V

V1

► Striate Cortex

V2, V3, V4, V5

► Extrastriate

Validity Scales (MMPI)

Richard Temple
CORE Health Care
Dripping Springs, TX, USA

Synonyms

F scale; K scale; L scale; Variable response inconsistency scale (VRIN, MMPI)

Definition

Validity scales on the MMPI and its revisions measure the extent to which respondents endorse items in a forthright manner. They are not to be confused with indices of test validity, which is the extent to which a test measures what it purports to measure.

Current Knowledge

Generally speaking, the validity of an individual's responses come into question when there is suspicion that they have responded randomly (i.e., without reading the item or did not understand the items), or significantly and systematically over- or underreported symptoms. The primary validity scales inherent to the MMPI include the F scale (endorsement of unusual symptoms), L scale (endorsement of socially laudable, but unusual traits), and K (defensiveness). There are also two scales designed to detect inconsistent responding; true response inconsistency scale (TRIN; tendency toward yea- or nay-saying) and the variable response inconsistency scale (VRIN; responding to similar content inconsistently). Although guidelines and cutoffs have been established for the various validity indices, the overall decision about the validity of a given profile is often based on clinical judgment and the overall pattern of validity indices. If a profile is deemed invalid, no further analysis of the clinical scales should take place.

Profile validity is particularly important in forensic neuropsychological settings. In addition to the validity scales inherent to the MMPI, there have been various efforts to construct validity scales specific to the forensic setting. One of the most researched of these scales is the Fake Bad Scale (Lees-Haley et al., 1991). Berry and Schipper (2007) deemed the MMPI-2 to be one of the best instruments for the detection of feigned symptom reports during forensic neuropsychological evaluations. It is important that the clinical neuropsychologist assess effort across both cognitive and personality measures, as individuals who exaggerate or otherwise distort their performance may not do so evenly across both types of measures. This may be related to the sophistication level of the patient or possibly attempts to selectively feign one type of impairment but not another (e.g., Temple et al., 2003). Readers are referred to the MMPI entry for further discussion of limitations of this self-report measure when used with neuropsychological populations (see also Gass, 2006 and Lezak, Howieson, & Loring, 2004).

Cross References

► F Minus K Index
► Fake Bad Scale
► Faking Good, Bad
► MMPI
► TRIN
► VRIN

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

References and Readings

Berry, D. T., & Schipper, L. J. (2007). Detection of feigned psychiatric symptoms during forensic neuropsychological evaluations. In G. J. Larabee (Ed.), *Assessment of malingered neuropsychological deficits*. Oxford: Oxford university Press.

Gass, C. (2006). Use of the MMPI-2 in neuropsychological evaluations. In J. Butcher (Ed.), *MMPI-2: A practitioner's guide* (pp. 301–326). Washington, DC: American Psychological Association.

Lees-Haley, P. R., English, L. T., & Glenn, W. J. (1991). A Fake Bad Scale for the MMPI-2 for personal injury claimants. *Psychological Reports, 68*, 203–210.

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

Temple, R. O., McBride, A. M., Horner, M. D., & Taylor, R. M. (2003). Personality characteristics of patients showing suboptimal cognitive effort. *The Clinical Neuropsychologist, 17*, 402–409.

Valproate

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

Generic Name

Valproate

Brand Name

Depakene, Depacon, Depakote, Depakote ER, Stavzor

Class

Anticonvulsant; Mood stabilizer; Migraine prophylaxis

Proposed Mechanism(s) of Action

While the certain mechanism is unknown, it is believed to block voltage-sensitive sodium channels and to increase brain concentrations of GABA.

Indication

Acute mania, complex partial seizure disorder, simple and complex absence seizures, migraine prophylaxis

Off Label Use

Maintenance treatment of bipolar disorder, bipolar depression, psychosis or schizophrenia

Side Effects

Serious

Polycystic ovaries, *hepatoxicity* (can be fatal), *pancreatitis* (can be fatal), history of inducing suicidal ideation

Common

Sedation, tremor, headache, abdominal pain, diarrhea, decreased appetite, vomiting, constipation, weight gain, hyperandronenism, hyperinsulinemia, problems controlling lipid level, diminished bone mineral density

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

Variable Response Inconsistency Scale (VRIN, MMPI)

Richard Temple
CORE Health Care
Dripping Springs, TX, USA

Synonyms

VRIN

Definition

A validity scale on the Minnesota Multiphasic Personality Inventory (MMPI)-2, consisting of 67 pairs of items (49 unique pairs) that either have similar or opposite content. Depending on the content of the item pair, inconsistent responding can take the form of the same (e.g., both "true" and "false") or different responses. Very high or very low scores are indicative of inconsistent responding. Greene (1991) suggested cutoff scores of seven or below, and 16 or higher, to indicate inconsistent responding (pg. 69). Pinsoneault (2007) found the VRIN scale to be the most sensitive to random responding of the available MMPI-2 validity scales. The clinical neuropsychologist integrates information from MMPI inconsistency measures together with other neuropsychological data and symptom validity scores to determine whether the inconsistency reflects random responding and/or carelessness versus a cognitive impairment issue (e.g., ► reading comprehension and ► attention). Readers are referred to the MMPI entry for a discussion of limitations of this self-report measure when used with neuropsychological populations (see also Gass, (2006) and Lezak, Howieson, & Loring, (2004)).

Cross References

► F Minus K Index
► F Scale
► Fake Bad Scale
► Faking Good, Bad
► K Scale
► L Scale
► MMPI
► True Response Inconsistency Scale (TRIN, MMPI)
► Validity Scales (MMPI)

References and Readings

Gass, C. (2006). Use of the MMPI-2 in neuropsychological evaluations. In J. Butcher (Ed.), *MMPI-2: A practitioner's guide* (pp. 301–326). Washington, DC: American Psychological Association.

Greene, R. L. (1991). *MMPI-2/MMPI: An interpretive manual.* Needham Heights, MA: Allyn and Bacon.

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

Pinsoneault, T. B. (2007). Detecting random, partially random, and nonrandom Minnesota Multiphasic Personality Inventory – 2 protocols. *Psychological Assessment, 19*, 159–164.

Variant

► Polymorphism

VAS

► Visual Analog Scale

Vascular Cognitive Disorder

► Vascular Cognitive Impairment

Vascular Cognitive Impairment

Holly Rau, Yana Suchy
University of Utah
Salt Lake City, UT, USA

Synonyms

Vascular cognitive disorder; Vascular cognitive impairment without dementia; Vascular dementia; Vascular disorders; Vascular mild cognitive impairment

Definition

The term vascular cognitive impairment (VCI) refers to a continuum of neurobehavioral deficits associated with different vascular pathologies. This continuum ranges from subtle deficits in executive functioning and processing speed to more frank dementia. Thus, VCI can be observed in patients who do not meet criteria for dementia, but screen positive for a history of vascular pathology, as well as in patients with vascular dementia. VCI can also be a comorbid condition in other mixed dementias, such as Alzheimer's dementia (AD) with vascular components. More recently, VCI has been proposed as a means to identify and treat patients in earlier stages of cerebrovascular disease.

Historical Background

During the late twentieth century, Alzheimer's disease was recognized as the leading cause of dementia. Because of this, the core diagnostic features of Alzheimer's disease – memory loss, multiple cognitive disturbances, and daily life impairment – permeated the diagnostic criteria for other classes of dementia. For vascular dementia, these core diagnostic features are typically present in later, more advanced stages of the disease, which complicated efforts aimed at early detection and prevention. As a result, a more general term was needed to describe the specific profiles of cognitive decline in patients who do not meet criteria for dementia, but who screen positive for a history of vascular pathology.

The term vascular cognitive impairment was adopted by the International Psychogeriatric Association in 1991 to describe the continuum of cognitive changes associated with vascular pathology (O'Brien et al., 2003). The introduction of this term has been met with some disagreement regarding its use, such as whether VCI should refer to all classes of cerebrovascular-related cognitive changes or to only those not severe enough to meet the criteria for dementia. Because of the no agreed upon standard currently exists for describing early stages of cognitive impairment associated with vascular factors, the National Institute of Neurological Disorders and Stroke (NINDS) and the Canadian Stroke Network (CSN) have proposed a set of research standards in 2005 to streamline diagnostic criteria and terminology (Hachinski et al., 2006). Until this new terminology becomes formalized, VCI continues to be used broadly and in reference to the entire spectrum of cognitive impairment associated with cerebrovascular disease. Therefore, the conscientious reader must consider the context and usage when interpreting its specific meaning.

Current Knowledge

Pathophysiology

VCI is characterized by cognitive changes resulting from either extracranial or intracranial vascular disease. Extracranial vascular disease, resulting from blockage to one of the three major cerebral arteries, typically produces a more abrupt decline in cognitive function. Conversely, intracranial vascular disease tends to progress more slowly and is associated with vascular damage to either subcortical regions, including the white matter and brainstem structures, or the cerebral cortex. Of the VCI subtypes, subcortical VCI is the most common, and is estimated to contribute to 40% of VCI cases. Subcortical vascular disease typically develops secondary to atherosclerosis or hypertension and presents as variety of vascular pathologies, ranging from small vessel ischemic disease to multi-infarct or lacunar states, all of which can be associated with cognitive impairment. The primary mechanism for subcortical VCI is small vessel cerebrovascular disease, in which cognitive decline occurs subsequent to numerous, diffuse, bilateral infarctions. The second manifestation of intracranial vascular disease, cortical VCI, occurs secondary to vascular damage to the cerebral cortex, which is typically caused by cerebral infarctions or hemorrhages. Although numerous pathologies have the potential to impair critical cognitive functions, the most important factors for determining cognitive impairment are location and total volume of brain destruction.

Prevalence Rates

Cases of pure VCI, vascular dementia, and dementia with a vascular component (e.g., mixed dementia) outnumber cases with pure Alzheimer's disease in adults aged 75–84 years. In the Canadian Study of Health and Aging (CSHA), VCI without dementia was the most prevalent form of VCI, with rates estimated at 2.6% for adults aged 65 years or older. Lesions associated with subcortical ischemic vascular disease can occur as early as 30 years of age, with prevalence rates increasing dramatically with age. For example, rates in the USA are estimated at 1.4% for adults over 65 years, and up to 3.8% in adults over 85 years. While sex differences are less pronounced in later stages of VCI, males are typically more affected by VCI than females in earlier stages.

