injury, stroke, multiple sclerosis, amputation, and burn (Whiteneck , Brooks et al., 1997). Results indicated that the cognitive subscale of the CHART was reliable and enhanced the appropriateness of the CHART in assessing handicap among persons having cognitive impairments (Mellick et al., 1999). Participant-proxy agreement across the six disability groups provided evidence in support of the inclusion of proxy data for persons with various types of disabilities (Cusick et al., 2001)

In an effort to reduce the number of items in the CHART, a short form was developed. A multidimensional analysis was performed which showed that fewer variables were needed to obtain CHART scores. Regression analyses were performed on each subscale with the dependent measure being the scale score and the variables contributing to the subscale acting as the predictor variables. All CHART subscale scores could be reduced by fewer questions to reach

90% explained variance except Economic Self-Sufficiency, which using the main variables could only explain 45%.

For additional information about the development, testing, and scoring procedures for the CHART and CHART-SF please consult the Guide for use of the CHART: Craig Handicap Assessment and Reporting Technique at www.craighospital.org/Research/CHART.

Clinical Uses

The CHART is a useful tool to measure handicap (participation limitations) in populations with injury or chronic illness with or without rehabilitation intervention. The CHART is designed as an interview tool, which can be administered face-to-face or by telephone. Each item on the instrument has been carefully and concisely worded to minimize ambiguity of interpretation. It is possible to use the instrument as a mailed questionnaire, although some valuable data potentially would be lost in the absence of interaction with an interviewer providing consistent prompts. There is no set time period for administering the CHART; however, it is recommended that multiple measurements be taken over the course of a person's lifetime to assess changes with adaptation to the disability and to gain insight into changes in participation which may occur over time. CHART has been demonstrated to be a reliable measure of societal participation limitations in a variety of populations including people with physical and/or cognitive functional limitations.

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► CHART Short Form

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Craig Handicap Assessment and Reporting Technique (CHART) Short Form

► CHART Short Form

Cramping

► Dystonia

Cranial Aerocele

► Pneumocephalus

Cranial Nerves

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Definition

Cranial nerves serve as conduits for communication between the brain and the body, providing motor and sensory innervation to structures in the head and neck as well as the thoracic and abdominal viscera. There are 12 pairs of cranial nerves, each of which is designated by a Roman numeral and a name (see Table 1). Roman numerals I–XII indicate the rostrocaudal order in which cranial nerves originate from the brain, while the name designated to each pair of cranial nerves denotes either its function or distribution.

Historical Background

The enumeration of the cranial nerves can be traced back to second century Greek physician–philosopher Galen; whose initial description included 7 of the 12 currently accepted pairs. English physician and neuroanatomist Thomas Willis' reclassification of the cranial nerves in the seventeenth century consisted of nine pairs and superseded Galen's previous description. German anatomist Samuel Thomas von Soemmerring introduced the contemporary classification system, comprising 12 pairs of cranial nerves, in the late eighteenth century.

Current Knowledge

Cranial Nerve Nuclei. The majority of cranial nerves (CNs) originate from collections of neurons (nuclei) located within the brainstem (exceptions to this rule are CNs I and II, which are associated with the forebrain and diencephalon, respectively). CNs III and IV emerge from the midbrain portion of the brainstem, CNs V–VIII arise from the pons, and the remaining cranial nerves (CNs IX–XII) originate in the medulla. Taken together, the cranial nerves convey both motor and sensory innervation. However, individual cranial nerves may transmit sensory information only (CNs I, II, and VIII), motor innervation only (CNs III, IV, VI, XI, and XII), or they may be considered mixed nerves, meaning that they carry a combination of motor and sensory fibers (CNs V, VII, IX, and X). As a general rule, the brainstem nuclei associated with sensory cranial nerve fibers are located more laterally in the brainstem, while the motor nuclei are positioned more medially. The sulcus limitans is the landmark that demarcates the boundary between these efferent (motor) and afferent (sensory) nuclear zones in the brainstem, which are typically arranged in a longitudinal columnar manner according to functional components (see Functional Components below). Motor neurons that comprise these brainstem nuclei send axonal projections, via cranial nerves, to control glandular tissue secretions and the contraction of various types of muscle (skeletal, smooth, and cardiac muscle). Conversely, the sensory cranial nerve fibers typically originate from sensory neurons located in sensory ganglia (collections of neurons residing outside of the brain) and function to transmit sensation from various types of sensory receptors (i.e., pain and temperature receptors on the skin) to the brainstem.

Functional Components. In addition to the generalized classification of cranial nerves as sensory, motor, or mixed nerves, the fibers that comprise each CN can be further categorized according to the specific nature of the afferent or efferent information being transmitted and the types of structures innervated. These fiber classifications, which are often referred to as functional components include somatic motor, visceral motor, branchial motor, somatic sensory, visceral sensory, and special sensory fibers. A cranial nerve can carry one or several of these functional components.

Motor Cranial Nerves. Three of the six functional classifications of cranial nerve fibers convey motor innervation: somatic motor, branchial motor, and visceral motor fibers. Somatic motor fibers originate in motor nuclei located in the medial-most cell column of the brainstem and function to transmit motor impulses to voluntary skeletal muscle (of developmental somatic myotome origin) in the head and neck. CNs III, IV, and VI carry somatic motor fibers that innervate the extraocular muscles, CNXI provides somatic motor innervation to two muscles located within the neck/shoulder region and CN XII conveys somatic motor information to the intrinsic muscles of the tongue.

Branchial motor fibers are similar to somatic motor fibers in that they provide motor innervation to voluntary striated muscles located within the head and neck region. However, branchial motor fibers and the muscles that they innervate are afforded a separate classification based on their embryologic derivation from branchial/

Cranial Nerves. Table 1 List of cranial nerves

Number	Name	Functional component[a]	Function	Deficits/symptoms of dysfunction
I	Olfactory N.	Special sensory	Olfaction (smell)	Hyposmia and anosmia
II	Optic N.	Special sensory	Vision	Anopsia
III	Oculomotor N.	Somatic motor	Extraocular muscles (superior, inferior, and medial rectus muscles)	Diplopia and ptosis
		Visceral motor	Ciliary and pupillary constrictor muscles	Pupil dilation
IV	Trochlear N.	Somatic motor	Extraocular muscle (superior oblique muscle)	Diplopia
V	Trigeminal N.	Somatic sensory	Face, mouth/jaw, teeth, sinuses, meninges, oral and nasal mucosa	Trigeminal neuralgia
		Branchial motor	Muscles of mastication	Asymmetric chewing
VI	Abducens N.	Somatic motor	Extraocular muscle (lateral rectus muscle)	Diplopia and medial deviation of eye
VII	Facial N.	Special sensory	Taste (anterior 2/3 of tongue)	Loss of taste
		Somatic sensory	Skin on ear and tympanic membrane	
		Branchial motor	Muscles of facial expression	Facial paralysis and Bell's palsy
		Visceral motor	All salivary glands (excluding the parotid gland) and lacrimal glands	
VIII	Vestibulocochlear N.	Special sensory	Audition and balance	Deafness, tinnitus, and vertigo
IX	Glossopharyngeal N.	Special sensory	Taste (posterior 1/3 of tongue)	Loss of taste
		Somatic sensory	Skin on ear, tympanic membrane, posterior 1/3 of tongue, and tonsillar fossa	
		Branchial motor	Stylopharyngeus muscle	
		Visceral motor	Parotid gland	
		Visceral sensory	Carotid body and carotid sinus	Blood pressure regulation deficits
X	Vagus N.	Special sensory	Taste (epiglottis)	Loss of taste
		General sensory	Skin on external ear	
		Branchial motor	Muscles of the larynx and pharynx	Dysphagia and dysphonia
		Visceral motor	Glands, smooth and cardiac muscle in the neck, thorax, and abdomen	
		Visceral sensory	Pharynx, larynx, and thoracic and abdominal viscera	
XI	Spinal Accessory N.	Somatic motor	Trapezius and sternocleidomastoid muscles	Shoulder drop and weakened neck rotation
XII	Hypoglossal N.	Somatic motor	Muscles of the tongue	Tongue deviation

[a]Alternative names for the functional components include following: General somatic efferent (GSE), somatic motor; general visceral efferent (GVE), visceral motor; special visceral efferent (SVE), branchial motor; general somatic afferent (GSA), somatic sensory; general visceral afferent (GVA), visceral sensory; special somatic afferent (SSA) and special visceral afferent (SVA), special sensory.

pharyngeal arches and the fact that branchial motor nuclei are located in distinct brainstem columns (immediately adjacent and lateral to the somatic motor nuclei). Muscles of branchial origin include the muscles of facial expression (innervated by CN VII), the pharyngeal and laryngeal muscles (innervated by CNs IX, X and the cranial portion of CN XI), and the muscles of mastication (innervated by CN V).

Visceral motor fibers provide autonomic (parasympathetic) innervation to the head, neck, thoracic, and abdominal viscera, where they control glandular secretions and smooth and cardiac muscle contraction. The motor neurons that regulate parasympathetic visceral motor processes are typically positioned immediately lateral to the branchial motor nuclei column in the brainstem. Visceral motor fibers in CN III transmit parasympathetic innervation to structures in the eye that regulate pupil constriction and lens accommodation. CN VII regulates the secretion of tears (via the lacrimal glands) and salivary gland secretions (along with CN IX). CN X conveys visceral motor innervation to glandular tissue, smooth and cardiac muscles of the gastrointestinal, pulmonary, and cardiovascular systems. CN X has the most extensive distribution of the cranial nerves, with its innervation spanning structures within the head and neck down to the thoracic and abdominal regions.

Sensory Cranial Nerves. The remaining three functional classifications of cranial nerve fibers convey sensory information and include somatic sensory, visceral sensory, and special sensory fibers. As previously mentioned, the brainstem nuclei associated with these sensory fibers are located more laterally in the brainstem relative to motor nuclei and are arranged in longitudinal columns according to functional components (from lateral to medial: special sensory, somatic sensory, and visceral sensory nuclei). Somatic sensory fibers carry information from exteroreceptors and proprioceptors in the skin, muscles, tendons, and joints of the head and neck, mediating the perception of pain, temperature, touch, and proprioception. CN V is the major somatic sensory nerve of the head, mediating cutaneous and proprioceptive sensation from the skin, muscles, and joints in the face, mouth, and jaw as well as sensory innervation of the teeth, oral and nasal mucosa, sinuses, and meninges. CN IX also transmits somatic sensory information from a portion of the oral mucosa (the posterior third of the tongue and tonsillar fossa) and, together with CNs VII and X, mediates sensation of the skin on the outer ear.

In contrast to somatic sensory fibers, visceral sensory fibers receive sensory input from receptive endings in visceral structures, such as walls of blood vessels or of the digestive tract. Congruent with its expansive distribution, CN X mediates the majority of visceral sensation in the pharynx, larynx, thoracic, and abdominal cavities. CN IX transmits visceral sensory information from the carotid sinus and carotid body, important structures in the regulation of blood pressure and respiration.

The final category of sensory cranial nerve fibers is the special sensory fibers, which convey sensory information relating to olfaction (CN I), vision (CN II), audition (CN VIII), balance (CN VIII), and taste (CNs VII, IX, and X). This special sensory designation is an all-encompassing classification in that it does not distinguish between the somatic senses (vision, audition, and balance) and the more visceral sensations (olfaction and taste).