Symptoms and Presentation

The clinical features associated with vascular dementia apply to a lesser degree in patients with VCI without dementia. For example, focal signs are less common in VCI without dementia, and patients usually present with a series of minor changes in cognitive function over time. In addition, patchy cognitive deficits are less common, as they are typically associated with strategically located infarcts (e.g., ▶ multi-infarct dementia). VCI deficits are distinct from those associated with AD in that executive dysfunction and psychomotor slowing, rather than memory per se, constitute the early hallmark deficits. For example, immediate

and delayed memory performances tend to remain more intact for VCI patients compared to those diagnosed with AD.

The specific profile of neurocognitive deficits associated with VCI depends on the underlying syndrome or cause, as well as the location and extent of neuronal damage. In light of this variability, executive dysfunction, psychomotor slowing, and impaired attention constitute three of the most prominent features of VCI. These deficits are associated with disruptions of the fronto-striatal-thalamic circuits, which typically result from subcortical vascular disease (e.g., ▶ lacunar infarcts, ▶ white matter lesions). Specific abilities that may be impaired include working memory, set maintenance, cognitive abstraction, planning, abstract reasoning, set formation, mental flexibility, initiation, and verbal fluency.

Noncognitive features include agitation, disinhibition, aggression, and possible motor deficits (e.g., imbalance, gait disorder, urinary frequency). In addition, there is a strong relationship between subcortical changes and white matter lesions and depression, even in the absence of dementia. Even small amounts of cerebral infarction have been associated with increased risk for depression. Other neuropsychiatric symptoms associated with VCI include apathy, anxiety, emotional lability, and psychosis.

Diagnosis and Assessment

There are currently no agreed upon, evidence-based, neuropathological criteria for diagnosing VCI, especially in the absence of dementia. However, because frontal and subcortical functions are often impaired in VCI patients, the phenotypic pattern of deficits typically involves executive dysfunction and psychomotor slowing. For example, compared to AD patients, VCI patients experience greater difficulty on more complex tasks requiring increased attention, vigilance, and cognitive flexibility. Therefore, neuropsychological batteries should be selected to assess executive functioning specifically, with timed executive function tasks being particularly sensitive to the added component of slowed information processing.

In order to assess a larger range of cognitive deficits accompanying the core executive and psychomotor features of VCI, the Neuropsychological Working Group of the NINDS-CSN has recommended testing four cognitive domains: executive/processing, speed/activation, language, visuospatial functions, and memory. Because of the disruption to frontal-subcortical circuits, patients with VCI are expected to perform more poorly across cognitive domains on tasks having an executive component (e.g., ▶ phonemic fluency, visual organization, ▶ procedural memory). Bedside tests often used in this context include semantic and phonemic fluency, digit span, digit symbol-coding (from the ▶ *Wechsler Adult Intelligence Scale* – third edition), trail making or maze tests, verbal learning tests, clock drawing, complex figure drawing, and confrontation naming.

Given the numerous neuropsychiatric complications associated with VIC, it is also recommended that changes in neurobehavioral status and mood be assessed in tandem with neurocognitive function. The NINDS-CSN recommends the neuropsychiatric inventory (NPI), given its assessment of behavioral domains often affected by VCI, in addition to a measure of depressive symptoms, such as the ▶ Center for Epidemiologic Studies-Depression Scale (CES-D).

Treatment

The principle method of treating VCI is prevention, which begins through early identification of patients at risk and continues through modification of risk factors. Early intervention is crucial, since vascular injuries can begin early in life, but difficult given the often silent occurrences. For example, the incidence of silent infarcts was estimated to exceed 9 million cases in the USA alone, based on imaging studies conducted in 1998. Therefore, intervening before clinical symptoms occur appears to be the most promising effort aimed at prevention. Current strategies emphasize prophylactic treatment of known primary risk factors for small vessel disease and stroke, which include hypertension, orthostatic hypotension, ischemic heart disease, homocysteine, high cholesterol, diabetes, obesity, and smoking.

Primary preventative efforts for stroke focus on reducing high blood pressure, since hypertension is the most powerful treatable risk factor. Reduction of systolic and diastolic blood pressure has been associated with significantly reduced incident dementia. Antihypertensive medications are predominantly preventative, since research has failed to demonstrate an association between blood-pressure lowering and reduction of stroke-related cognitive decline. Secondary treatments, which aim to prevent future occurrences of stroke, include antiplatelets, anticoagulants, carotid endarterectomy, and blood-pressure lowering medication.

Inflammation may be another important target of preventative treatments for VCI. Inflammatory markers associated with risk of cognitive impairment include high-sensitivity C-reactive protein and interleukin-6.

The marker lipoprotein-associated phospholipase A2 (Lp-PLA2), belonging to a family of enzymes that hydrolyze phospholipids, may also confer risk of cognitive impairment by increasing the likelihood of stroke and heart disease.

Initial studies aimed at improving cognitive functioning examined pharmacological agents including vasodilators, nosotropics, and antioxidants, but the evidence failed to reveal significant cognitive benefits. Memantine, nimodipine, and propentofylline have each been associated with relatively modest cognitive improvements in patients with vascular dementia, but have demonstrated little change in global functioning and the clinical significance of the cognitive improvements has not been established. Acetylcholinesterase inhibitors, such as galantamine and donepezil, have been associated with improved cognition, global functioning, and activities of daily living.

Prognosis

VCI in the absence of dementia has been associated with an increased risk of vascular dementia, Alzheimer's disease, institutionalization, and mortality. In a large-scale community follow-up study, approximately half of non-dementia VCI patients progressed to dementia over the course of 5 years; of these, 43% were diagnosed with vascular dementia, 35% with AD, 13% with mixed AD, and 9% with unclassified dementia. Compared to VCI patients who did not develop dementia, these incident dementia cases performed worse at baseline on tests of free and cued memory recall and category fluency.

Patients with a history of stroke are at increased risk for developing dementia. It is expected that 20–30% of poststroke patients will develop dementia within 3 months, and an additional 25% of patients will develop dementia over the next 3 years. The mechanisms underlying the development of these delayed cases of dementia are unclear. However, increasing evidence suggests that vascular changes produce a cascade of events (e.g., hypoxia, hypoperfusion, vessel wall thickening, reduced efficiency of perivascular drainage, etc.) leading to increased pathology. In addition, this cascade is thought to contribute to the formation of Alzheimer-type pathology, especially if genetic factors, such as apolipoprotein E, are present.

Future Directions

Because most of the research conducted during the past 2 decades has used diagnostic criteria for vascular dementia, research related to VCI, especially cases without dementia, is relatively limited. The primary needs surrounding VCI are clarification of the cognitive syndrome, diagnostic criteria (e.g., neurobehavioral features, brain-imaging), and related clinical terminology, and development and validation of more sensitive neurocognitive test batteries designed to detect early signs of VCI. More specifically, long-term population-based studies aimed at identifying early precursors to VCI are needed to improve knowledge of the history, stages, and outcomes; inform future clinical trials; decrease rates of cognitive disorders; and reduce public healthcare burden. To promote more internal consistency, researchers studying VCI are encouraged to consult a comprehensive set of guidelines published by the NINDS and the CSN.

Cross References

- ▶ Alzheimer's Dementia
- ▶ Alzheimer's Disease
- ▶ Cortical-Subcortical Loop
- ▶ Cortical Motor Pathways
- ▶ Frontal Temporal Dementia
- ▶ Mild Cognitive Impairment
- ▶ Multi-Infarct Dementia
- ▶ Periventricular White Matter
- ▶ Small Vessel Ischemic Disease
- ▶ Vascular Dementia
- ▶ White Matter

References and Readings

Bowler, J. V. (2007). Modern concept of vascular cognitive impairment: Review. *British Medical Bulletin, 83*, 291–305.

de Haan, E. H., Nys, G. M., & van Zandvoort, M. J. (2006). Cognitive function following stroke and vascular cognitive impairment: Review. *Current Opinion in Neurology, 19*(6), 559–564.

Hachinski, V., Iadecola, C., Petersen, R. C., Breteler, M. M., Nyenhuis, D. L., Black, S. E., et al. (2006). National Institute of Neurological Disorders and Stroke—Canadian Stroke Network Vascular Cognitive Impairment Harmonization Standards. *Stroke, 37*(9), 2220–2241.

Vascular Cognitive Impairment Without Dementia

- ▶ Vascular Cognitive Impairment

Vascular Dementia

Holly Rau, Yana Suchy
University of Utah
Salt Lake City, UT, USA

Synonyms

Atherosclerotic dementia; Binswanger's disease; Cerebrovascular disease; Dementia due to vascular disease/lacunar state; Multi-infarct dementia; Subcortical dementia; Subcortical leukoencephalopathy; Vascular cognitive impairment; Vascular disorders

Definition

Vascular dementia (VaD) refers to a progressive decline in cognitive functioning caused by cerebrovascular brain damage. The dementia associated with vascular factors is different from the prototypic Alzheimer's dementia in that executive impairments and psychomotor slowing, rather than memory per se, constitute the most prominent deficits. Syndromes related to different vascular mechanisms include VaD due to hemorrhagic lesions, lacunar lesions, or a single strategic infarct; multi-infarct dementia; subcortical vascular dementia; mixed dementia; Binswanger disease; and mild vascular cognitive impairment. Various vascular pathologies contribute to a broad continuum of neurocognitive impairments, with VaD lying at one end of this continuum.

Historical Background

Cerebrovascular disease and dementia were described as different syndromes until the late twentieth century, when Mayer-Gross observed vascular etiology in nearly 50% of dementia patients. The broad term vascular dementia, coined by Loeb in 1985, was an extension of Hachinski's use of the more specific term multi-infarct dementia introduced in 1974.

Current Knowledge

VaD is characterized by cognitive impairments, resulting from either extracranial or intracranial vascular disease. Extracranial vascular disease, resulting from blockage to one of the three major cerebral arteries, typically produces a more abrupt decline in cognitive function. Conversely, intracranial vascular disease tends to progress more slowly, and is associated with vascular damage to the cerebral cortex, subcortical and brainstem structures, and the white matter. The two primary mechanisms contributing to the development of VaD are large artery pathology and small vessel disease.

Large artery vascular pathology can produce cerebrovascular accidents (CVAs), or strokes, which are classified according to etiology as either infarctions (tissue death due to lack of oxygen) or cerebral hemorrhages (bleeds). Infarctions are the most common cause of CVAs and occur typically secondary to either a thrombotic or embolic vascular occlusion of major cerebral vessels (e.g., carotid arteries and the distributions of anterior, middle, and posterior cerebral arteries). Thrombotic occlusions develop slowly over time as a result of plaques on vessel walls. While cholesterol is the most common source of plaques, other sources exist, including by-products of tumors or infections. Plaques have a tendency to trap coagulated blood, which eventually obstructs blood flow. In contrast to thrombotic occlusion, embolic occlusions occur more rapidly, and are caused by a plaque particle that breaks off and migrates via circulation to a smaller vessel where it becomes lodged. Cerebral hemorrhages are considerably less common than infarction, and typically result from an aneurysm rupture, or a rupture of small deep-penetrating arteries due to hypertension.

The second mechanism contributing to VaD is small vessel cerebrovascular disease. There are two primary pathophysiological processes that are associated with small vessel disease. The first process involves microatheromata (micro-occlusions in the orifices of the penetrating arteries) secondary to atherosclerosis. The second process is associated with hypertension and consists of loss in the vessel elasticity due to thickening and narrowing of blood vessels, leading to a chronic decrease in oxygen delivery to the periventricular white matter. These processes result in expansion of the Virchow–Robin spaces (e.g., enlarged perivascular spaces), perivascular parenchymal rarefaction, and gliosis. Two major syndromes that result from small vessel cerebrovascular disease are (1) lacunar state, which consists of multiple subcortical infarctions and (2) subcortical leukoencephalopathy, which consists of diffuse white matter damage.

However, other causes of VaD exist, including cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), cerebral amyloid angiopathy, and cerebral vasculitis.

Prevalence Rates, Incidence Rates, and Demographics

In the USA, Australia, and Latin America, VaD is considered the second most common cause of dementia after Alzheimer's disease, accounting for approximately 15–20% of dementia cases. In other countries, including Sweden, Russia, and Asia, VaD has been reported as the most common cause of dementia.

VaD is seen in men more often than women, and is more prevalent among African Americans, likely due to higher rates of hypertension in this group. Consistent with the increase of cerebrovascular disease in later life, older adults are at greater risk. The prevalence rate of 1% at age 60 is thought to double every 5 years, with rates reaching 30–50% by age 85.

Symptoms/Presentation

The clinical presentations of VaD are heterogeneous and depend on the type, number, volume, and location of vascular injuries. For example, patients who have suffered a single event, such as a CVA due to hemorrhage or a strategically located infarction, demonstrate sudden onset of symptoms with more rapid declines accompanied by acute focal neurologic signs such as hemiplegia, aphasia, apraxia, or hemispatial neglect. In contrast, the combined effect of multiple smaller or apparently silent cortical infarctions, or multiple subcortical infarctions such as those seen in lacunar state, produces a stepwise decline, where a plateau of initial deficits is followed by sudden onset of additional deficits. Impairments associated with lacunar state include shuffling gait or imbalance, dysarthria and/or dysphagia, and pseudobulbar palsy. Finally, in the case of small vessel disease, cognitive changes are usually subtle in initial presentation with a more gradual and slow progression over time and typically occur in the absence of defined events.

Neurocognitive Impairments

The constellation of neurocognitive impairments depends on the type of pathological process. For patients with infarction or hemorrhages due to large vessel disease, impairments tend to be focal and are a function of the extent and location of damage. For example, syndromes of aphasia are associated with vascular damage to the perisylvian regions of the dominant hemisphere; visual disorders (e.g., ▶ alexia and visual agnosia) are associated with vascular damage to the occipitotemporal or occipitoparietal regions; and amnesia is associated with vascular damage to diencephalic or medial temporal structures.

In contrast, patients with extensive deep white matter disease may exhibit impaired psychomotor speed, executive deficits, and memory retrieval problems. Early symptoms of VaD are distinct from those associated with Alzheimer's dementia (AD) in that executive dysfunction and psychomotor slowing, rather than memory, are most prominent. Compared to patients with AD, patients with VaD may have better delayed recognition memory and fewer recall or recognition intrusions, but poorer phonemic fluency, increased perseverative behaviors, slower processing speed, decreased working memory capacity, and greater executive deficits.

Neuropsychological Assessment

In order to assess both the core executive and psychomotor features as well as the broader range of cognitive deficits associated with VaD, the workgroup of the National Institute of Neurological Disorders and Stroke (NINDS) and the Canadian Stroke Network (CSN) recommends that four cognitive domains be tested: executive/processing, speed/activation, language, visuospatial functions, and memory (Hachinski et al., 2006). Because of the disruption to frontal-subcortical circuits, patients with VaD are expected to perform more poorly across cognitive domains on tasks having an executive component (e.g., ▶ phonemic fluency, visual organization, and ▶ procedural memory). Neurocognitive tests often used in this context include semantic and phonemic fluency, digit span, digit symbol-coding (subtest from the *Wechsler Adult Intelligence Scale* – third edition), trail making or maze tests, verbal learning tests, clock drawing, complex figure drawing, and confrontation naming.

Neuropsychiatric Symptoms

While VaD refers to a decline in cognitive functioning of sufficient severity to impair functional activities of daily living, most patients, whether affected by small or large vessel disease, also develop comorbid psychiatric symptoms that can complicate the treatment and course of the disease. Symptoms of depression are quite common,

particularly among patients with infarctions in the left frontal lobe. If left untreated, depression can further exacerbate cognitive and functional impairments, as well as increase risk for suicide. While aggressive treatment of depression can improve daily functioning and quality of life, diagnosis can be difficult because patients may not endorse depressed mood. Therefore, providers often rely on behavioral cues, such as social withdrawal or decreased psychomotor activity, when establishing a diagnosis.

Patients with executive deficits may demonstrate socially inappropriate or reckless behaviors (e.g., undressing in public, cursing, and careless driving). Agitation is also frequent, with approximately 15% of community-dwelling dementia patients exhibiting some form of agitated behavior (e.g., repetitive behaviors, hitting, and screaming). Psychotic symptoms, including hallucinations and especially delusions, are common, with prevalence rates estimated at 9–40%.

Circadian rhythm disturbance is common in dementia, and VaD patients are frequently observed to have increased disruption of sleep (e.g., restlessness and nocturnal wandering) and lower subjective sleep quality. Over time, chronic sleep disturbances may disrupt the natural sleep–wake cycle, causing patients, especially those in more advanced stages of dementia, to be awake during the night and asleep during the day. Sundowning, an associated phenomenon characterized by increased agitation and confusion during the late afternoon or early evening, may also be present. While sleep disturbance is common, no evidence has linked the extent of sleep disruption with severity of cognitive decline.

Diagnosis

When evaluating patients for VaD, evidence must establish the presence of both dementia and vascular etiology. Criteria used to diagnose VaD vary somewhat based on the source and can be found in the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, text revision (DSM-IV-TR) criteria; the *International Classification of Diseases*, tenth edition criteria; the National Institute of Neurological Disorders and Stroke – Association International pour la Recherché at L'Enseignement en Neurosciences (NINDS-AIREN) criteria; the Alzheimer's Disease Diagnostic and Treatment Center criteria, and the Hachinski Ischemic Score.

A typical evaluation involves obtaining a thorough history of the patient, including the degree and types of cognitive difficulties (e.g., memory, visual, or language impairments), the presence of vascular risk factors (e.g., hypertension and smoking), and evidence of prior vascular damage (e.g., strokes, extensive white matter infarcts, and focal damage). Although the Folstein mini-mental state examination and the cognitive abilities screening instrument are mental status screens commonly used by physicians to document and monitor cognitive decline, neuropsychological evaluation is recommended in early stages of dementia, or when a differential diagnosis needs to be made between vascular and other types of dementia, including Alzheimer's dementia, Lewy body dementia, frontotemporal dementia, dementia due to head trauma, and others.

Treatment/Prevention

No treatments are currently available to halt, reverse, or repair the effects of VaD. However, detection, diagnosis, and management of underlying risk factors can minimize the effects. History of stroke is one considerable risk factor. The prevalence rate of dementia is nine times higher in stroke patients, and approximately 25–30% of stroke patients develop new onset dementia within 1 year. Other risk factors that may contribute to stroke or coronary ischemic damage include cerebrovascular disease, hypertension, atrial fibrillation, coronary artery disease, atherosclerosis, congestive heart failure, diabetes, smoking, alcoholism, and high cholesterol. Preventative efforts generally combine pharmacological (e.g., ► cholinesterase inhibitors, memantine, antiplatelet, and hemorrheologic agents) and behavioral interventions (e.g., exercise, diet, smoking cessation, and alcohol reduction).

Prognosis

Patients with VaD are less likely to survive than patients with Alzheimer's disease, which is attributable to the coexistence of other atherosclerotic diseases. Life expectancy is shortened by approximately 50% in men, in patients with lower education, and in patients demonstrating greater impairments on neuropsychological tests. Death is typically caused by complications associated with dementia or cardiovascular disease. Mortality rates are substantially higher in patients who have had a stroke; the 5-year survival rate is 39% compared with 75% for age-matched controls.

Cross References

- ▶ Alzheimer's Dementia
- ▶ Aphasia
- ▶ CADASIL
- ▶ Leukoaraiosis
- ▶ Senile Dementia
- ▶ Small Vessel Ischemic Disease

References and Readings

Alagiakrishnan, K. (2007). *Vascular dementia*: http://www.emedicine.com/med/TOPIC3150.HTM

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

American Psychiatric Association (1997). Practice guideline for the treatment of patients with Alzheimer's disease and other dementias of late life. *American Journal of Psychiatry, 154*(supp. 5), 1–39.

Chui, H. D. (2005). Neuropathology lessons in vascular dementia: Review. *Alzheimer's Disease and Associated Disorders, 19*(1), 45–52.

Duthie, E. H., Katz, P. R., & Malone, M. (Eds.). (2007). *Practice of geriatrics* (4th ed.). Philadelphia: Saunders.