Intracranial Courses. Cranial nerves must traverse through foramina (small holes) within the skull in order to navigate the path between the brain and the various structures to which they provide innervation. Often, cranial nerves will travel through these foramina together in groups as they exit the cranium. For example, CNs IX, X, and XI pass through the jugular foramen on their descent to structures in the neck, thoracic, and abdominal cavities; while CNs III, IV, and VI all traverse the superior orbital fissure to enter into the orbit to innervate the extraocular muscles. Knowledge of the origins of cranial nerves (i.e., brain stem nuclei), their intracranial course and the cranial foramina through which they pass is crucial to any neurological exam, as the diagnosis of dysfunction of specific nerves can help to pinpoint the site of and provide valuable information about damage or injury to the brain. For example, one of the early symptoms of a pituitary adenoma is impaired vision, which results from the close proximity between the pituitary gland and fibers of CN II, which can become compressed as a result of the tumor bulk. Similarly, CN VI has a very long intracranial course and, due to its emergence near the bottom of the brain and its course through the cavernous sinus, it is often the first cranial nerve to be affected in the case of elevated intracranial pressure, common symptoms of which include painful eye movement and blurred or double vision (diplopia). Unlike the majority of cranial nerves, which exit from the ventral surface of the brainstem, CN IV exits the midbrain dorsally and wraps around the lateral surface of the brainstem to enter the orbit. Due to this long peripheral course around the brainstem, CN IV is particularly susceptible to head trauma, where damage to this nerve is manifested by diplopia or blurred vision. Such examples demonstrate the manner in which cranial nerve dysfunction can provide insight into the various pathological situations that can occur in the brain.

Cranial Nerve Dysfunction. Cranial nerve dysfunction is not uncommon and can result from a variety of underlying pathologies, ranging from brain trauma to various forms of neurological disease. Common cranial nerve dysfunctions/disorders include Trigeminal Neuralgia (CN V), Bell's Palsy (CN VII), Ramsay Hunt Syndrome (CN VII), acoustic neuroma (CN VIII), Glossopharyngeal Neuralgia (CN IX), eye movement-related cranial nerve

palsies (CNs III, IV, and VI), hyposmia/anosmia (CN I), and various anopsias (CN II), among others. Due to its expansive innervation, CN X damage/dysfunction can result in a variety of deficits relating to visceral processes in the heart, lungs, and abdomen. Interestingly, CN X nerve stimulation is an emerging adjunctive treatment for certain types of intractable epilepsy and refractory (treatment-resistant) depression; however, the manner in which CN X stimulation exerts its therapeutic effects is yet to be fully established.

Cross References

- ▶ Acoustic Neuroma
- ▶ Anosmia
- ▶ Auditory Pathway
- ▶ Auditory System
- ▶ Autonomic Nervous System
- ▶ Bell's Palsy
- ▶ Deaf/Hearing Impairment
- ▶ Diplopia
- ▶ Dysphagia
- ▶ Dysphonia
- ▶ Medulla
- ▶ Midbrain
- ▶ Neurologic Examination
- ▶ Olfaction
- ▶ Olfactory Bulb
- ▶ Optic Nerve
- ▶ Optic Neuropathy
- ▶ Pons
- ▶ Ptosis
- ▶ Pupillary Light Response
- ▶ Taste
- ▶ Tinnitus
- ▶ Trochlear Nerve
- ▶ Vestibulocochlear Nerve
- ▶ Visual Field Deficit
- ▶ Visual System
- ▶ Vertigo

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Cranial Pneumocyst

▶ Pneumocephalus

Craniectomy

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Synonyms

Decompressive craniectomy

Definition

Craniectomy or decompressive craniectomy is a surgical procedure in which a section of the skull is removed and not immediately replaced (Hutchinson, Timofeev, & Kirkpatrick, 2007; Aarabi, Hesdorffer, Ahn, Aresco, Scalea, & Eisenberg, 2006). This procedure is most frequently used when increased intracranial pressure following traumatic brain injury does not respond to other less aggressive interventions. Following brain trauma, the brain may expand within the skull. The resulting increased intracranial pressure can compromise brain function, particularly in the brain stem. Compression of the brain stem can compromise its basic life support functions, that is, cardiac and respiratory regulation, creating a life-threatening situation. By removing part of the skull, the swelling brain is provided room to expand, reducing intracranial pressure and pressure on the brainstem. Although the section of the skull that is removed in a craniectomy is not immediately replaced, the bone removed may be stored and replaced at a later date when brain swelling is reduced and stable. Artificial materials may also be used to replace the removed skull. In a *craniotomy,* a section of the skull is removed and replaced as part of the initial surgical procedure. Craniotomy is more frequently performed as part of surgical intervention for disorders such as brain tumor or arteriovenous malformation. However, a craniectomy may be preferred in such cases if the condition appears to be associated with brain swelling. Craniectomy may have no advantage over craniotomy in long-term outcome after severe brain injury (Woertgen, Rothoerl, Schebesch, & Albert, 2006).

However, standard craniectomy appears to result in better outcomes than limited craniectomy (Jiang et al., 2005).

Cross References

- ▶ Brain Swelling
- ▶ Craniotomy

References and Readings

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Craniopharyngioma

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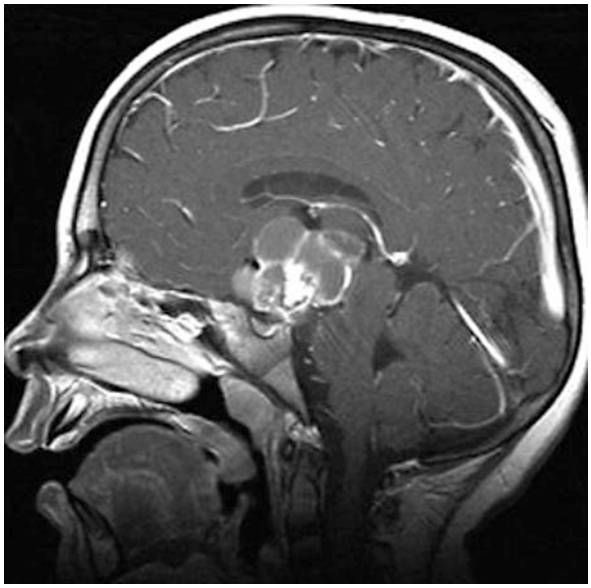
Definition

Craniopharyngioma is a slow-growing, extra-axial, epithelial-squamous, calcified cystic tumor. It occupies the suprasellar/sellar region and shows benign histology but malignant behavior, as it may invade surrounding areas and recur after the treatment (Fahlbusch, Honegger, Paulus, Huk, & Buchfelder, 1999). Craniopharyngiomas may develop embryogenetically, arising from remnants of the craniopharyngeal duct and/or Rathke cleft or metaplastically because of residual squamous epithelium. The most common presenting symptoms are endocrine dysfunction, headache, and visual disturbances. Craniopharyngiomas are treated with surgery or surgery followed by radiotherapy.



Craniopharyngioma. Figure 1 Courtesy Michael Fisher, MD, Peter C. Phillips, MD. The Children's Hospital of Philadelphia

References and Readings

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Cranioplasty

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Definition

Cranioplasty or replacement of bone flap or prosthesis is a surgical procedure usually performed to fill in, or replace a defect in the skull following a craniectomy or removal of a bone flap (Fig. 1).

Further Reading

Cranioplasty can be performed using the patients own bone flap, which has been frozen or stored in the patient's abdomen, or multiple materials such as titanium mesh, hydoxyapatite, and polymethylmethacrylate can be used

Cranioplasty. Figure 1 Source: Winder, Cooke, Gray, Fannin, and Fegan (1999)

as alternative to create a cranioplasty flap, where necessary computerized techniques may be used to generate a custom made cranioplasty (Long, 2007).

The decision to perform and the timing of the cranioplasty to replace the skull defect following craniectomy can be variable. Originally considered to be largely a cosmetic rather than a therapeutic procedure, cranioplasty may be now be performed to prevent late neurological complications associated and identified with craniectomy including the so called "syndrome of trephined" (Dujovny, Agner, & Aviles, 1999; Long, 2007). Characteristics of this syndrome include headache, dizziness, irritability, epilepsy, discomfort, and psychiatric symptoms, and in addition, the skin overlying the skull defect may become indented (Dujovny, Aviles, Fernandez, & Charbel, 1997). The pathophysiology of the "the syndrome of the trephined" is unknown, however, a number of factors have been implicated including atmospheric pressure, cerebral blood flow, and cerebrospinal fluid changes (Dujovny et al., 1997). Neurological and functional improvements have been shown to improve following cranioplasty in this syndrome, for example, in 1945 Gardner described the improvement in symptoms in some patients following cranioplasty with tantalum (Dujovny et al., 1997; Gardner, 1945).

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Craniospinal Irradiation

▶ Craniospinal Radiotherapy

Craniospinal Radiotherapy

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Synonyms

Craniospinal irradiation; CSI

Definition

Craniospinal radiotherapy is an irradiation that is directed at the whole brain and length of the spinal axis, including the meninges, as part of the cancer treatment to control malignant cells. It serves as a radical (curative) antineoplastic therapy, as a prophylaxis against a neoplasm's involvement with the central nervous system, or as a palliative recourse when cure is impossible. Craniospinal irradiation (CSI) is technically challenging, and is used with computed tomography (CT) simulation and multimodality MRI registration to define a large target volume, which spares healthy tissues, and assures exact reproducibility of treatment from day-to-day.

MRI evidence of the craniospinal radiation injury to the brain has been seen in l'Hermitte's sign (a side effect of radiotherapy on the spinal cord, experienced as shock sensations), telangiectasia (dilated capillaries), white matter changes, basal ganglia change, necrosis, and cerebral atrophy. Although the differential sensitivity of specific brain regions to radiotherapy has not been determined,

factors relating to total dose, dose per fraction, and interval between fractions have been identified as important variables influencing the brain's response to radiation. Present research focuses on the development of treatment protocols based on the efficacy in tumor control while using the least dose of craniospinal radiotherapy (1,800 cGy), often in conjunction with chemotherapy, as the efficacy of CSI dose reduction in ameliorating neuroendocrine and neurocognitive sequelae remains unclear. Today, in contrast to the much higher doses used in the past decades, CSI doses of 2,400 and 3,600 cGy (with daily fractions of 150 or 180 cGy) are standard. Studies continue to evaluate how low a dose will remain effective in a risk-adapted setting. Controversy also exists on the expression of radiation effects on specific neurocognitive domains. Attention and memory are known to bear a vulnerability to neurotoxicity, but issues of individual differences, including premorbid and disease-related risk factors, are expected to influence neuropsychological outcomes.

Cross References

- ▶ Radiation Injury
- ▶ Radiation Oncology
- ▶ Radiotherapy

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Craniotomy

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Synonyms

Craniectomy; Trephination

Definition

Neurosurgical procedure involving the opening of the skull as a means of decreasing intracranial pressure and/or for purposes of removal of a mass lesion.

Current Knowledge

Craniotomy as a treatment for increased ICP from a mass lesion has its foundation early in the history of neurosurgery. Decompressive craniotomy (DC) initially was introduced to lower the intracranial pressure (ICP) in patients with inoperable tumors and in managing uncontrolled ICP after traumatic brain injury (Brit & Hamilton, 1978). In recent years, DC has been recommended as an alternative treatment for space occupying acute hemispheric infarction with or without massive medically uncontrolled brain edema (Schwab, 1998). During the acute period following cerebral infarction, neurologic decline is often attributed to surrounding edema. Apart from relieving the mass effect, restoration of the microcirculation around the infarcted area is the target of DC. The management of increased intracranial pressure is a common clinical scenario in neurosurgery. Strategies for the management of ICP fall into two general categories: to reduce the volume of the intracranial compartment (medical management) and to remove the mechanical constraints imposed by the cranial vault (surgical).

In patients who sustain a severe non-penetrating head injury, overall 25–45% require a craniotomy for evacuation of a hemorrhagic mass lesion, including epidural, subdural, and intracerebral hematomas (Miller, 1981). There is little debate in the surgical management of a rapidly deteriorating patient with a focal neurological deficit and neuroimaging findings of an expanding intracranial hematoma associated with significant mass effect and midline shift. For less obvious situations controversy remains given the lack of class I and II data to support any treatment standard. Complete removal of a brain tumor without inflicting neurological deficits is a desirable end result in neurosurgical practice. Craniotomy was tailored to encompass tumor plus adjacent areas presumed to contain eloquent cortex. Magnetic brain stimulation or intraoperative cortical stimulation can be used to guide resection of functional cortex.

DC remains a controversial procedure in spite of a number of studies published in the literature on its use in the treatment of intracranial hypertension secondary to malignant cerebral edema, traumatic brain injury, aneurysmal subarachnoid hemorrhage, central venous

thrombosis, encephalitis, intracerebral hematoma, and metabolic encephalopathies. It has been shown to be effective in reduction of ICP refractory to medical therapy but evidence supporting an improvement in clinical outcome has not been conclusive.

Cross References

► Temporal Lobectomy

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Cretinism

► Hypothyroidism

Creutzfeldt-Jakob Disease

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Synonyms

CJD; Prion disease; Transmissible spongiform encephalopathy (TSE)

Short Description or Definition

Creutzfeldt–Jakob Disease (CJD) is a rare, fatal neurodegenerative disease, which is one of the transmissible spongiform encephalopathies or prion diseases. These conditions are characterized pathologically by neuronal loss, spongiform change, and astrocytic gliosis. Cell loss can be seen microscopically as multiple perforations to the brain tissue creating the characteristic "sponge-like" appearance. Prion diseases are caused by infectious agents, which are abnormal self-replicating forms of a normal brain protein, prion protein.

Categorization and Epidemiology

Creutzfeldt–Jakob Disease (CJD) may be sporadic (that is develop spontaneously without apparent cause), familial (inherited), or acquired (transmitted by infection).

CJD occurs worldwide with a mean annual incidence of approximately 1–2 cases per million population (Ladogana et al., 2005). Except for variant and iatrogenic CJDs, which are in decline, the disease has a relatively stable incidence with no convincing evidence of geographical clustering (although there are regions with an increased incidence of familial cases). The gender incidence is equal.

Natural History, Prognostic Factors, Outcomes

Historical Background

Sporadic and familial CJDs have been recognized as prion diseases for many years, showing wide geographical spread; however, variant CJD has been confined largely to the United Kingdom, with the first cases reported in 1996. It is thought that meat products intended for human consumption contained contaminated brain and spinal cord from animals affected by the epidemic of a prion disease in cattle (Bovine Spongiform Encephalopathy) in the 1980s (Hilton, 2006). Initial fears of a catastrophic epidemic of vCJD among humans have been partially quelled, with a peak in new cases during 2000 reducing steadily to only one new case in 2007 (http://www.cjd.ed.ac.uk). However, all vCJD cases to date have shared a common genotype (Methionine/Methionine) raising the possibility of subsequent peaks, which occur due to lengthened incubation times among other genotypic groups. In addition, evidence from Kuru, an acquired prion disease arising from cannibalistic funeral practices among the Fore people of Papua New Guinea carried out until 1950s, suggests that incubation times for acquired prion disease may stretch to several decades (Collinge et al., 2006). It is

also possible for a person-to-person spread to occur, as has been seen in four cases of blood transfusion-associated vCJD infection (most recent incidence figures are available from http://www.cjd.ed.ac.uk).

Natural History and Prognosis

Different CJD phenotypes develop and progress at different rates, although all subtypes develop profound dementia and multiple neurological features progressing to loss of awareness and death. A summary of clinical presenting features is given in Table 1.

sCJD presents with cumulative multifocal neurological deficits in association with a rapidly progressive dementia. The cardinal clinical signs are dementia and myoclonus, with a significant proportion of cases exhibiting ataxia and paratonic rigidity of the limbs. The mean survival from onset to death is only 4 months, although patients in the younger age groups often survive for more than a year. sCJD affects predominantly the older age groups with the mean age being 65 years at death.

The clinical presentation in familial CJD is often similar to sCJD, but the age of onset is about 10 years earlier, and in some forms, there may be early ataxia and/or slow progression.

Iatrogenic CJD may present as in sCJD, but human pituitary hormone recipients typically develop progressive ataxia and cognitive impairment develops late, if at all.

Variant CJD presents with a psychiatric syndrome, including depression and anxiety, for about 6 months before there is a progressive neurological and cognitive decline as in sCJD, although chorea and dystonia occur as well as myoclonus. The mean survival is 14 months, and vCJD affects a younger age group, with the mean age being 29 years at death.

Neuropsychology and Psychology of Creutzfeldt–Jakob Disease

The Presentation of the CJD Patient

The differential diagnosis is often the question at referral, in particular, the distinction between a psychiatric or neurological basis for the presenting symptoms or to distinguish CJD from other neurological conditions. A thorough history, as always, is essential in establishing both the profile of the presenting symptoms and the course of the illness, and care should be taken to obtain corroborative reporting from relatives given the difficulties of accurate history taking in individuals with cognitive decline. Sporadic CJD can often be distinguished from other disorders by the speed and degree of cognitive decline, the short duration of illness, and the associated neurological signs. Some cases of fCJD present very similarly to sCJD; however, in fCJD, there is often a younger age of onset, a longer disease duration,

Creutzfeldt-Jakob Disease. Table 1 Forms, causes, and incidence of CJD

Form	Phenotype	Cause	Incidence
Sporadic	Sporadic Creutzfeldt–Jakob Disease (sCJD)	Unknown.	Approximately one case per million population. Accounts for around 85% of CJD cases.
Familial	Familial Creutzfeldt–Jakob Disease (fCJD)	Inheritance of mutation in the PrP gene.	Approximately 10–15% of CJD cases are familial.
Acquired	Iatrogenic	Case-to-case transmission via contaminated neurosurgical instruments, human dura mater grafts, or exposure to human pituitary hormones. The variant form of CJD can also be transmitted via blood transfusion.	Less than 1% of CJD cases arise through acquired infection.
	Variant CJD (vCJD)	Ingestion of contaminated meat products from cattle infected with Bovine Spongiform Encephalopathy.	166 cases in total in the UK, 23 cases in France, and a total of 18 cases elsewhere in the world (as of May 2008).

Creutzfeldt-Jakob Disease. Table 2 Clinical presentation of CJD

Form	Age at onset	Approximate duration of illness	Early clinical features	Neuropsychological findings	Exceptions
Sporadic	90% of cases between 50 and 80 years, mean 65 years	4 months (65% of cases survive <6 months from symptom onset).	Neurological signs including cerebellar ataxia, cognitive impairment (global dementia or specific deficits in earlier stages), followed by myoclonus, rigidity, and rapid deterioration to loss of speech, voluntary movement, and awareness.	Areas of early cognitive deficit vary in this heterogeneous group, but there is often marked impairment by the time of a referral for testing. Most cases show rapid progression to a wide-ranging dementia including verbal and nonverbal memory, executive dysfunction, and nominal skills. Poor memory and/or attention are often early features, but there may be occasional cases where initial deficits reflect dysphasia or visual disturbance.	Rare cases occur younger than 50. Young cases may present with some psychiatric symptoms initially, similar to the vCJD presentation, and may have longer disease duration.
					19% sCJD cases have a disease duration >12 months
					Heidenhain form: very focal visual difficulties for weeks/months before other cognitive symptoms.
				Look for: Rapid cognitive decline over multiple test sessions.	Brownell–Oppenheimer form: pure cerebellar syndrome for several weeks or months before cognitive decline
				Fluctuations in responsiveness and distractibility.	
				Intrusion errors.	
				Verbal and motor perseveration.	
Familial	Dependent on the specific mutation of the prion protein gene, but most often between the ages of 30 and 50 years.	Dependent on the specific mutation, but on average 2–5 years	Dependent on mutation, may present in a similar fashion to sCJD or with predominant sleep and autonomic disturbance or cerebellar ataxia (see Exceptions, right). Deterioration is usually slower than in vCJD or sCJD with a longer disease course.	fCJD cases may demonstrate less severe/rapid cognitive decline at the early stages of a longer disease course than sCJD or vCJD cases. A single case in 2000 showed specific isolated deficits in delayed verbal memory and word finding prior to global involvement. One study suggests that naming ability may be preserved in some cases in comparison to sCJD and vCJD. Look for: Family history of CJD or other (possibly misclassified) neurological disease	Some cases can have an illness duration of years.
					Fatal Familial Insomnia: early sleep disturbance and autonomic dysfunction is prominent
					Gerstmann Straussler syndrome: progressive cerebellar ataxia

Creutzfeldt-Jakob Disease. Table 2 (Continued)

Form	Age at onset	Approximate duration of illness	Early clinical features	Neuropsychological findings	Exceptions
Iatrogenic	Dependent on age at exposure. Incubation period following intracerebral exposure is 19–46 months, extending to many years or decades with peripheral exposure.	Following dura mater graft similar to sCJD, With human pituitary hormones 12–18 months	Human growth hormone infection: progressive cerebellar syndrome, delayed onset of dementia Human dura mater infection: rapidly progressive dementia, similar to sCJD	Dependent on mode of transmission; human growth hormone patients present with ataxia with relatively preserved cognitive function until later stages, while intracerebral cases present with broad-ranging dementia with rapid deterioration as seen in sCJD. Look for: Rapid cognitive decline over multiple test sessions for intracerebral exposure cases. History of relevant exposure to differentiate from sCJD.	
Variant	Median age 28 (range 12–74)	Median 14 months	Most commonly, initial presentation is psychiatric disturbance including depression, agitation, and behavioral changes, although in some cases cognitive changes may be the first sign of abnormality. A delay of months is possible before distinct neurological signs, although cognitive changes may be found earlier in the disease course. Sensory symptoms such as pain or odd sensation in limbs or face may be reported. Ataxia, myoclonus, and significant cognitive impairment (such as memory) develop as the disease progresses	Measurable impairments on tests of both verbal and nonverbal memory, executive function, speed of attention, and nominal skills have characterized descriptions of published cases. Language, verbal reasoning, and visuoperceptual skills may be less frequently impaired, although this may reflect an earlier disease stage at the time of testing. One study suggests possible preserved ability in some components of visuoperception in comparison to patients suffering from sCJD or fCJD. Global involvement follows with rapid disease progression. Look for: Fluctuating attention and effort during testing. Cognitive impairment more profound than expected for depression or including areas of deficit unusual for psychiatric disorders.	In 15%, neurological symptoms precede psychiatric.
				Significant cognitive decline over follow-up assessment sessions.	

Sources of information for this table are referenced under ''References and Readings''.

Creutzfeldt-Jakob Disease. Table 3 Investigations used in the diagnosis of CJD

Phenotype	MRI	EEG	CSF 14–3–3	Tonsil biopsy	Blood test	History
Sporadic (sCJD)	Important in excluding other conditions. High signal on FLAIR or DWI sequences in the caudate and putamen in about 70% of cases	In 60–80% of cases, generalized bi- or triphasic periodic sharp wave complexes at 1/s. May not appear until later stages of the disease	A positive 14-3-3 CSF immunoassay is strongly supportive of a diagnosis of sCJD in the appropriate clinical context i.e., rapidly progressive dementia. Positive in 90% of cases of sCJD	Negative	At codon 129 of the prion protein gene: 70% methionine homozygous	
Familial (fCJD)	Some cases similar to sCJD	Characteristic periodic pattern is less frequently seen than in sCJD.	Positive less frequently than sCJD	Negative	Analysis of the prion protein gene for mutations	Positive family history of CJD in about 30% of cases
Iatrogenic	Similar to sporadic CJD	Characteristic periodic pattern is less frequently seen than in sCJD.	Positive in a proportion of cases	Negative	Mainly homozygous methionine or valine	Relevant exposure risk such as treatment with cadaveric derived human growth hormone or human dura mater graft
Variant (vCJD)	Characteristic high signal in the posterior thalamic region (the "pulvinar sign") in over 90% of cases on FLAIR or DWI sequences.	Normal or nonspecifically abnormal. Characteristic periodic pattern in two cases late in clinical course.	Positive in 50% of cases, but does not distinguish from sCJD	As vCJD, unlike the other CJD phenotypes, involves the lymphoreticular system, abnormal protein may be found in the biopsy of tonsil tissue in about 90% of cases.	All tested cases methionine homozygous	

Adapted from The National Creutzfeldt–Jakob Disease Surveillance Unit website (http://www.cjd.ed.ac.uk/investigations.htm).

and a family history of either CJD or another neurological disorder. In iatrogenic cases, there should be a clear history of a relevant exposure. The history in vCJD cases includes early psychiatric symptoms (including mood, delusion, and agitation) and may also reveal cognitive decline in everyday activities, of the type that might typically be attributed to depression. In some cases, neuropsychological symptoms may precede psychiatric or neurological indicators that develop as the disease progresses. The early presenting features in all CJD subtypes are summarized in Table 2.