Hachinski, V., Iadecola, C., Petersen, R. C., Breteler, M. M., Nyenhuis, D. L., Black, S. E., et al. (2006). National Institute of Neurological Disorders and Stroke—Canadian Stroke Network Vascular Cognitive Impairment Harmonization Standards. *Stroke 37*(9), 2220–2241.

Strub, R. (2003). Vascular dementia review. *Southern Medical Journal, 96*(4), 363–366.

University of California at San Francisco Memory and Aging Center (2008). *Vascular dementia*: http://memory.ucsf.edu/Education/Disease/vad.html

Vascular Disorders

- ▶ Stroke
- ▶ Vascular Cognitive Impairment
- ▶ Vascular Dementia

Vascular Endothelial Proliferation

- ▶ Endothelial Proliferation

Vascular Malformations

Elliot J. Roth
Northwestern University
Chicago, IL, USA

Synonyms

Arteriovenous malformations; AVMs

Definition

Vascular malformations are congenital birth defects that affect arteries or veins or both.

Current Knowledge

Various types of vascular malformations exist, including arteriovenous malformations, cavernous angiomas, venous malformations, and telangiectases. Although they are present at birth, they often do not present clinically until much later. They can cause clinical syndromes because they prevent the normal flow of sufficient oxygen-rich blood from filling the capillaries that supply tissues of the body, and also allow a buildup of waste products in the tissue that would normally be eliminated by the veins. Symptoms depend on the type and location of the malformation. Cerebral arteriovenous malformations have the most significant clinical implications, and can cause seizure, headache, and neurological consequences of hemorrhagic stroke, including paralysis, loss of vision, and aphasia.

Diagnosis is made based on neuroimaging studies and cerebral angiography. Many vascular malformations require only physician monitoring for signs of cerebral hemorrhage, and never require formal treatment. When it is managed surgically, the arteries that feed the malformation are tied off and removed. The malformation also can be embolized using a chemical to induce a clot in the abnormal vessels. Finally, radiosurgery can be performed using highly specialized technology, which provides highly focused radiation into the malformation. Often, arteriovenous malformations are treated with combinations of these strategies.

Cross References

- ▶ Arteriovenous Malformation (AVM)
- ▶ Hemorrhagic Stroke

- ▶ Intracerebral Hemorrhage
- ▶ Intracranial Hemorrhage
- ▶ Intraventricular Hemorrhage
- ▶ Subarachnoid Hemorrhage

References and Readings

Ondra, S. L., Troupp, H., George, E. D., & Schwab, K. (1990). The natural history of symptomatic arteriovenous malformations of the brain: A 24 year follow-up assessment. *Journal of Neurosurgery, 73*, 387–391.

Truwit, C. L. (1992). Venous angioma of the brain: History, significance, and imaging findings. *American Journal of Roentgenology, 159*, 1299–1307.

Uchino, A., Hasuo, K., & Matsumoto, S. (1996). Cerebral venous angiomas associated with hemorrhagic lesions. Their MRI manifestations. *Clinical Imaging, 20*, 157–163.

Vascular Mild Cognitive Impairment

- ▶ Vascular Cognitive Impairment

Vascular Ultrasound

- ▶ Blood Flow Studies

Vasculitis

Elliot J. Roth
Northwestern University
Chicago, IL, USA

Synonyms

Angiitis; Arteritis

Definition

Vasculitis, also known as angiitis or arteritis, is a group of diseases characterized by inflammation and subsequent damage of the walls of blood vessels.

Current Knowledge

The causes of these conditions are not fully understood, but they tend to be autoimmune in origin, and as such, involve abnormalities of the immune system and inflammatory changes of the vascular wall. Examples include Kawasaki disease, Behcet's disease, polyarteritis nodosa, Wegener's granulomatosis, cryoglobulinemia, Takayasu's arteritis, and giant cell arteritis. At times, vasculitis accompanies certain infections (such as hepatitis and herpes zoster), use of certain chemicals (such as cocaine), cancers (such as lymphoma and multiple myeloma), and collagen vascular diseases (such as rheumatoid arthritis and systemic lupus erythematosus). Symptoms reflect the specific organ involved.

Cerebral vasculitis is not common, but has significant implications. It is seen in systemic lupus erythematosus ("lupus cerebritis"), cocaine use, and other conditions, or can occur on an isolated basis ("isolated cerebral angiitis"). It can cause both hemorrhagic and ischemic strokes because of the vascular wall abnormalities. Diagnosis is made by the presentation of symptoms, and also by a classic finding on cerebral angiography of a "beading" appearance of the affected blood vessels. Treatment varies depending on the condition, but use of steroids and other anti-inflammatory agents are common.

Cross References

- ▶ Cerebral Angiitis
- ▶ Collagen Vascular Disease
- ▶ Lupus Cerebritis
- ▶ Vasospasm

References and Readings

Rehman, H. U. (2000). Primary angiitis of the central nervous system. *Journal of the Royal Society of Medicine, 93*, 586–588.

Scolding, N. J., Jayne, D. R., Zajicek, J. P., Meyer, P. A., Wraight, E. P., & Lockwood, C. M. (1997). Cerebral vasculitis – recognition, diagnosis and management. *The Quarterly Journal of Medicine, 90*, 61–73.

Vasogenic Edema

- ▶ Cerebral Edema

Vasospasm

Elliot J. Roth
Northwestern University
Chicago, IL, USA

Definition

Cerebral vasospasm is the sudden acute narrowing of cerebral blood vessels that can occur 3–4 days after the onset of a subarachnoid hemorrhage (SAH) stroke resulting from a rupture of a cerebral aneurysm.

Current Knowledge

By causing reduced cerebral blood flow to the affected area, it causes cerebral ischemia, and consequently vasospasm is a significant cause of morbidity and mortality following SAH. Arterial vasospasm is seen in 40–70% of SAH patients on cerebral angiogram, but symptoms occur in about 20–30%. It can cause confusion, reduced consciousness, and ultimately coma and death. When it is less severe, neurological recovery occurs as the arterial narrowing resolves. Treatment involves administration of selected medications and fluids to reduce the vasospasm.

Cross References

► Subarachnoid Hemorrhage
► Vasculitis

References and Readings

Dietrich, H. H., & Dacey, R. G. (2000). Molecular keys to the problems of cerebral vasospasm. *Neurosurgery, 46*, 517–530.
Weir, B. (1995). The pathophysiology of cerebral vasospasm. *British Journal of Neurosurgery, 9*, 375–390.

VCI

► Verbal Comprehension Index

Vegetative Equivalent

► Masked Depression

Vegetative State (Persistent)

Jacob Kean
Indiana University School of Medicine
Indianapolis, Indiana, USA

Synonyms

Coma vigile

Short Description or Definition

A vegetative state (VS) is a type of unconsciousness in which the patient is capable of wakefulness but not awareness. It is characterized by intermittent and sometimes prolonged wakeful eye opening in the absence of cognitive function, evidenced by lack of meaningful response or purposeful activity.

Categorization

Jennett (1997) noted that patients who were unconscious but demonstrated wakefulness have been described in the literature using terms, such as *apallic syndrome* and *coma vigile*; the term *vegetative* was applied to these patients beginning in 1963. Persistent vegetative state (PVS) was a term coined by Jennett and Plum (1972) in an attempt to bring some order to the inconsistent clinical description of patients who were unconscious but who had emerged from coma, demonstrated by eye opening and the presence of a discernable wakeful state. Jennett and Plum stated that the term was not meant to imply irreversibility, but the condition was later interpreted to be irreversible in certain circumstances by The Multi-Society Task Force on PVS (1994). Reports of late recovery from PVS prompted The Aspen Neurobehavioral Conference Work Group to recommend that the term PVS be abandoned. Currently, the VS that continues for longer than one month is commonly labeled as *persistent* despite the

recommendation by the Aspen Work Group that the term be disregarded (Giacino, Ashwal, Childs, Cranford, Jennett, Katz, D. I., et al., 2002).

Physically, the patient in VS is often not only in a decorticate position (arms flexed and legs fully extended) but also sometimes in a decerebrate position (both arms and legs fully extended). A positive clinical feature of VS includes periods of wakeful eye opening without sustained visual tracking. Reflexive responses, including orienting or a startle response to a visual or auditory stimulus, may occur without sustained pursuit of or fixation on the stimulus. For example, a patient may respond to a change in room lighting or the introduction of a light or object into the visual field, but the response will quickly cease. Such a response is reflexive and not indicative of awareness. Reflexive response may also be evidenced in motor movements, which are nonpurposeful and sometimes groping. For example, a patient may demonstrate nonpurposeful movement of the arm, causing the hand to rub against an object in the patient's personal space and grasp reflexively. Patients in VS may respond to pain, but like response to other stimuli, the response to pain is inconsistent and not commonly thought to be realized by the patients at a conscious level (but see Howsepian, 1996 and Borthwick, 1996 for opposing views). The patient in VS may have verbal output, including occasional laughter or other emotional behavior, although the sounds are generally unarticulated vowel-like groans. Moreover, patients in VS may demonstrate a swallow reflex and adequately manage saliva but not the coordinated management of food or drink necessary for safe oral intake.

The VS is a state of unconsciousness, which differs from coma in that it includes distinguishable periods of wakefulness and sleep, reflexive response to auditory or visual stimuli, and occasional nonpurposeful movement. Jennett and Plum (1972) supported a clear distinction between the unconscious states of coma and VS and the states of partial consciousness, such as delirium, stupor, obtundation, and the minimally conscious state. Unlike patients in VS, those in acute and chronic partially conscious states often demonstrate impaired cognition and perception, but have variable awareness of themselves and the environment.

Jennett (2004) cautioned that family members and caregivers may misperceive the reflexive motor responses or other reflexive behavior as evidence of returning consciousness. Medical personnel need to be cognizant of changes that indicate consciousness. Although some patients do regain consciousness, careful observation commonly reveals that responses misinterpreted by caregivers to be consistent actually demonstrate no relationship with a stimulus. The perceptions of family members have medico–legal significance, discussed by Wijdicks (2006) in a discussion of the well-publicized cases of Terry Wallis and Terri Schiavo.