Neuropsychological Testing

Patients with CJD often present at too late a stage for formal testing with a full neuropsychological battery. This is reflected in the literature, in which reports focus on small cohorts and case studies. Efforts should be made to obtain sufficient breadth across cognitive domains when testing to aid diagnosis and enable repeat testing if appropriate. Observations of test behavior will also be helpful. A study is currently underway to establish whether a brief bedside screening test might be sufficient to give an

indication of whether the degree or pattern of impairment seen in a patient could indicate CJD.

Evaluation

Neuropsychological testing may occur prior to, in parallel with, or following medical investigations and may provide support for a diagnosis of CJD or prompt the clinician to instigate more extensive investigation than would usually be undertaken in patients presenting, for example, with primarily psychiatric complaints as in vCJD. A number of further investigations may be used in the diagnosis of CJD (Table 3).

Treatment

CJD is fatal. There is currently no effective treatment for the disease itself, despite ongoing trials of Quinacrine, Pentosan Polysulfate, and Flupirtine (Stewart, Rydzewska, Keogh, & Knight, 2008; see the MRC New Therapies Scrutiny Group for Prion Disease website for up-to-date information concerning recent treatment studies). Medical management should focus on alleviating discomfort, including the use of medication to manage myoclonic jerks or pain and, as the disease progresses, the management of issues such as feeding or continence. Intervention may be needed for the management of mood or psychotic symptoms. Since a high level of care will inevitably become necessary, planning for the provision of this should begin early, in consultation with the family. In the case of a diagnosis of familial CJD, the family will face difficult decisions regarding genetic screening and should be guided through such a process by an appropriately qualified professional.

It is often the case that the cognitive symptoms of CJD show extremely rapid deterioration and, in view of this, cognitive rehabilitative efforts are unlikely to produce helpful returns. However, in cases with early referral or a longer disease course, supportive aids (such as a calendar for orientation) or environmental adaptations may produce improvements in activities of daily living, self-efficacy, and mood, at least in the early days, as is the case in other dementias (Clare, 2007; Smith-Bathgate, 2005).

While the patients themselves are likely to lose awareness of their predicament as their cognitive ability declines, their families observe a devastating deterioration in their loved ones. There is a role for the clinical psychologist, nurse practitioner, or other qualified healthcare professionals in supporting individuals and their families through a period of acceptance, adjustment, loss, and ultimately grief. Provision might also be made for the counselling of healthcare staff involved in these distressing cases.

Cross References

- ▶ Prion Disease
- ▶ Spongiform Encephalopathy

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Criminal Forensics

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Definition

Within the field of forensic psychology, the utilization of clinical neuropsychological expertise for criminal forensic cases can be considered a subspecialty of the field. Denney and Wynkoop (2000) modified Mrad's (1996) multiple data source model (MDSM) to the practice of criminal forensic neuropsychology. The purpose of the model is to provide a framework for clinicians to evaluate all relevant sources of information, most notably information relevant to the defendant's mental state at the time of the offense. The model covers three time points of analysis: present, time of offense, and prior history. Moreover, the model assesses symptoms/behaviors, explanations, etc. via the self-report of the defendant as well as via other sources of data (e.g., neuropsychological tests, mental status exam, medical/neurological exam, arrest reports, witness statements, physical evidence, hospital/psychiatric records, employment records, family/friend reports, etc.). Once all of the relevant pieces of information are gathered, spanning the three time points, it is the role of the forensic neuropsychologist to consolidate the information and formulate opinions. Evaluators involved in criminal forensics typically have very different roles compared to general practitioners. Specifically, in forensic evaluations, the client is typically not the person being examined, and the ultimate goal is to *evaluate the facts*, not to maintain an alliance with the examinee as is the case in a clinical context. Moreover, forensic criminal evaluations typically involve much more extensive application of corroborative information as well as validated assessments of negative response bias, symptom validity, and malingering.

Cross References

► Criminal Litigation

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Criminal Litigation

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Definition

In civil litigation, a lawsuit is filed by a private party, seeking damages from another party as a result of some type of injury, negligence, or malpractice. In criminal litigation, the case is filed by the government against a defendant whom the government believes has committed a crime. Crimes are classified into one of two categories: misdemeanors or felonies. Punishment for misdemeanors involve a maximum possible sentence of less than 1 year of incarceration; felonies carry a maximum possible sentence of more than 1 year of incarceration. The burden of proof in criminal litigation is always assumed by the state. Thus, it is the state's responsibility to prove that the defendant is guilty of having committed a crime. However, if a defendant claims insanity (e.g., cannot appreciate the wrongfulness of the act nor conform their conduct to the requirements of the law), then the burden of proof in proving one's insanity falls on the defendant. Under criminal litigation, the state must demonstrate that the accused satisfied each element of the statutory definition of the crime and prove the defendant's involvement beyond a reasonable doubt. In the context of criminal litigation, forensic neuropsychologists often provide determinations regarding "mens rea" (e.g., guilty mind) or not guilty by reason of insanity (NGRI), competent waiver of *Miranda* rights, and/or competence to proceed (e.g., stand trial, to be sentenced, etc.).

Cross References

► Actus Rea
► Criminal Forensics

► Insanity
► Mens Rea

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Criminal Responsibility

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Definition

Criminal responsibility, or the conclusion of guilt for a criminal offense, centers on four elements: (1) The defendant must have committed the act (*actus reus*). (2) The defendant's actions must have caused the crime. (3) The defendant must have committed the crime with a guilty state of mind (*mens rea*). (4) There must be no circumstance constituting a legal defense for the charged crime (e.g., self-defense). In short, there must be the criminal act and the criminal intent and both must be proven, beyond a reasonable doubt. Mental health professionals are typically involved with establishing intent and mens rea, which involves the assessment and professional opinions related to a defendant's sanity and/or diminished capacity (which includes a decreased level of intent).

Criminal responsibility evaluations are also called sanity evaluations or assessment of mental state at the time of the offense evaluations. Determining whether or not a defendant was sane at the time of the offense is one of the most controversial questions forensic examiners are asked to address given that the purpose of the exam is to identify individuals who should not be held morally responsible ("not guilty by reason of insanity") for their acts (Yates & Denney, 2008). An insanity plea is pursued in about 9 out of 1,000 cases, and it is successful approximately 25% of the time (Wrightsman, Greene, Nietzel, & Fortune, 2002). It is very rare when a defendant is acquitted secondary to insanity caused by a brain-injury-related issue. Nonetheless, neuropsychologists are increasingly being called upon to determine whether or not brain pathology may contribute to criminal behavior (Barr, 2008).

Cross References

► Actus Rea
► Diminished Capacity
► Diminished Responsibility
► Insanity
► Intent
► Mens Rea

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Crisis Intervention

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Synonyms

Emergency mental health treatment

Definition

Crisis intervention techniques target the resolution of an immediate crisis and restoration of the affected individual's previous level of functioning. Roberts (2005) defines a crisis as "a period of psychological disequilibrium, experienced as a result of a hazardous event or situation that constitutes a significant problem that cannot be remedied by using familiar coping strategies."

Current Knowledge

It is important to stress that most individuals who experience a crisis or traumatic event tend to recover on their own accord (i.e., crisis intervention may not be needed).

Crisis Intervention Approaches

Critical incident stress management (CISM; Mitchell & Everly, 1993) is an approach to crisis intervention that has garnered considerable attention since its initial development in the 1980s. A key component of CISM is the critical incident stress debriefing (CISD), a group session held shortly after an incident. Led by CISM-trained facilitators, the CISD session allows participants to share their experiences of the incident, and the leaders provide psychoeducation about stress reactions and recommended coping strategies. In the 1990s, a controversy developed surrounding CISM, as research evidence mounted that failed to show its effectiveness. A National Institute of Mental Health (NIMH) report (2002) suggested that stand-alone CISD does not consistently prevent post-traumatic disorders, and that some individuals, such as those with high arousal, may be put at heightened risk for adverse outcomes as a result of CISD-type interventions. Thus, mandatory participation in group debriefing sessions is considered very questionable.

An alternative to CISM is psychological first aid (PFA; National Child Traumatic Stress Network and National Center for PTSD, 2006). PFA is an evidence-informed, pragmatic approach that targets acute stress reactions and the immediate needs of persons exposed to a critical incident (NIMH, 2002). The goals of PFA include enhancement of safety (both objective and subjective), reduction of stress-related symptoms, restoration of rest and sleep, linkage with resources, and facilitation of social support.

Roberts (2005) developed a seven-stage crisis intervention model:

1. Conduct a biopsychosocial/imminent danger assessment
2. Establish a collaborative relationship
3. Identify the major problems and crisis precipitants
4. Encourage exploration of feelings
5. Generate alternatives and new coping strategies
6. Restore functioning via implementation of an action plan
7. Plan follow-up and booster sessions

Hospitals, clinics, and schools typically have in place a set of policies and procedures for how staff should intervene in the crisis situations that are most likely to be encountered. Staff trainings on these protocols should be conducted routinely to ensure a high level of preparedness.

Cross References

▶ Stress Management

References and Readings

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Criterion Validity

▶ Test Validity

Criterion-Referenced Testing

▶ Domain Referenced Test Interpretation

Critical Periods

► Sensitive Periods

Crossed Aphasia

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Definition

Crossed aphasia is an acquired language impairment following a lesion in the right hemisphere in a right-handed individual.

Current Knowledge

The term "crossed aphasia" (CA) was coined by Byrom Bramwell (1899) to indicate an aphasia caused by a cerebral lesion ipsilateral to the dominant hand regardless of handedness. Currently, CA only refers to right-handed individuals. The frequency of CA among stroke survivors is rare (1–3%).

Although some earlier authors considered CA to be the consequence of a weaker language lateralization, it appears that individuals with CA have language as strongly lateralized as those with left-hemisphere aphasia (LHA), mainly because both populations show a similar prognosis.

CA can be *mirror image* or *anomalous* (Alexander, Fischette, & Fischer, 1989). *Mirror-image* CA denotes the expected correspondence between symptomatology and lesion location within the language-dominant hemisphere, whereas *anomalous* CA implies the presence of unexpected language symptoms given the lesion location (e.g., Wernicke's aphasia following a frontal lesion [Basso, Capitani, Laiacona, & Zanobio, 1985], a mixed transcortical aphasia with preserved naming [Fujii, Yamadori, Fukatsu, Ogawa, & Suzuki, 1997]). Close to two thirds of CA cases are mirror image. The CA language symptomatology is virtually indistinguishable from LHA, and all aphasia types have been reported in CA.

The cause of CA is essentially unknown, but the influence of left-handedness in the family has long been considered an important causal factor, a hypothesis called familial sinistrality. However, familial sinistrality is absent in the majority (63%) of reported CA cases (Coppens, Hungerford, Yamaguchi, & Yamadori, 2002). Further, there does not seem to be any difference in CA symptomatology when comparing patients with and without familial sinistrality, and the presence of familial sinistrality does not increase the number of *anomalous* cases, as could be expected if language laterality were weak (Coppens et al., 2002).

Individuals with CA also often display typical right-hemisphere symptoms (e.g., left-side neglect, visuospatial/visuoconstruction problems, affective dysprosody, and spatial agraphia) which indicates that those skills may lateralize independent of language. The aphasic difficulties may obscure these right-hemisphere signs, but these represent a major difference in symptomatology between CA and LHA.

Cross References

► Aphasia
► Handedness

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Cross-Examination

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Definition

A deposition or actual trial testimony consists of two parts: the direct examination and the cross-examination.

Direct examination precedes the cross-examination and involves testimony brought forth by the retaining attorney. Cross-examination occurs immediately after the direct examination and is carried out by the opposing attorney. The main purpose of cross-examination is to test the "reliability, accuracy and credibility" of witnesses' testimony produced during the direct examination. Questions posed during cross-examination typically fall into two categories: those intended to expose weaknesses or errors in the expert witnesses' data acquisition or interpretations, and those related to expose biases in the testimony. During cross-examination, expert witnesses are expected to give responsive answers. That is, they are to provide relevant answers, but the answers need not be those implicitly desired by the opposing attorney. The opposing attorney may use several tactics during cross examination including: challenging credibility, establishing doubt, leading questions, feigned ignorance, the cut-off (e.g., testimony of witness terminated to stop the witness from providing further information that could be detrimental to the opposing attorney's position), intentional ambiguity, implying impropriety, rattling the witness, and many others.

Cross References

► Direct Examination

References and Readings

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CRS

► Coma Recovery Scale
► Conners Rating Scales

CRS-R

► Coma Recovery Scale

Crystallized Intelligence

► Intelligence

CSI

► Craniospinal Radiotherapy

CS-PFP

► Physical Functional Performance

CS-PFP10

► Physical Functional Performance

CT

► Category Test
► Computed Tomography

CT Scan

► Computed Tomography

Cue

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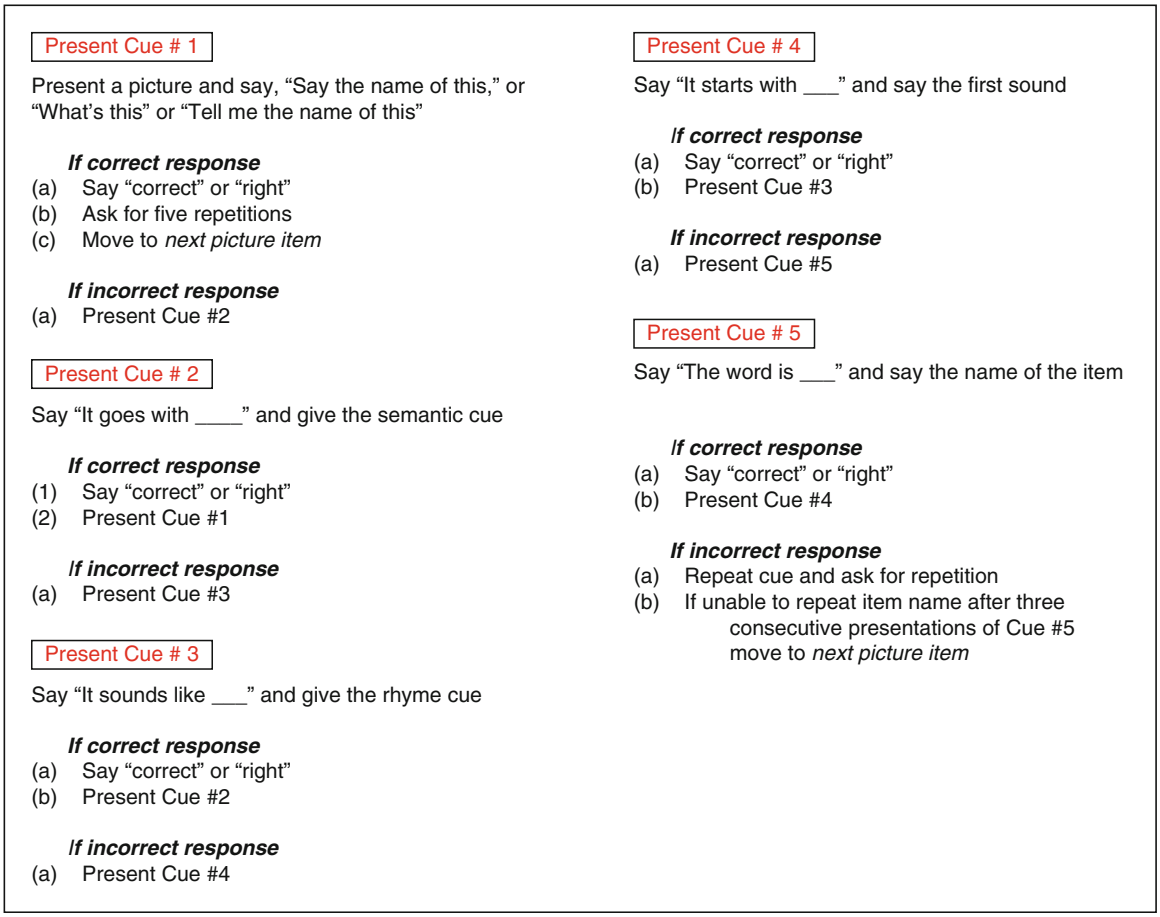
Synonyms

Discriminative stimulus; Prime; Prompt

Definition

A cue is a verbal or nonverbal instruction to induce behavior change. Cues are self-generated or may come from the environment (including an examiner). They may appear spontaneously as a behavior is unfolding in order to effect immediate change. For example, when one sees the cue of a deer running into the road one veers away to avoid a collision, or when one is engaged in conversation and hears the cue of his or her sentence that does not adequately express the intended meaning one immediately changes the sentence. Cues may also appear as a learned strategy and be repeated in a consistent format. For example, drivers know that when the yellow traffic light appears they should slow the car in preparation to stop when the red traffic light appears, or in a communication interaction when another individual offers a greeting one should respond. Some examples of self-generated, internal cues are reminders for future action (e.g., remember to walk the dog), mental sequences used in the tip-of-the-tongue state (e.g., telling oneself that the



Cue. Figure 1 An example of a cueing hierarchy for oral naming using semantic and phonological cues

name rhymes with sock), or teaching/treatment strategies (e.g., using an association strategy for naming, such as reciting the alphabet to cue the name). Examples of external cues are environmental signs (e.g., walk/do not walk signals), preprogrammed reminders (e.g., alarm clock), or teaching/treatment techniques (e.g., semantic-phonological cueing hierarchy). A cueing hierarchy is a set of cues progressing from weak cues providing little information about the target response, to strong cues providing much information (Wambaugh, Linebaugh, Doyle, Martinez, Kalinyak-Fliszar, & Spencer, 2001) (Fig. 1).

Cross References

- ▶ Cue Dominance
- ▶ Cued Recall
- ▶ Free Recall
- ▶ Phonemic Cue
- ▶ Recency Effect
- ▶ Semantic Cue
- ▶ Semantic Fluency
- ▶ Verbal Fluency

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Cue Dominance

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Synonyms

Cue salience; Orienting stimulus; Stimulus strength

Definition

Cue dominance refers to the tendency to perceive or respond to a particular stimulus or class of stimuli over others in the environment. Stimuli may either have intrinsic properties that give them strength or "dominance" based on physical attributes (e.g., loudness, color), or may acquire dominance (i.e., propensity to elicit a response) as a function of associative learning and task demands.

Current Knowledge

Cue dominance is an important principle derived from behavioral studies of animal conditioning and learning theories. It provides a theoretical foundation for the neuroscience of selective attention, linking basic behavioral and learning processes with higher order information processing.

In the context of behavioral conditioning, cues refer to stimuli that have the capacity to elicit an orienting response, anticipation, subsequent attention, and response intention and preparation. Cues lack the inherent biological salience of unconditioned stimuli, and do not elicit unconditioned Pavlovian responses, but can become conditioned stimuli through associative learning. Studies of discrimination learning conducted in the middle of the twentieth century established many of the operational characteristics underlying cue dominance. For example, studies examining intrinsic cue dominance demonstrated that monkeys have a natural preference for response to color over other perceptual dimensions, such as size and shape (Draper, 1965). Experiments with human infants demonstrated cue dominance for certain shapes over others based on whether they resembled the shape of a human face (Fantz & Miranda, 1975). While certain stimulus features have intrinsic cue dominance across many animal species, the ability to form complex cues based on the association of simple cues appears greatest among humans, as is the ability to exhibit reversal learning in order to shift response from one cue to another (Kendler & Ward, 1972; Kendler, 1971).

In neuropsychological studies and assessment methods, cues are often used to either facilitate attention to particular spatial locations, semantic information, or response demands. Alternatively, cues are used to create interference to test the effects of distraction or the redirection of attention away from the primary demands of a task. This is a fundamental element of the spatial selective attention paradigms that involve cueing to spatial locations (Posner, Snyder, & Davidson, 1980). Furthermore, the tendency of the semantic value associated with a word

to be dominant over color and to interfere with attention to and naming of the actual color that is being presented underlies the Stroop effect. Researchers and clinicians studying or assessing attentional processes need to account for both the inherent and acquired cue dominance of stimuli being used in the context of particular tasks.

Cross References

- ▶ Enhancement
- ▶ Selective Attention
- ▶ Visual Discrimination

References and Readings

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Cue Salience

- ▶ Cue Dominance

Cued Recall

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Definition

Cued recall is the retrieval of memory with the help of cues. Such cues are often semantic. Cued recall differs from free recall in that a cue or word is presented that is related to the information being remembered. This aides in the process of memory retrieval. Some examples of cued recalls are the names of the categories in which words were originally grouped or the presentation of related words. For instance, in remembering the word feather, the word bird may be used as a cued recall.

Current Knowledge

Tests of Cued Recall

There are many tests of cued recall. One of the most commonly used tests of cued recall is the California Verbal Learning Test (CVLT) developed by Delis et al. This semantic test, like many other tests of memory, utilizes both free and cued recall.

Clinical Uses of Cued Recall Testing

In addition to the use of free recall tests, tests of cued recall may be used in identifying memory impairments in a wide array of disorders including mild cognitive impairment and Alzheimer's disease. In some instances, tests of cued recall may be more accurate than free recall tests in detecting cognitive changes. For example, Ivanoiu et al. (2005) found that a cued recall test is more reliable in testing memory impairment in Alzheimer's disease than free recall testing. While both tests of memory are impaired in such individuals, cued recall may be a more accurate measure of cognitive decline. This is because deficits other than pure memory deficits, such as impaired attention or depression, may account for a decreased performance in free recall. Conversely, cued recall tests may be used to more directly measure the encoding and retrieval that takes place in memory tasks.

Cross References

- ▶ California Verbal Learning Test (California Verbal Learning Test-II)
- ▶ Cue
- ▶ Free Recall

References and Readings

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Cultural Diversity in Neuropsychology

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Definition

The term "cultural diversity" generally refers to the differences in defining cultural features that exist between people (or within a given population), such as language, dress, and traditions, as well as more abstract concepts involving significant variations in societal organization and interaction styles between individuals and environments. With respect to neuropsychology, the term encompasses the racial and ethnic diversity among neuropsychologists themselves and the populations they interact with, as well as issues related to the influences of race and ethnicity on neuropsychological evaluations and treatments.