Epidemiology

Jennett (2004) examined the few available epidemiological studies of VS. Little information is available regarding the epidemiological aspects of VS because it is not recognized by the International Classification of Diseases (ICD). The range of prevalence of VS is between 5 and 140 cases per million population (PMP). The Multi-Society Task Force on PVS (1994) endorsed an estimate of 56–140 PMP for the United States. Furthermore, Jennett (2004) extrapolated data from available studies and estimated the incidence of acute VS at 3 months post brain injury at 8, 27, 40 cases PMP for UK, US, and France, respectively. The incidence of VS decreases at time points further post-injury as patients die or emerge from the VS.

Natural History, Prognostic Factors, Outcomes

The natural history of VS is highly variable for the first year in terms of rate and extent of recovery. Potential outcomes range from death to independence, with the majority of patients remaining severely disabled and dependent and with prognosis for children consistently better than prognosis for adults. Whyte and colleagues (2005) reported that neuroimaging findings and injury characteristics were not significant predictors of functional status in a study of 124 patients in minimally conscious or vegetative states. Moreover, Whyte and colleagues found that time post-injury, current level of functioning, and rate of functional change predicted the degree of functional improvement in patients in either minimally conscious or vegetative states. The most comprehensive data to date come from review conducted by The Multi-Society Task Force on PVS (1994), who reported outcome for 754 patients at 1, 3, 6, and 12 months post-injury. Table 1 below shows the percentage of patients in VS at 1, 3, and 6 months post-insult that were conscious and independent at one year post-insult.

The Task Force determined that VS could be declared permanent when caused by non-traumatic injuries if it persisted longer than 3 months and declared permanent

Vegetative State (Persistent). Table 1

	Conscious	Independent
1 month	43	19
3 months	30	14
6 months	13	4

when caused by traumatic injuries if it persisted longer than 12 months. This recommendation was superseded by a recommendation by the Aspen Work Group (Giacino et al., 2002), who suggested that factors such as the nature and extent of injury and the time post-insult are more constructive means of prognostication.

Evaluation

The diagnosis of patients in VS has been most influenced by criteria developed by The Multi-Society Task Force (1994). Those criteria are as follows:

1. No evidence of awareness of themselves or their environment; they are incapable of interacting with others
2. No evidence of sustained, reproducible, purposeful, or voluntary behavioral responses to visual, auditory, tactile, or noxious stimuli
3. No evidence of language comprehension or expression
4. Intermittent wakefulness manifested by the presence of sleep–wake cycles
5. Sufficient preserved hypothalamic and brainstem autonomic functions to survive if given medical and nursing care
6. Bowel and bladder incontinence
7. Variably preserved cranial nerve (pupiliary, oculocephalic, corneal, vestibulo-ocular, and gag) and spinal reflexes

Wijdicks and Cranford (2005) suggested that in addition to basic neurological examination of common reflexes, visual and auditory orienting and tracking can be assessed easily with the introduction of stimuli and careful observation for consistency of response. Jennett (2004) noted that vegetative patients do not habituate (i.e., produce less and less response) to a repetitious stimulus. The JFK Coma Recovery Scale-Revised (Giacino, Kalmar, & Whyte,2004) is a tool designed to detect neurobehavioral subtleties that differentiate patients in states of dysconsciousness. Giacino and Whyte (2005) detail procedures for individual quantitative assessment of patients in states of impaired consciousness. The Aspen Work Group (Giacino et al., 2002) suggested utilizing conditions in repeated evaluations, which maximize arousal, minimize distractions, and which are sensitive to the physical limitations and anecdotal reports of caregivers and families.

Treatment

Wijdicks and Cranford (2005) cautioned that medical management of patients in states of impaired consciousness is complex. Specialized care for patients in vegetative and minimally conscious states depends on the availability of appropriate facilities and, in privatized systems of medical care, adequate payer support. Rehabilitation efforts during acute states of impaired consciousness are directed toward preventing contractures, ensuring proper nutrition, and maintaining integrity of the skin. If the VS persists, respiratory and urinary tract infections can be troublesome and require prompt attention. Lombardi and colleagues (2002) conducted a Cochrane review of programs designed to enhance the rate or extent of recovery through sensory stimulation. The results of the review indicated that none of the available studies offers valid results that support this type of intervention for patients in unconscious states (i.e., VS and coma).

Dopaminergic agents have been used in the majority of pharmacological studies with severe brain injury, although not specifically with patients who have clearly defined disturbances of consciousness. One longitudinal observational study of 124 patients in a minimally conscious or vegetative state suggested that amantadine hydrochloride was associated with improved functional status at 16 weeks post-injury, whereas dantrolene was associated with poorer recovery (Whyte et al., 2005). A Cochrane review of the use of psychostimulants after brain injury concluded that there was insufficient evidence to recommend their use to improve acute recovery (Forsyth & Jayamoni, 2003). In both vegetative and minimally conscious states, the likelihood of functional improvement diminishes as time passes (Giacino et al., 2002). Neuropsychological support for families and caregivers is important throughout the duration of the patient's recovery from brain injury (Rotundi, Sinkule, Balzer, Harris, & Moldovan, 2007). Jennett (2004) discussed the issues families face at the time when rehabilitative efforts end, including decisions about cardiopulmonary resuscitation, antibiotics, dialysis, and artificial nutrition and hydration.

Cross References

- ▶ Coma
- ▶ Decerebrate Posturing
- ▶ Decorticate Posturing
- ▶ Minimally Conscious State
- ▶ Stupor

References and Readings

Borthwick, C. J. (1996). The permanent vegetative state; ethical crux, medical fiction? *Issues in Law & Medicine, 12*, 167–185.

Forsyth, R., & Jayamoni, B. (2003). Noradrenergic agonists for acute traumatic brain injury. *Cochrane Database Systematic Review, 1*, CD003984.

Giacino, J. T., Ashwal, S., Childs, N., Cranford, R., Jennett, B., Katz, D. I., et al. (2002). The minimally conscious state: Definition and diagnostic criteria. *Neurology, 58*, 349–353.

Giacino, J. T., Kalmer, K., & Whyte, J. (2004). The JFK Coma Recovery Scale – Revised: Measurement characteristics and diagnostic utility. *Archives of Physical Medicine and Rehabilitation, 85*, 2020–2029.

Giacino, J. T., & Whyte, J. (2005). The vegetative and minimally conscious states: Current knowledge and remaining questions. *Journal of Head Trauma Rehabilitation, 20*, 30–50.

Howsepian, A. A. (1996). The 1994 Multi-Society Task Force consensus statement on the persistent vegetative state; A critical analysis. *Issues in Law & Medicine, 12*, 3–29.

Jennett, B. (1997). A quarter century of the vegetative state: An international perspective. *Journal of Head Trauma Rehabilitation, 12*, 1–12.

Jennett, B. (2004). *The vegetative state*. Cambridge: Cambridge University Press.

Jennett, B., & Plum, F. (1972). Persistent vegetative state after brain damage. A syndrome in search of a name. *Lancet, 1*, 734–737.

Lombardi, F., Taricco, M., De Tanti, A., Telaro, E., & Liberati, A. (2002). Sensory stimulation of brain-injured individuals in coma or vegetative state: Results of a Cochrane systematic review. *Clinical Rehabilitation, 16*, 464–472.

The Multi-Society Task Force on the Persistent Vegetative State. (1994). Statement on medical aspects of the persistent vegetative state. *New England Journal of Medicine*, 330, 1499–1508, 1572–1579.

Rotundi, A. J., Sinkule, J., Balzer, K., Harris, J., & Moldovan, R. (2007). A qualitative needs assessment of persons who have experienced traumatic brain injury and their primary family caregivers. *The Journal of Head Trauma Rehabilitation, 22*, 14–25.

Whyte, J., Katz, D., Long, D., Dipasquale, M. C., Polansky, M., Kalmar, K., et al. (2005). Predictors of outcome in prolonged posttraumatic disorders of consciousness and assessment of medication effects: a multicenter study. *Archives of physical Medicine and Rehabilitation, 86*, 453–462.

Wijdicks, E. F. M. (2006). Minimally conscious state vs persistent vegetative state: The case of Terry (Wallis) vs the case of Terri (Schiavo). *Mayo Clinic Proceedings, 9*, 1155–1158.

Wijdicks, E. F. M., & Cranford, R. E. (2005). Clinical diagnosis of prolonged states of impaired consciousness in adults. *Mayo Clinic Proceedings, 8*, 1037–1046.

Venlafaxine

John C. Courtney[1], Cristy Akins[2]
[1]Children's Hospital of New Orleans
New Orleans, LA, USA
[2]Mercy Family Center
Metarie, LA, USA

Generic Name

Venlafaxine

Brand Name

Effexor, Effexor XR

Class

Serotonin norepinephrine reuptake inhibitor

Proposed Mechanism(s) of Action

Increases serotonin and norepinephrine by blocking both serotonin and norepinephrine reuptake pumps.

Indication

Depression, generalized anxiety disorder, and social anxiety disorder.

Off Label Use

Panic disorder, posttraumatic stress disorder, and premenstrual dysphoric disorder.

Side Effects

Serious

Seizures, hypomania, and suicidal ideation.

Common

Hyponatremia, headache, nervousness, insomnia, sedation, nausea, diarrhea, appetite disturbance, sexual dysfunction, sweating, asthenia, and syndrome of inappropriate antidiuretic hormone secretion.

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

Venous Thromboembolism

► Venous Thrombosis

Venous Thrombosis

Elliot J. Roth
Northwestern University
Chicago, IL, USA

Synonyms

Deep venous thrombosis; DVT; Venous thromboembolism; VTE

Definition

Venous thrombosis occurs when a blood clot forms in a vein.

Current Knowledge

Venous stasis, vessel wall injury, and hypercoagulable state, collectively known as Virchow's Triad, contribute to the development of venous thrombosis. Deep vein thrombosis (DVT) typically begins in the deep veins of the calf, and about 20% will propagate proximally. A small proportion of proximal DVTs may embolize to the pulmonary circulation or elsewhere, which in some cases, can cause death from massive pulmonary embolism (PE). DVT is common in hospitalized patients, and incidence estimates are 30–90% among people with recent stroke. It is associated with many other conditions that limit mobility or increase hypercoagulability, including spinal cord injury, traumatic brain injury, cancer, and prolonged bedrest for other illnesses. Recent research has demonstrated the effectiveness of prophylactic anticoagulation and mobilization to prevent DVT and pulmonary embolism in selected patients. Treatment consists of administration of anticoagulants.