Current Knowledge

Multicultural competency is a developing area of focus within the field of neuropsychology. As the ethnic and racial diversity of the United States increases, so do efforts to provide neuropsychological services to ethnic minorities. Thus, clinical and research studies have begun to examine how cultural variables influence performance on neuropsychological measures. While the past decade has witnessed some publications providing test norms for specific racial and ethnic groups, the use of racial and ethnic norms for neuropsychological measures remains a contentious issue. Readers are encouraged to examine the summary of the 2008 Diversity Summit for additional details (Romero et al., 2009). Highlights of the summit and recent literature are provided below.

Recent studies emphasize that the deconstruction of race is critical for improved clinical care of ethnically diverse populations. The construct has been identified as a "proxy for more meaningful but complex variables" such as acculturation and indicators of quality of education, which account for significant proportions of racial and ethnic differences in neuropsychological test scores (Romero et al., 2009, p. 765). However, increasing emphasis has been placed on the importance of the clinician knowing when to apply race- and ethnic-based norms, as their use can actually reduce detection of neuropsychological impairment. The complexity of this issue was demonstrated in one of the Mayo's Older African American Normative Studies (MOAANS) project studies. In this study, adjustment of test scores for reading level surprisingly reduced detection of cognitive impairment in African Americans. Lucas and colleagues (2005) suggest that their findings indicate that cohort differences in reading level may be more reflective of individual differences in cognitive ability rather than contextual differences such as educational backgrounds. While race may be a very efficient and parsimonious variable for clinicians to use at times, the confounding of causes and effects of these variables presents a significant challenge. The Diversity Summit participants unanimously agreed that guidelines for neuropsychological practice among ethic and racial minorities are needed to further improve clinical service for ethic and racial minorities.

In an effort to initiate the development of practical guidelines regarding the use of demographic corrections, clinical criteria have been developed. As detailed in the Diversity Summit proceedings, demographic corrections *are* useful to identify and characterize acquired neurocognitive impairments in adults who (1) are natives of the country of assessment, (2) developed normally, (3) received mainstream education, and (4) speak English as their first language (for the U.S. norms). Demographic corrections *are sometimes* useful to identify and characterize acquired neurocognitive impairments in (1) teenagers or young adults who have not completed their education, (2) adults who may have had a mild developmental disorder, or (3) anyone with a linguistic, cultural, or educational background not well represented in the normative subject sample. Demographic corrections are *not* recommended when the clinician is asked (1) to characterize "absolute" levels of functioning, (2) to identify or characterize possible acquired impairment in capital punishment cases or when determining qualifications for special services, or (3) in cases of possible acquired impairment in individuals who have developmental disability, (4) to characterize acquired cognitive impairment in individuals who

have major background differences from the normative sample, (5) to predict future performance in employment or academic settings, and (6) for employment selection decisions. In addition to these clinical guidelines, emphasis is being increasingly placed on correctly using probabilistic statistics to predict group membership (i.e., diagnosis) for individuals. Readers are encouraged to review recent publications regarding likelihood ratios, which express the risk associated with certain test scores (Smith, Ivnik, & Lucas, 2008).

While additional study of the deconstruction of race is needed, development of ethnic group norms itself is a challenging and complex task. Test publishers are increasingly making efforts to include important background variables in normative samples. However, the cost of recruiting and assessing large cohorts is prohibitive for individual centers and researchers and is often even a challenge for larger test publishing companies. While inclusion of demographic variables in research articles is an important piece to further develop this area, federal and foundation-funded grants may be necessary to support multicenter partnerships, ideally suited to study large, diverse cohorts.

Despite the development of racially and ethnically diverse norms, including the widely used Heaton norms for African American and Caucasian adults, the almost endless variety possible with regard to individuals' language, culture, and education backgrounds limits the application of demographically corrected norms for every possible clinical situation (Heaton, Miller, Taylor, & Grant, 2004). Local norms may be helpful for clinicians working with population groups with less common background variables; however, integration of test scores with a person's individual context is ultimately critical for formulation of an accurate diagnosis. In clinical practice, other issues regarding assessment of ethnic, linguistic, and cultural minorities are also present. The use of translated tests, interpreters, and bilingual psychometrists are all current methods for evaluating non-English-speaking patients. However, the shear complexity of language fluency is important to consider. Language fluency of an individual can vary widely with regard to (1) age of acquisition, (2) proficiency, (3) manner of learning, (4) amount of language exposure, (5) predominant mode of bilingual interaction, and (6) language structure variables (i.e., alphabetic versus logographic languages and phonemic versus nonphonemic status). Currently, the use of translated tests and official interpreters or bilingual psychometrists are preferred over using family members as interpreters and site translation, which refers to the practice of quickly translating a test with no standardization procedures. The importance of training in cross-cultural neuropsychology will become increasingly important as the Spanish-speaking Latino population in the United States grows.

In keeping with the demographic changes in the United States, training programs and national organizations are brainstorming ways to promote recruitment and retention of students with diverse racial and ethnic backgrounds in the study of neuropsychology. Emphasis has been placed on student exposure to neuropsychology earlier in academic careers, mentoring with minority and nonminority faculty who are sensitive to diversity issues and financial support of minority students. The study of cultural diversity itself has gained support and many licensing bureaus require documentation of diversity training in graduate school for licensure. Numerous organizations, including the American Psychological Association (APA) Division 40, the International Neuropsychological Society (INS), and the National Academy of Neuropsychology (NAN), promote awareness of diversity issues and foster collaborations among clinicians and researchers interested in diversity issues. Readers are encouraged to review current guidelines, including the APA Ethical Principles (2002) and the APA Multicultural Guidelines (2002) for further details.

Diversity in neuropsychology has become of increasing interest and importance; however, measures to improve diagnostic assessment of ethnic minorities may not fully address the issue of improving clinical care of ethnic minorities, given limited health care access of non-English-speaking patients. Access to care and other factors affecting ethnic minority patient outcomes have increasingly been examined in the field of neuropsychology. Readers are encouraged to review two issues of the Journal of Head Trauma Rehabilitation, dedicated to U.S. and international cultural issues in the rehabilitation of survivors of traumatic head injury ("Cultural Issues," 2007 and "International Programs and Perspectives," 2007). Articles in these issues highlight complex relationships between functional outcomes, disability severity, return to work, and utilization of professional psychological services in ethnic minority groups. The international issue describes models of care used in the context of various health care systems as well as international perspectives on research and development in the field of traumatic brain injury rehabilitation.

The study of health disparities among ethnic minority populations is a complex, but critical field of research. Multiple dimensions of disparity exist, including many of the variables discussed regarding the deconstruction of race such as ethnicity, education, income, occupation, and geography. Continuous study of these variables is critical to improve clinical care for ethnic minorities.

Cross References

- ▶ AACN Practice Guidelines
- ▶ Clinical Practice Guidelines
- ▶ Cultural Sensitivity
- ▶ Culture Fair Tests
- ▶ Ethics in the Practice of Neuropsychology

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Cultural Sensitivity

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Synonyms

Multicultural

Definition

Awareness of how a patient's background, including ethnicity and demographic factors, affects treatment, assessment, and research.

Current Knowledge

There are a couple of pertinent definitions, or applications, of cultural sensitivity to the field of neuropsychology.

The position of the American Psychological Association (APA), which is reflected in the 2002 APA ethical principles of psychologists and code of conduct, state "Psychologists are aware of and respect cultural, individual, and role differences, including those based on age, gender, gender identity, race, ethnicity, culture, national origin, religion, sexual orientation, disability, language, and socioeconomic status and consider these factors when working with members of such groups. Psychologists try to eliminate the effect on their work of biases based on those factors, and they do not knowingly participate in or condone activities of others based upon such prejudices."

Cultural sensitivity is also reflected in the 2002 APA Guidelines on multicultural education, training, research, practice, and organizational change for psychologists. The APA defines "multicultural" specifically as referring to interactions with "individuals from minority ethnic and racial groups in the United States and the dominant

European-American culture." This document states that all individuals are in a social, political, and economic context. Therefore, it is increasingly important for psychologists to be aware of the specific needs of individuals based on their ethnic/racial heritage and social group identity. Guidelines relating to research direct psychologists to "recognize the importance of conducting culture-centered and ethical psychological research among persons from ethnic, linguistic, and racial minority backgrounds."

A second area of cultural sensitivity that directly relates to neuropsychology is the impact of culture on testing and on normative data. It is important that an individual examinee's test performance be related to individuals of similar backgrounds. For example, the normative group of an elderly, well-educated African American man should closely match his background. Without close matching of cultural, demographic, and educational background, unacceptable problems with misclassification of normal test results as abnormal can result in misdiagnosis of the patient. There is evidence that examiners belonging to a culture different from that of the examinee can elicit some anxiety, which may have a deleterious effect on test scores on more demanding cognitive tasks (Nagra, Skeel, & Sbrage, 2007). Additionally, cultural background, demographics and educational history, can have a sizeable impact on normative data. Smith, Ivnik and Jucan (2008) discuss the development of norms specifically for older adults on commonly used neuropsychological tests. They subsequently developed more specific norms for older African Americans, as it was found that culture had a significant impact on normative data. Heaton, Miller, Taylor and Grant (2004) have published norms that take into account age, education, and cultural background. This area of cultural sensitivity plays an important role in neuropsychology in terms of diagnosis and test evaluation.

Cross References

► Cultural Diversity
► Normative Data

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Culturally Reduced Test

► Culture Fair Test

Culture Fair Test

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Synonyms

Culture free test; Culturally reduced test

Definition

Culture fair test is a test that is equally fair to all cultural groups. Fairness is related to a lack of bias in the interpretation or use of a test to classify or diagnose. In a culture fair test, the validity of the interpretation is similar across different cultural groups. It is unlikely that any test can entirely eliminate the influence of learning and cultural experience, given that the test content, language, directions, and validity criteria are culturally bound. However, avoiding culturally loaded items, items that are found to be unfair to certain groups of people, increases the likelihood of it being a culturally fair test. Culturally loaded items, such as those that utilize pictures or general information that are differentially prevalent for certain cultures, decrease the likelihood of a culturally fair test.

Cross References

► Cultural Sensitivity
► Raven's Progressive Matrices

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Culture Free Test

► Culture Fair Test

Cuneate Nucleus

► Nucleus Cuneatus

Cuneus

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Synonyms

Broadmann area 17

Structure

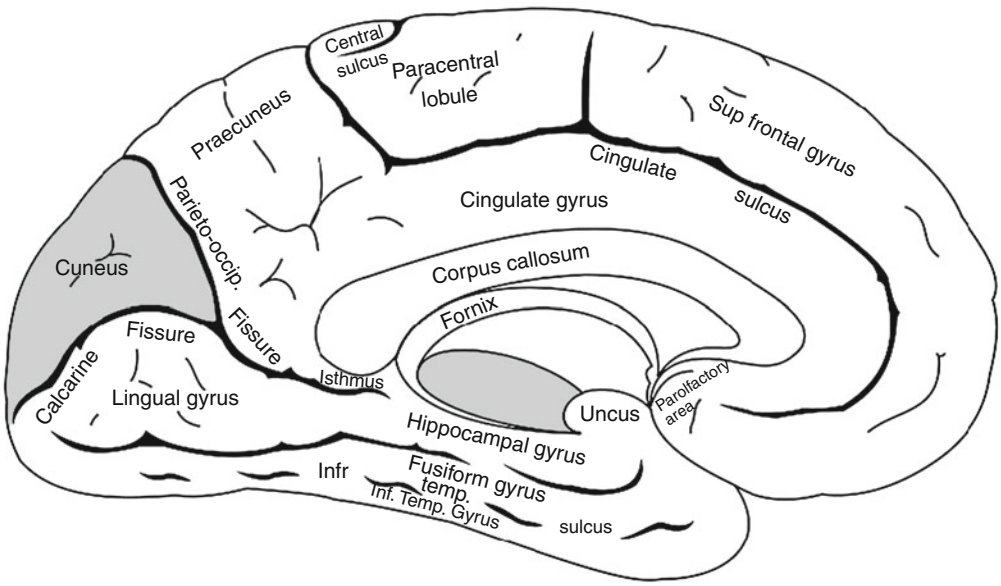
The term cuneus comes from the Latin term for wedge, which reflects the shape of this occipital brain area. The cuneus is a wedge-shaped cortical area located in the medial occipital gyri, superior to the calcarine fissure and posterior to the parietal–occipital fissure. The cuneus is part of the occipital lobe, corresponding to Broadmann area 17. Pyramidal cells in the cuneus (striate cortex) project to *extrastriate* cortices (Broadmann areas 18 and 19). The cuneus consists of both striatal and extrastriatal visual cortex consisting of five layers. The striatal areas are largely posterior and are idiotypic homomodal (Mesulam's classification), while the extrastriatal areas contain heterotypic cell types that respond to more complex visual information. The cuneus receives input from the contralateral superior retina corresponding to the lower visual field. From the extrastrial areas of cuneus, information is processed through both ventral and dorsal pathways. The dorsal pathway is particularly important for higher spatial analysis and visual integration.

Function

The cuneus plays as essential role in primary visual processing. The striatal regions that contain primary visual processing areas contain neurons with small receptive fields that are sensitive to very basic visual frequency information related to position, local orientation, spatial-frequency, and color (Beason-Held et al., 1998; Jeannerod, 2004; Ulbert, Karmos, Heit, & Halgren, 2001; Ungerleider & Pribram, 1977). However, more anterior to the primary visual areas are neurons that respond to more complex information contained in the visual information processed by the primary receptors (Ungerleider & Haxby, 1994). Accordingly, the cuneus plays a role in both primary and secondary visual processing. Extrastriatal areas of the cuneus are known to respond to reward, anticipatory, attention, and working memory manipulations, and there is even evidence that posterior striatal areas respond to attentional signals, providing evidence that this cortical plays some role in higher cognitive function involving basic visual information.

Illness

Damage to the cuneus would typically occur among patients experiencing focal brain lesions. Most often such lesions occur secondary to cerebral infarction (stroke) typically involving the posterior cerebral circulation, though neurosurgery to remove neoplasm may result in focal damage to the cuneus as well. Some atypical neurodegenerative diseases are also known to cause tissue loss in this occipital area. The effects of focal damage to the cuneus are known primarily from experimental ablation in primate studies and to some extent from single-



Cuneus. Figure 1 Sagital illutration showing the cuneus in the posterior cortex

case studies of stroke. Posterior cuneus lesions tend to disrupt primary visual perception, particularly response to stimuli occurring in the lower visual fields. Damage to more anterior areas of the cuneus also affects visual functions (e.g., movement perception), though often for higher level operations involved in object perception and spatial analysis, including attention (De Weerd, Peralta, Desimone, & Ungerleider, 1999).

Interestingly, there is also evidence that cuneus volume is related to other behavioral processes not obviously tied in a direct way to visual processing. For example, one recent study found greater cuneus volume to be associated with greater inhibitory control among patients with bipolar disorder affective disorder (Haldane, Cunningham, Androutsos, & Frangou, 2008). Another study found that compulsive gamblers had increased activity in dorsal visual processing pathways, including the cuneus when compared with controls (Crockford, Goodyear, Edwards, Quickfall, & el-Guebaly, 2005). Whether such findings reflect a specific role of the cuneus in these psychiatric disorders versus more incidental findings needs to be determined in future investigations.

Cross References

- ▶ Feature Detection
- ▶ Spatial Frequency Analysis
- ▶ 'What System'
- ▶ 'Where System'

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Cushing's Syndrome

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Short Description or Definition

Endogenous Cushing's syndrome (CS) is a rare disorder occurring in about 0.7–2.4 per million population per year (Newell-Price, Bertagna, Grossman, & Nieman, 2006). CS includes symptoms and signs resulting from chronic supraphysiological exposure to glucocorticoids (GC). The classical clinical features of CS are centripetal obesity with abnormal fat distribution, mainly affecting the face, neck, trunk, and abdomen, and sparing the extremities. Other findings are facial plethora, easy bruisability, purple abdominal striae, hirsutism, muscle weakness, hypertension, and glucose intolerance (Stewart, 2008). In addition, mood alterations and psychiatric diseases – mainly depressive and anxiety disorders – as well as cognitive impairment are highly prevalent in CS patients (Bourdeau et al., 2005).

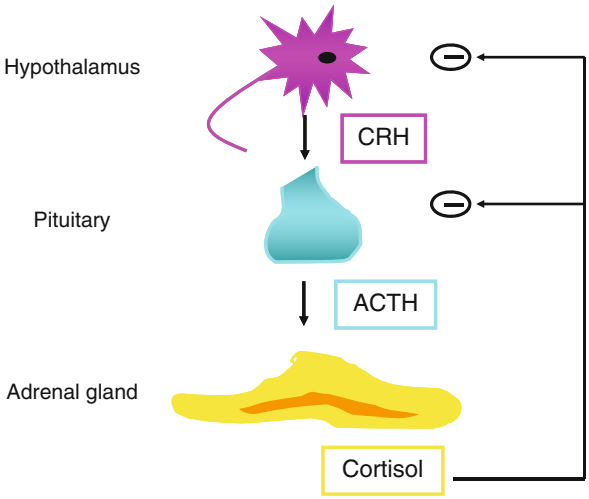
The principal aim of this chapter is to present psychiatric and cognitive data on adult patients with CS. The recognition of psychoneurological abnormalities associated with hypercortisolism is of clinical importance in the management of patients affected by CS.

Etiology

Exogenous CS refers to iatrogenic CS resulting from chronic GC therapy. GC, which have potent anti-inflammatory and immunologic actions, are widely used for the treatment of various diseases such as inflammatory bowel disease, asthma, rheumatoid arthritis, and organ transplantation. Thus, exogenous CS is the most common form of CS. Endogenous CS may be caused by dysregulation at various levels of the hypothalamic–pituitary–adrenal axis (HPA), resulting in cortisol overproduction. The regulation of cortisol synthesis and secretion is mediated by the hypothalamus, the pituitary, and the adrenal glands. Corticotropin (ACTH) is synthesized and secreted by corticotrophs of the pituitary which are mainly regulated by two hypothalamic hormones, corticotropin-releasing hormone (CRH), and arginine-vasopressin. Pituitary ACTH is secreted in the peripheral circulation and reaches cells in the cortex of the adrenal gland where cortisol biosynthesis is initiated. Cortisol itself exerts negative feedback control on ACTH and CRH secretion, maintaining a normal secretion rate in case of HPA activation, in stress for example (Fig. 1). ACTH is released physiologically in a series of secretory episodes, followed by an equal number of cortisol bursts in plasma. Thus, plasma cortisol levels are maximum in the early morning hours around awakening, gradually decline throughout the morning, and reach nadir values late in the evening. Midnight serum cortisol >200 nmol/L strongly suggests the diagnosis of CS (Stewart, 2008). Variations of ACTH and cortisol values constitute the normal circadian rhythm. CS is caused by pituitary or adrenal abnormalities or may be secondary to ectopic ACTH or CRH secretion by various nonpituitary tumors, such as lung carcinoma. CS is classified into ACTH-dependent and -independent causes which are described in Table 1. Cushing's disease refers to CS due to excessive pituitary ACTH secretion from pituitary tumors.

Evaluation or Diagnosis

The diagnosis of endogenous CS may be challenging and require the expertise of an endocrinologist. Authors will briefly discuss the evaluation and various tests used to diagnose CS. The first step is to confirm CS by biochemical investigation. Then, the cause of the disease should be



Cushing's Syndrome. Figure 1 Schematic representation of the hypothalamus–pituitary axis. Normally cortisol negatively regulates corticotropin-releasing hormone (CRH) and ACTH secretion

Cushing's Syndrome. Table 1 Classification of the etiologies of endogenous Cushing's syndrome (CS)

ACTH-dependent CS (80% of cases)
Pituitary adenoma (Cushing's disease) (80%)
Ectopic ACTH or corticotropin-releasing hormone (CRH) secretion from nonpituitary neoplasms (20%)
ACTH-independent CS (20% of cases)
Adrenal adenoma (~60%)
Adrenal carcinoma (~40%)
Bilateral ACTH-independent macronodular adrenal hyperplasia (rare)
Bilateral micronodular adrenal hyperplasia (primary pigmented nodular adrenocortical disease and nonpigmented adrenal hyperplasia) (rare)

identified. During the investigation, it is important to evaluate patients for other illnesses, drugs, alcohol, or depression. All these conditions may lead to misinterpretation of the endocrine test results. The overnight 1-mg dexamethasone suppression test, a screening tool for the diagnosis of CS, consists of administering 1 mg of dexamethasone at bedtime (23:00 h) with measurement of plasma cortisol the following morning (08:00–09:00 h). Normal subjects should suppress plasma cortisol to less than 50 nmol/L. Measurement of free cortisol in 24-h urine collection is useful to confirm the diagnosis; values fourfold greater than the upper limit of normal are highly suggestive of CS. More recently, late-night salivary cortisol has proven useful in the diagnosis of CS (Nieman et al., 2008). Once a diagnosis of CS is confirmed, the next step is to establish the etiology. Plasma ACTH will distinguish between ACTH-dependent and -independent CS. ACTH values lower than 1.1 pmol/L indicate ACTH-independent CS, and CT imaging of the adrenal glands should be performed (Newell-Price et al., 2006). ACTH values above 3.3 pmol/L suggest ACTH-dependent CS, and further endocrine investigations should be undertaken, including magnetic resonance imaging of the pituitary glands and other tests that are beyond the scope of this chapter.

Psychiatric Diseases and Neuropsychological Disturbances Associated with CS

Understanding the importance and complexity of adrenal hormones impacting cerebral function, particularly affect and cognition, has increased considerably in the recent years. McEwen, Weiss, and Schwartz (1968) demonstrated the presence of glucocorticoid receptors (GR) in the rat brain. Loss of brain volume was documented in patients with endogenous and exogenous CS (Bourdeau et al., 2002). In addition, cerebral magnetic resonance spectroscopy revealed abnormalities of cerebral metabolism in patients with CS (Khiat et al., 1999).

Psychiatric disturbances occur in patients with both exogenous and endogenous CS. Interestingly, patients with endogenous depression frequently have high cortisol levels, and present HPA axis dysregulation, disclosed by abnormal suppression on the dexamethasone test. However, diurnal rhythm is usually maintained in depressed patients, and they do not develop physical signs of CS. CS is also associated with an increased prevalence of cognitive impairments, particularly of attention, learning, and memory. Patients with CS represent a natural model that illustrates the interactions between hypercortisolism and brain function. The following section focuses on the psychiatric and neuropsychological anomalies in CS.

Psychiatric Disorders Associated with CS

Depression is a frequent feature of CS. In fact, several reports indicate that more than 50% of CS patients present severe depressive symptoms that reach the threshold of a major depressive disorder. Haskett (1985) observed a lifetime history of psychiatric symptoms and signs in patients with proven CS. He found that the majority of them experienced psychiatric disturbances that closely resembled typical syndromes, with episodes of major affective disorder (endogenous depression, mania, or hypomania). Moreover, Haskett (1985) noted that there were no significant differences between pituitary-dependent and -independent forms of CS in the occurrence of major depression. His observations were later confirmed by independent investigations (Kelly, 1996; Sonino, Fava, Belluardo, Girelli, & Boscaro, 1993). Using DSM-III criteria (American Psychiatric Association, 1980), Hudson, Hudson, Griffing, Melby, and Pope (1987) reported a high lifetime diagnosis of mood disorders in CS patients, of whom 14% were assessed to have major affective disorder. Loosen, Chambliss, DeBold, Shelton, and Orth (1992) recognized major depressive disorder in 68% of adult patients with CS. In addition, this disorder was frequently associated with anxiety disorder (generalized anxiety disorder and/or panic disorder), indicating a syndrome of anxious depression in

patients with active CS (Loosen et al., 1992). Focusing on Cushing's disease, Sonino, Fava, Raffi, Boscaro, and Fallo (1998) found major depression in more than half of their patients studied. Moreover, major depression was significantly correlated with older age, female gender, higher pretreatment urinary cortisol levels, and more severe clinical conditions. Kelly, Kelly, and Faragher (1996) reported that some patients with CS were diagnosed to have depression and were more depressed than patients with other pituitary tumors. Concentrating on the question of whether depression in CS more consistently resembles any of the clinical subcategories, Dorn et al. (1995) established that patients with active endogenous CS exhibited significant psychopathology, expressed primarily by "atypical" depression. This concept of atypical depression refers to the presence of weight gain or increased appetite, fatigue, hypersomnia, leaden paralysis, and longstanding interpersonal rejection sensitivity (American Psychiatric Association, 1994).