Cross References

► Anticoagulation
► Antiplatelet Therapy
► Central Venous Thrombosis
► Heparin
► Thrombosis
► Warfarin (Coumadin)

References and Readings

Hirsh, J., Guyatt, G., Albers, G. W., Harrington, R., & Schünemann, H. J. (2008). Executive summary: Antithrombotic and thrombolytic therapy, 8th edition: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest, 133*, 71S–105S.

Snow, V., Qaseem, A., Barry, P., et al. (2007). Management of venous thromboembolism: A clinical practice guideline from the American College of Physicians and the American Academy of Family Physicians. *Annals of Internal Medicine, 146*, 204–210.

Ventral Posterior Lateral Nucleus

John E. Mendoza
Tulane University Medical Center
New Orleans, LA, USA

Definition

Specific somatosensory relay nucleus of the thalamus. The major input into this nucleus is via the medial lemniscus and spinal thalamic tracts, which carry information regarding proprioception, both fine and crude touch, vibratory sensations, pain, and temperature from the arms, legs, trunk, and scalp. In turn, the major cortical projection area for this nucleus is to the primary somatosensory cortex (Brodmann's areas 3, 1, 2).

Ventral Posterior Medial Nucleus

John E. Mendoza
Tulane University Medical Center
New Orleans, LA, USA

Definition

Specific somatosensory relay nucleus of the thalamus. The major input into this nucleus is via the ventral and dorsal trigeminothalamic tracts and the mesencephalic tract of V, which carry information regarding proprioception, both fine and crude touch, vibratory sensations, pain, and temperature from the face and forehead. In turn, the major cortical projection area for this nucleus is to the facial area of the primary somatosensory cortex (Brodmann's areas 3, 1, 2).

Ventral Spinothalamic Tract

► Spinothalamic Tract

Ventral Stream

► Ventral Visual Pathway

Ventral Tegmental Area

► Mesolimbic Dopaminergic Projections
► Ventral Tegmental Area of Midbrain

Ventral Tegmental Area of Midbrain

Michelle L. Block
Virginia Commonwealth University
Richmond, VA, USA

Synonyms

Ventral tegmental area

Definition

The ventral tegmental area (VTA) is comprised of multiple nuclei and is located in the mesencephalon, dorsomedial to the substantia nigra and ventral to the red nucleus. The VTA is comprised of dopamine, glutamate, and GABA neurons and is an essential component of both the mesolimbic pathway and the mesocortical pathway.

Current Knowledge

The mesolimbic dopamine projection from the VTA to the nucleus accumbens has been implicated in motivation, emotion, the positive symptoms of schizophrenia, and the rewarding effects of drugs of abuse. The dopaminergic neurons in the mesocortical pathway, which originates from the VTA and projects to the cortex, are associated with motivation, attention, planning, social behavior, and the negative symptoms of schizophrenia. For more detailed information, see Ikemoto (2007).

Cross References

► Limbic System
► Nucleus Accumbens

References and Readings

Ikemoto, S. (2007). Dopamine reward circuitry: Two projection systems from the ventral midbrain to the nucleus accumbens-olfactory tubercle complex. *Brain Research Reviews, 56*(1), 27–78.

Ventral Visual Pathway

Giulia Righi, Jean Vettel
Brown University
Providence, RI, USA

Synonyms

'What system'; Ventral stream

Structure

The ventral visual pathway is a functional stream involved in the visual recognition of objects. The anatomical substrates to the ventral visual pathway were initially identified in macaque monkeys by Mishkin and Ungerleider (1982). They observed that visual input from primary visual cortex is projected to the inferior temporal cortex (areas TEO and TE) via prestriate cortex (Mishkin, Lewis, & Ungerleider, 1982; Mishkin Ungerleider, & Macko, 1983). An analogous pathway is present in the human brain. This pathway consists of visual input from primary visual cortex V1 relayed through areas V2 and V4, and ultimately projected into the inferior temporal cortex. While areas V1, V2, and V4 are involved in the processing of basic level visual features such as edges, contours, and color, the inferior temporal cortex is suggested to process complex shapes (Ungerleider & Haxby, 1994).

Function

Since the 1960s, researchers had suggested that the visual system could be divided into two separate components, one responsible for object recognition and one responsible for the localization of objects in space (Held, 1968; Schneider, 1969). Mishkin and Ungerleider (1982) provided the first empirical evidence for this intuition, by showing that monkeys with inferior temporal cortex lesions had problems in recognizing objects by their shape (what), while monkeys with parietal lobe lesions had problems processing the location of objects in space (where).

Since the seminal study by Mishkin and Ungerleider, much research has been devoted to understand the function of the ventral visual pathway in the human brain. Lesion studies were the first to suggest that the what/where dissociation observed in monkeys could also be found in brain injury patients, suggesting that the human visual system is organized in a similar manner. Patients with occipito-temporal lesions had difficulties in recognizing a variety of objects (what), while patients with parietal lesions had difficulties in spatial tasks (where), including reaching for objects (Milner & Goodale, 1991; Newcomb et al., 1969, 1987). These findings confirmed the hypothesis that the ventral temporal pathway is involved in the processing and recognition of objects.

While the function of the ventral visual pathway is widely accepted, there is much debate in the literature regarding the functional organization of this pathway. Case studies of patients with different occipito-temporal lesions have hinted that different groups of neurons within the occipito-temporal cortex may be selectively active for specific classes of objects. This idea was motivated by reports of patients showing deficits primarily in the recognition of faces and not other objects, or in the recognition of letters and not faces, or even in the recognition of one or more specific categories of objects (see De Renzi & Saetti, 1997). These case studies motivated a series of investigations of the functional organization of the ventral stream using techniques such as PET and fMRI. Recently, several proposals regarding the functional organization of object representations in occipito-temporal cortex have been put forth (see Grill-Spector, 2004; for a review).

All these proposals agree on the presence of generic-object selective cortex bilaterally in the lateral portion of the occipital lobe (area LO, Malach et al., 1995). These regions are the first portions of cortex activated more strongly by objects as wholes when compared with a large range of texture patterns (Malach et al., 1995). Malach and colleagues have suggested that LO represents an intermediate processing stage that leads to object recognition, but that these areas are not capable of recognizing specific exemplars.

A divergence among opinions emerges with regard to the organization of ventral temporal cortex, considered to be the site where exemplar recognition takes place. Based on the studies of patients with selective recognition impairments, researchers have proposed that ventral temporal cortex may contain object representations that are separable on the basis of the object category; however, the functional principles underlying the separation of

category-specific information is a matter of active debate. Some researchers have suggested that there might be module-like sub-regions within the ventral portion of the temporal cortex that respond selectively to specific object categories such as faces, places, and tools (Aguirre et al., 1998; Epstein & Kanwisher, 1998; Kanwisher et al., 1997; Martin et al., 1996). The modules may be organized based on their ecological importance (often argued in the case of faces) (Kanwisher et al.), or possibly the organization of semantic knowledge related to the class (Martin et al.,1996). A second explanation has focused on the manner of processing that is applied to different object categories. This proposal argues that the functional segregation among object classes in ventral temporal cortex is determined by the computations required to recognize them, such as specialized processing for expert categories where the specific exemplars are recognized quickly and efficiently (Gauthier et al., 1999). Lastly, it has been proposed that the ventral temporal cortex may contain feature-based object topographies. That is, the object categories could be represented by a distributed network of neurons that code for its different visual attributes, with category organization based on the visual similarity among the object categories (Haxby et al., 2000).

Illness

Damage to the ventral visual pathway can produce a set of deficits termed visual agnosias. Visual agnosias are impairments in the perception and/or recognition of objects. These patients have spared basic perceptual abilities, such as acuity, motion detection, and contrast sensitivity, and as such are not cortically blind, but they are greatly impaired in matching, recognizing, and discriminating objects and shapes visually. Visual agnosias have been divided into two types (Farah, 1990; Lissauer, 1890).

The first type has been termed apperceptive, and it is described as primarily a disorder of perception. Apperceptive agnosias have difficulty in creating an accurate and unified percept of what they see. They can perceive parts of an object, but cannot group the parts together to recognize a full object. These patients will usually show very extensive brain damage to the occipital lobes bilaterally (Farah, 1990).

The second type of visual agnosia has been termed associative, and it is primarily a deficit in the association of a visual percept with its stored representation and its meaning. These patients can form a perceptually "normal" representation of what they see, and they are usually able to copy and match objects and shapes; however, they cannot access any stored object representation that is connected to semantic knowledge of the object. These patients have usually damage either bilaterally or unilaterally to more anterior portions of the ventral stream, such as the lateral occipital cortex and the inferior temporal lobes (Farah, 1990). The severity of the impairment is usually correlated with the extent of the lesion. Patients with smaller lesions will have the most trouble in recognizing specific classes of objects (i.e. faces, letters), while they might be able to recognize some object types. Patients with larger lesions will be impaired on the recognition of most types of objects or shapes.

Cross References

- ▶ Dorsal Visual Pathway
- ▶ Visual Agnosia
- ▶ Visual Object Agnosia

References and Readings

Aguirre, G. K. (1999). Face recognition turned upside-down. *Neuron, 22*(1), 5–6.

De Renzi, E., & Saetti, M. C. (1997). Associative agnosia and optic aphasia: Qualitative or quantitative difference? *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior, 33*, 115–130.

Epstein, R., & Kanwisher, N. (1998). A cortical representation of the local visual environment. *Nature, 392*(6676), 598–601.

Farah, M. J. (1990). *Visual agnosia: Disorders of object recognition and what they tell us about normal vision.* Cambridge, MA: MIT Press.

Grill-Spector. (2004). *The functional organization of the ventral visual pathway and its relationship to object recognition.* In N. Kanwisher, & N. J. Duncan (Eds.), J. Oxford: Oxford University Press.

Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2000). The distributed human neural system for face perception. *Trends in Cognitive Sciences, 4*(6), 223–233.

Kanwisher, N., McDermott, J., & Chun, M. M. (1997). The fusiform face area: A module in human extrastriate cortex specialized for face perception. *The Journal of Neuroscience, 17*(11), 4302–4311.

Lissauer, H. (1890). A case of visual agnosia with a contribution to theory. *Archiv fur Psychiatrie, 21*, 222–270.