In summary:

1. More than 50% of patients with CS report severe depressive symptoms
2. There are no significant differences between ACTH-dependent and -independent forms of CS in the occurrence of major depression
3. Major depressive disorders are commonly associated with anxiety disorders
4. Major depression is significantly correlated with older age, female gender, higher pretreatment urinary cortisol levels, and more severe clinical conditions
5. A high prevalence of atypical depression features is seen in CS

Neuropsychological Disorders Associated with CS

Neuropsychological disorders have been detected in about two-thirds of patients with CS. The first such investigation evaluated unselected CS patients on the Michigan Neuropsychological Test Battery (Whelan Schteingart, Starkman, & Smith, 1980). This test battery included standardized and objective measures with a broad range of discrete language, verbal and nonverbal reasoning, auditory and visual memory, and sensory and motor functions. The study revealed diffuse bilateral cerebral dysfunction in CS with impairment in nonverbal, visual–ideational, visual-memory, and spatial–constructional abilities. Starkman, Schteingart, and Schork (1981) reported generalized impairment of cognitive functions (decreased concentration and memory, perceptual distortions). More recently, Starkman, Gebarski, Berent, and Schteingart (1992) observed moderate-to-severe deficits in a wide variety of language and nonverbal subtests among more than two-thirds of their CS patients. Difficulties with reasoning ability, comprehension, and processing of new information also were found. Deficits in these areas of cognition were confirmed in a recent controlled study of 19 CS patients (Forget, Lacroix, Somma, & Cohen, 2000). In fact, memory was the most studied function in CS because the emphasis was on the hippocampus which is rich in GR. However, the distribution of GR in many areas of the cerebral cortex suggests that extrahippocampic sites could also be the target of cortisol, and generalized impairment of cognitive functions after chronic hypercortisolism may be related to this wide dispersion of GR.

Treatment

The goal of CS treatment is to correct the hypersecretion of adrenal hormones. In most cases, tumor-specific surgery will be performed. In Cushing's disease, pituitary tumors should be resected, and in adrenal CS adrenalectomy performed. In cases of ectopic ACTH secretion secondary to benign tumors, the latter should be removed; but in most cases, there are metastatic malignant tumors. If surgery is unsuccessful, drugs that block steroid synthesis may be administrated to achieve eucortisolism (Biller et al., 2008; Stewart, 2008).

Natural History, Prognostic Factors, Outcomes

The reversibility of brain volume loss after the correction of hypercortisolism in patients affected by CS was previously demonstrated (Bourdeau et al., 2002). Similarly, treatment of hypercortisolism is often efficacious in decreasing the depressive components of illness. Significant improvement in depressive symptoms was observed when patients with CS were assessed by psychometric methods before and after the treatment of their endocrine condition, (Dorn et al., 1997; Forget, Lacroix, & Cohen, 2002; Kelly et al., 1996; Starkman, Giordani, Gebarski, & Schteingart, 2006; Starkman, Schteingart, & Schork, 1986). In a longitudinal study of depressive and

neuropsychological symptoms in CS, Forget et al. (2002) reported a significant improvement in Beck Depression Inventory scores 1 year after the correction of hypercortisolism. More recently, Starkman et al. (2006) showed significant changes in depression and anxiety subscales on SCL-90-R after the successful treatment of hypercortisolism in CS. However, psychological distress does not always disappear upon proper treatment. For example, Dorn et al. (1997) disclosed that the incidence of atypical depression progressively decreased after the correction of hypercortisolism – this disorder, however, continued to be the prevailing psychiatric diagnosis after the successful cure of CS. It is likely that the psychopathology symptoms still seen after the correction of CS may be related to the as yet unexplored interaction between endocrine and psychosocial factors. Indeed, despite the correction of hypercortisolism, patients with CS experience a considerable decrease in quality of life, even after long-term cure (Lindsay, Nansel, Baid, Gumowski, & Nieman, 2006; van Aken et al., 2005). The authors postulated that there might be irreversible changes in central neural function in these patients (Heald et al., 2004).

Few studies have objectively examined the reversibility of cognitive disorders after successful treatment. Mauri et al. (1993) demonstrated significant amelioration in verbal memory performance and attentive and visuomotor functions 6 months after surgery. No changes in other cognitive functions could be detected. On the other hand, no differences between Cushing's patients and their controls were found in cognitive function 12 months post-treatment (Dorn & Cerrone, 2000). Forget et al. (2002) evaluated subjects who presented with CS on a battery of tests, including attention, visuospatial processing, memory, reasoning, and verbal fluency. Except for one task of visual organization, the results showed little change in performance, suggesting that prolonged exposure to high cortisol levels can cause long-lasting deleterious effects on cognitive function. The data indicate that correction of hypercortisolism is not necessarily correlated with short-term improvement in cognitive function. More recently, Hook et al. (2007) postulated that the age of Cushing's patients appeared to be a significant factor determining when and how cognitive function manifested signs of recovery.

In conclusion, the psychiatric and neuropsychological disturbances associated with CS may compromise quality of life and produce a broad array of handicaps and disabilities. Moreover, these functional impairments may persist long after successful treatment and in clinical remission. Thus, such symptoms warrant clinical attention.

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Customized Employment

► Supported Employment

Customized Job Retention Services

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Definition

Job retention is the process of facilitating the tenure of an individual in a particular employment setting. Customized job retention services begin with the completion of a thorough assessment to determine the individual's strengths, interests and preferences, and support needs. The results of the assessment will inform the job development and selection process. Placement and retention plans are individualized; however, general guidelines insure job retention including promoting consumer choice and involvement, identifying jobs that are meaningful in terms of wages earned, benefits, inclusion, and opportunities for career growth, and the provision of long-term supports. Research indicates that the provision of ongoing supports increases the employment retention rate of individuals with significant disabilities. Retention services may also include supports addressing skill acquisition, production demands, the development of soft skills such as interpersonal communication, problem-solving skills and responding to the daily expectations and stressors associated with the respective position. Retention services must also ensure that the needs of the employer are understood and met.

Job retention is directly related to quality of life, as reported by individuals with disabilities. Employment statistics (i.e., employment rate, ability to stay in a job overtime) document the need for the provision of customized job retention services. Improved job tenure rates for individuals with disabilities are a primary objective for rehabilitation services.

Cross References

► Customized Employment
► Supported Employment

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Cut Scores

► Cut Off Scores, Cutting Scores

Cut Off Scores, Cutting Scores

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Synonyms

Cut scores

Definition

Cutoff scores, also referred to as cutting scores, are scores that differentiate the levels of performance.

Current Knowledge

The reliability of a measure is an essential factor to consider when using cutoff scores. Nunnally and Bernstein (1994) suggest that a test must have an internal consistency coefficient of at least 0.90, and ideally above 0.95, to designate a particular score as a diagnostic cutoff point, although this might be a high standard. For instance, if a child is demonstrating a learning disorder, the tests used to assess the disorder must be reliably able to measure the particular abilities, taking into consideration the span of the standard error, to place an education-altering label on that child. If such standards are not followed, then clinicians run the risk of basing a decision on a "false-positive" (the discovery of a limitation when none exists in actuality) or a "false-negative" (the missing of a limitation when one truly exists).

To consider an example, a cutoff score may be the score differentiating between intact performance and impairment on a particular test. To speak in terms of diagnosing cognitive impairment, in a normal distribution of scores, a cutoff score would be any score below 95% which is obtained by the intact group within a particular standardization sample.

Clinicians may adjust cutoff scores because of individual or cultural variables that render the reliability of a test nonapplicable to the individual or sample being assessed. Otherwise, the clinician could run the risk of pathologizing an individual who does not truly exhibit the pathology.

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CVA

► Stroke

CVLT

► California Verbal Learning Test (California Verbal Learning Test-II)

CVLT-C

► California Verbal Learning Test – Children's Version

Cyst

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Synonyms

Nodule; Polyp

Definition

Cyst refers to an abnormal growth of benign cells, typically containing normal cerebrospinal fluid. Central nervous system cysts may occur as neoplasms or subsequent to leptomeningitis, hemorrhage, or surgery. Most patients presenting with cyst are asymptomatic, though headache; calvarial bulging; intracranial hypertension; craniomegaly; developmental delay; visual loss; precocious puberty; and seizures, with focal neurologic signs can occur. Additional cognitive consequences may include symptoms of dementia and inattention. Arachnoid cysts represent the most common form of benign cyst and occur in the cerebrospinal axis. Size of arachnoid cysts is not directly affected by changes in the ventricular system.

Cross References

► Neoplasm

Cytochrome P450

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Synonyms

P450 isoforms; P450 isoenzymes; P450 system

Definition

The liver-mediated cytochrome P450 isoenzymes (abbreviated as CYP) constitute an enzyme family that plays a major role in drug metabolism and is the cause for some adverse drug reactions. Metabolism and clearance of a drug are presumed to be stable, allowing for basic predictions about a drug's dose, blood levels, and pharmacological effects. Drugs are typically metabolized by a limited number of mechanisms, some of these include the P450 enzyme pathways. Drugs, foods, or herbal preparations that alter the efficiency of those pathways may also alter the plasma concentration of some drugs by inhibiting or inducing (increasing) the efficiency of a particular enzymatic pathway. There are drugs/herbs that can simultaneously induce and inhibit different isoenzymes (St. John's Wort), or simply induce their own metabolism (carbamazepine).

Although over the counter preparations are less likely to be mentioned, some effect P450 enzyme pathways (e.g., cigarettes or the topical antifungal ketoconazole). Smokers, for example, may require increased doses of drugs because cigarette smoking has the ability to induce an enzyme that deactivates some medication. As a corollary, an abrupt cessation of smoking would then increase the plasma level of these drugs unless and until the dose is modified.

P450 isoenzymes are widely distributed, but often thought of as liver enzymes because the liver metabolizes drugs through oxidation as well as reduction and hydrolysis. The p450 cytochrome system is critical to a number of oxidative reactions (the addition of an oxygen molecule or the removal of hydrogen from a molecule). The P450 isoenzymes share a heme (iron) cofactor, although they may use a number of different molecules as a basis for enzyme reactions. The term cytochrome arose because the isoenzymes were differentiated by their cellular (*cyto*) location and their color (*chrome*) or wavelength during spectrophotometric studies. P450 enzymes absorb light at 450 nm. Specific CYP enzyme pathways are noted in shorthand (e.g., CYP3A4 or CYP 2D6). Typically, authors include the prefix CYP before a three character alphanumeric 3A4, but it may be abandoned if the author is writing casually or for an audience familiar with the terms. The alphanumeric denotes the gene family, subfamily, and individual gene. The three most common isoenzymes involved in human drug metabolism are CYP3A4, CYP2D6, and CYP2C9.

Cross References

► Cmax
► p450 Cytochrome System

► Pharmacokinetics
► Side Effects

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Cytotoxic Edema

► Cerebral Edema