Malach, R., Reppas, J. B., Benson, R. R., Kwong, K. K., Jiang, H., Kennedy, W. A., et al. (1995). Object-Related activity revealed by functional magnetic resonance imaging in human occipital cortex. *Proceedings of the National Academy of Sciences of the United States of America, 92* (18), 8135–8139.

Martin, C. L., Wiggs, L. G., Ungerleider, L., & Haxby, J. V. (1996). Neural correlates of category-specific knowledge. *Nature, 379*, 649–652.

Mishkin, M., Lewis, M. E., & Ungerleider, L. G. (1982). Equivalence of parieto-preoccipital subareas for visuospatial ability in monkeys. *Behavioural Brain Research, 6*(1), 41–55.

Mishkin, M., Ungerleider, L. G., & Macko, K. A. (1983). Object vision and spatial vision: Two cortical pathways. *Trends in Neurosciences, 6*, 414–417.

Ungerleider, L. G., & Haxby, J. V. (1994). "What" and "where" in the human brain. *Current Opinion in Neurobiology, 4*(2), 157–165.

Ventricles

Randall E. Merchant
Virginia Commonwealth University Medical Center
Richmond, VA, USA

Synonyms

Cerebral ventricles

Definition

The cerebral ventricular system is a set of four fluid-filled structures in the brain that communicate with each other via small-diameter channels.

Current Knowledge

The ventricular system is a set of four fluid-filled structures in the brain: the paired right and left lateral ventricles that lie in each cerebral hemisphere, a third ventricle within the diencephalon, and a fourth ventricle in the hindbrain. The lateral ventricles are connected to the third ventricle via the interventricular foramina (of Monro), and the third ventricle communicates with the fourth ventricle via the cerebral aqueduct. Cerebrospinal fluid (CSF) is produced by the choroid plexuses that are found in all four ventricles and flows from the ventricles into the subarachnoid space surrounding the brain and spinal cord, as well as into the central canal of the cord. A blockage of the communicating channels between the ventricles will cause an abnormal accumulation of CSF within the ventricles upstream of the blockage and ventricular dilation, a condition known as hydrocephalus.

Cross References

► Hydrocephalus

Ventricular Enlargement

Adam Conley
Virginia Commonwealth University Medical Center
Richmond, VA, USA

Synonyms

Ventriculomegaly

Definition

Ventricular enlargement is a brain condition that occurs when the lateral ventricles become dilated.

Current Knowledge

Ventricular enlargement is a brain condition that occurs when the lateral ventricles become dilated. The most common definition uses a volume to brain index value derived from MRI and CT reconstruction of the lateral and third ventricles.

Enlargement of the ventricles may occur for a number of reasons, such as loss of brain volume (due to conditions such as cortical atrophy, traumatic brain injury, or cerebral vascular accident), impaired outflow, or absorption of cerebrospinal fluid from the ventricles. Often, however, there is no identifiable cause. The interventricular foramen may be congenitally malformed, or may have become obstructed by infection, hemorrhage, or rarely by tumor, which may impair the drainage of cerebrospinal fluid, and thus accumulation in the ventricles.

In neonatal patients, this diagnosis is generally found in routine fetal anomaly scans at 18–22 weeks gestation. It is one of the more common abnormal brain findings on prenatal ultrasound, occurring in around 1–2 per 1,000 pregnancies. In many cases of mild ventricular enlargement, there is resolution during the pregnancy.

In adults, ventricular enlargement is associated with disease processes that reduce the brain volume through cortical atrophy. Disease processes such as schizophrenia (Gaser, 2004), Alzheimer's disease (Fox, 2000), bipolar disorder (Elkins, 1995), and multiple sclerosis have been associated with ventricular enlargement. Several studies have suggested a linear relation in disease process and cortical atrophy. Ventricular enlargement is present in a majority of patients with Alzheimer's disease; however,

the presence of ventricular enlargement alone is not diagnostic of any clinical process. Enlargement may be reversible as is seen in cases of anorexia nervosa. Cerebral ventricular enlargement correlates with the degree of malnutrition and is reversible.

The presence of ventricular enlargement in the absence of preexisting conditions should prompt a thorough history and physical, including a family history for any diseases known to involve ventricular enlargement. An MRI of the brain should be obtained to evaluate for underlying cortical disease. Patients with new presentation of ventricular enlargement should be evaluated in conjunction with a neurological specialist. The presence of symptoms of intracranial hypertension such as headache, nausea, emesis, visual disturbances, or papilledema warrants rapid assessment and appropriate referral.

Cross References

► Alzheimer's Disease
► Atrophy
► Hydrocephalus
► Intracranial Pressure
► Multiple Sclerosis
► Ventricles

Ventriculoatrial Shunt (VA Shunt)

► Shunts

Ventriculomegaly

► Ventricular Enlargement

Ventriculoperitoneal Shunt (VP Shunt)

► Shunts

Ventriculopleural Shunt

► Shunts

Ventriculostomy

Susan Ladley-O'Brien
Denver Health Medical Center
Denver, CO, USA

Definition

Measurement of intracranial pressure through an open catheter placed through the skull and right frontal lobe into the lateral ventricle.

Current Knowledge

Intracranial pressure (ICP) monitoring is standard for patients with severe closed head injury. In those patients with a GCS < 9, the neurological examination is not sensitive to a decline in status.

Other monitoring devices include intraparenchymal monitor and an epidural bolt. The advantage of the ventriculostomy is that cerebrospinal fluid may be drained through the catheter to treat elevated pressures, if present.

However, the ventriculostomy is also the most invasive method, has the highest infection rate, and is the most difficult one to place. Occasionally, the ventriculostomy becomes occluded due to severe brain edema.

All ICP devices should generally be changed or removed after 5–7 days due to the risk of infection.

Cross References

► Intracranial Pressure

References and Readings

Bulgar, E. M., Nathens, A. B., Rivara, F. P., Moore, M., MacKenzie, E. J., & Jurkovich, G. J. (2002). Management of severe head injury: Institutional variations in care and effect on outcome. *Critical Care Medicine, 30*, 8.

Lycette, C. A., Doberstein, C., Rodts, G. E., & McBride, D. Q. (2003). Neurosurgical critical care. In F. S. Bongard & D. Y. Sue (Eds.), *Current critical care diagnosis & treatment* (2nd ed.). New York: McGraw-Hill.

Rangel-Castillo, L. (2006). Management of intracranial hypertension. *Critical Care Clinics, 22*(4), 713–732.

Soumitra, R. E. (Ed.), (2009). In Merck Manual Online. Critical care Medicine Approach to the Critically Ill patient Patient Monitoring and Testing. Hagerstown, MD: Lippincott.

Verbal Auditory Agnosia

► Pure Word Deafness

Verbal Comprehension Index

Rael T. Lange
British Columbia Mental Health and Addiction Services
and University of British Columbia
Vancouver, BC, Canada

Synonyms

VCI; Verbal comprehension scale (WAIS-IV)

Definition

The Verbal Comprehension Index (VCI) is a score derived from the administration of selected subtests from the third and fourth edition Wechsler Adult Intelligence Scale (WAIS) and Wechsler Intelligence Scale for Children (WISC). The VCI is designed to provide a measure of verbal acquired knowledge and verbal reasoning.

Current Knowledge

Wechsler Intelligence Scales: The WAIS and WISC are two of the most widely used tests to assess general intellectual ability in adults aged 16 years or above (WAIS) and children aged 6–16 years (WISC). Since the original development of these scales (WAIS, 1955; WISC, 1949), both tests have been revised on several occasions. The most recent revisions were published in 2003 (WISC-IV) and 2008 (WAIS-IV).

History: Until recently, one of the most prominent features of the Wechsler Intelligence Scales (WIS) was the derivation and interpretation of IQ scores. Using this framework, the Full Scale IQ score provided a measure of general intellectual ability that was differentiated by more specific verbal (Verbal IQ) and visuospatial (Performance IQ) skills. However, the diagnostic utility of IQ scores has been questioned for some time because these scores measure a variety of skills and abilities rather than a pure cognitive construct. For example, early research examining the factor structure of the WAIS and WAIS-R demonstrated that there are *at least* three cognitive constructs measured by the subtests, rather than the two cognitive constructs originally conceptualized using the Verbal IQ and Performance IQ scores. Although factor-analytic researchers did not agree on the number of meaningful constructs, three factors consistently emerged and were labeled as Perceptual Organization, Verbal Comprehension, and Freedom from Distractibility/Attention. Regardless of the number of factors, the importance of the multidimensionality of the WIS was quickly recognized, which prompted a number of researchers to develop statistical methods that enabled factor-based interpretation of WAIS-R scores not included in the original test manual (e.g., Atkinson, 1991).

Evolution: Factor-based interpretation of the WIS was first included in the WISC-III (1991). The addition of a new subtest (i.e., Symbol Search) resulted in the introduction of a four factor scoring system, defined by a Verbal Comprehension Index (VCI), Perceptual Organization Index (POI), Freedom from Distractibility Index (FDI), and Processing Speed Index (PSI). The same four factor scoring system was also included in the WAIS-III (1997) following the inclusion of two new subtests (i.e., Symbol Search and Letter-Number Sequencing), with the exception that the FDI was renamed as the Working Memory Index (WMI). For the WISC-III and WAIS-III, the index scores were initially introduced as an "alternative" system for scoring and interpretation that co-existed with the traditional IQ scores, which remained unchanged. However, the recent publication of the WISC-IV and WAIS-IV represents a significant deferment from the Wechsler scale tradition. The Verbal IQ and Performance IQ scores were excluded for the first time and only the Full Scale IQ score was retained. For the first time in WIS history, the interpretation of the WAIS and WISC is largely focused on the Index scores that are believed to provide a more precise measurement of multiple cognitive abilities assessed by these batteries. For the WISC-IV, the index scores include VCI, WMI, PSI, and the renamed POI – Perceptual Reasoning Index (PRI). For the WAIS-IV, the four index scores (now known as "scales") include the Verbal Comprehension Scale, Perceptual Reasoning Scale, Working Memory Scale, and Processing Speed Scale.

Subtest Composition: The core subtests used to derive VCI vary across the WAIS-III/IV and WISC-III/IV. For the WAIS-III/IV, the subtests contributing to the VCI are Vocabulary, Information, and Similarities. For the WISC-III, the same three subtests are used in addition to the Comprehension subtest. For the WISC-IV, the Information subtest was not included in the calculation

Verbal Comprehension Index. Table 1 Core Subtest Composition of VCI

	IN	VO	SI	CO
WAIS-III	•	•	•	
WAIS-IV	•	•	•	
WISC-III	•	•	•	•
WISC-IV		•	•	•

IN = Information; VO = Vocabulary; SI = Similarities; CO = Comprehension

of VCI and includes only the Vocabulary, Similarities, and Comprehension subtests. The core subtests used to derive VCI across the WAIS and WISC batteries and revisions are presented below. Only the core subtests (not supplementary subtests) are shown.

Cross References

- ▶ Freedom from Distractibility (WISC-III)
- ▶ Intelligence
- ▶ Perceptual Organization Index
- ▶ Performance IQ
- ▶ Processing Speed Index
- ▶ Verbal IQ
- ▶ Wechsler Adult Intelligence Scale (All Versions)
- ▶ Wechsler Intelligence Scale for Children
- ▶ Working Memory Index

References and Readings

Atkinson, L. (1991). Some table for statistically based interpretation of WAIS-R factor scores. *Psychological Assessment, 3*(2), 288–291.

Kaufman, A. S., & Lichtenberger, E. O. (2006). *Assessing adolescent and adult intelligence* (3rd ed.). Hoboken, NJ: Wiley.

Sattler, J. M. (2008). *Assessment of children: Cognitive foundations* (5th ed.). San Diego, CA: Sattler Press.

Tulsky, D. S., Saklofske, D. H., & Zhu, J. (2003). Revising a standard: An evaluation of the origin and development of the WAIS-III. In D. S. Tulsky, et al. (Eds.), *Clinical interpretation of the WAIS-III and WMS-III* (pp. 43–92). San Diego, CA: Academic Press.

Verbal Comprehension Scale (WAIS-IV)

- ▶ Verbal Comprehension Index

Verbal Fluency

Janet Patterson
California State University
Hayward, CA, USA

Synonyms

Category fluency; CFL test; COWA; COWAT; F-A-S test; Letter fluency; Phonemic fluency; Semantic fluency; Word fluency

Definition

Verbal fluency is a cognitive function that facilitates information retrieval from memory. Successful retrieval requires executive control over cognitive processes such as selective attention, selective inhibition, 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 (1) semantic fluency, tested by asking the examinee to generate semantic category exemplars (most commonly names of animals); and (2) phonemic fluency, assessed by asking the examinee to generate words beginning with a single letter, most commonly F, A, and S.

Historical Background

Verbal fluency has been studied in healthy and clinical populations since at least the 1940s (e.g., Bousefield & Sedgewick, 1944). Standardized tests of verbal fluency date back to the written fluency test of Thurstone and Thurstone (1962), clearly a method of limited value in assessment of persons with dominant upper extremity limitations such as hemiparesis or psychomotor slowing. This problem was circumvented by the oral verbal fluency measures developed by Borkowski, Benton, and Spreen (1967), early proponents of systematically examining word fluency in persons with brain damage. These authors identified a series of "easy" versus "moderately difficult" letters based on word frequency in English, including the "easy" letters F, A, and S, that became a subtest of the *Neurosensory Center Comprehensive Examination for Aphasia* (Spreen & Benton, 1969) and continue to be used today (e.g., Heaton, Miller, Taylor, &

Grant, 2004). They also presented word fluency data for adults with versus without brain damage, supporting their hypotheses of the utility of word fluency assessment.

Since the initial publication, word fluency tasks have been investigated in detail for normative and descriptive data, and included in assessment tasks. For example, the Controlled Oral Word Association Test (COWAT) appears as part of the *Multilingual Aphasia Examination* (Benton & Hamsher, 1978; Benton, Hamsher, Rey & Sivan, 1994). In addition to literally hundreds of worldwide experimental studies using a broad variety of cues (e.g., animals, first names, colors, fruits, towns, modes of transportation, and letters (C, F, L, S, P, S, N, and F)), there currently are many standardized versions using both phonemic cues (e.g., the *Delis-Kaplan Executive Function System* includes B, H, and R as well as F, A, and S; Delis, Kaplan, & Kramer, 2001) and semantic cues (e.g., ► Western Aphasia Battery [Kertesz, 2006] includes animal naming), and versions exist in multiple languages, including French, Spanish, Chinese, Norwegian, and Flemish (e.g., P, R, and V in French; Cardebat, Doyon, Puel, Goulet, & Joanette, 1990).

Current Knowledge

Correlates of Verbal Fluency

Although studies have produced differing results, in general verbal fluency is correlated with age, increasing through childhood and decreasing in older age. Within-group variability in test scores also increases in older age. As noted by Barry, Bates, and Labouvie (2008), while vocabulary is considered a crystallized ability that may improve throughout the lifespan as knowledge is acquired, verbal fluency requires executive functions such as "the ability to initiate and maintain effort and organize information for retrieval", which is thought to decline with age beginning in midlife. Age effects may be confounded with the impact of motor speed, however, as well as the possibility that individuals with early-stage degenerative diseases or comorbid conditions such as depression were included in the study sample. Test scores also correlate with years of education. Women typically have slightly higher scores than men, although there is some evidence that these differences are related to hormone levels rather than gender per se.

Overall performance with a given stimulus cue is thought to be related to a combination of the number of items available to that person (e.g., via years of education or idiosyncratic specialized knowledge) and the number of words meeting that criterion in a given language (Cardebat et al., 1990). Thus, for example, the category of *fruits* yields fewer items than *animals* in English, and the letter *Z* yields fewer items than the letter *F*. As time progresses during the task, adults have been observed to generate less typical exemplars on semantic tasks and lower-frequency words on phonemic tasks (Crowe, 1998).

Neuropsychology of Verbal Fluency

Both semantic and phonemic verbal fluency tasks clearly require the complex interplay of a variety of cognitive functions, including selective and sustained verbal attention, vocabulary knowledge, storage and retrieval of long-term semantic and lexical knowledge, and aspects of executive function such as strategic search and switching. Imaging studies suggest that the two types of fluency may engage different cognitive processes, as they are differentially sensitive to certain experimental manipulations (e.g., phonemic fluency is more disrupted by concurrent repetition of digits in reverse order than by object identification tasks, where the reverse is true for semantic fluency), and are differentially affected in clinical populations (Gierski & Ergis, 2004). Neuroimaging studies suggest that the two types of tasks recruit different brain regions. In general, phonemic fluency tasks appear to be more dependent on frontal systems related to strategic search, such as left dorsolateral prefrontal cortex, whereas verbal fluency impairments are more commonly linked to temporal lobe systems related to semantic knowledge. This might reflect the different strategies used to perform the task (Stuss et al., 1998).

Verbal Fluency in Clinical Populations

Impaired verbal fluency has been associated with virtually every disease and disorder affecting the brain, including dementia, traumatic brain injury, Parkinson's Disease, Huntington's Disease, depression, and schizophrenia, as well as in individuals with psychiatric and developmental disorders (see chap. 11 in Mitrushina, Boone, Razani, & D'Elia, 2005 for a summary). Attempts to find consistent patterns in patients with unilateral versus bilateral lesions or left- versus right-hemisphere lesions have yielded inconsistent results, although as expected, individuals with left-hemisphere lesions associated with aphasia have difficulty on both types of tasks and those with right-

hemisphere disorders have particular difficulty with stimuli that focus on visual attributes.

Performance on verbal fluency tests has been widely studied in individuals with Alzheimer-type dementia (e.g. Jones, Laukka & Backman, 2006). Verbal fluency tasks – particularly semantic fluency tasks such as animal naming – are becoming increasingly popular in the early or even preclinical detection of Alzheimer-type dementia, which is based on a growing body of literature documenting both impairments in semantic fluency early in the disease and the finding that rate of decline in scores mirrors disease progression. The sensitivity of semantic fluency to Alzheimer-type dementia is consistent with the notion that this type of fluency is highly dependent on temporal lobe improve clarity. As might be expected, there also is evidence that phonemic fluency is affected more than semantic fluency in frontotemporal dementia, although to date it has not been used in early detection of the disease.

Psychometric Properties of Verbal Fluency Tests

In general, verbal fluency measures have demonstrated strong inter-rater reliability, with more modest test-retest reliability. Validity and reliability data should be considered for individual tests, however, and in the context of the intended purpose of administering the test (e.g., screening for dementia vs. measuring change over time in recovery from an acquired neurological disorder). Readers are referred to entries for specific tests, such as the *Controlled Oral Word Association Test* and *F-A-S Test*.

Future Directions

Much remains to be learned about the cognitive processes underlying semantic and phonemic fluency. The frontal–temporal dichotomy is useful for heuristic reasons, but does not account for the considerable variability within and among clinical groups and is underspecified in the context of current models of dynamic cortical networks and intracortical connectivity (e.g., Kennedy et al., 2009). The finding of impaired verbal fluency is so ubiquitous in clinical populations that it may have limited diagnostic utility without consideration of characteristics such as error types and patterns of recall over time. Advances in this area may inform differential diagnosis, as well as early detection of disease processes.

Cross References

- ▶ Multilingual Aphasia Examination
- ▶ Western Aphasia Battery

References and Readings

Barry, D., Bates, M. E., & Labouvie, E. (2008). FAS and CFL forms of verbal fluency differ in difficulty: A meta-analytic study. *Applied Neuropsychology, 15*, 161–166.

Benton, A. L., & Hamsher, K. dS. (1978). *Multilingual Aphasia Examination.* Iowa City IA: AJA Associates.

Benton, A. L., Hamsher, K. dS., Rey, G. L., & Sivan, A. B. (1994). *Multilingual Aphasia Examination* (2nd ed.). Lutz, FL: Psychological Assessment Resources.

Borkowski, J. G., Benton, A. L., & Spreen, O. (1967). Word fluency and brain damage. *Neuropsychologia, 5,* 135–140.

Bousefield, W. A., & Sedgewick, C. H. W. (1944). An analysis of sequences of restricted associative responses. *Journal of General Psychology, 30,* 149–165.

Cardebat, D., Doyon, B., Puel, M., Goulet, P., & Joanette, Y. (1990). Formal and semantic lexical evolution in normal subjects: Performance and dynamics of production as a function of sex, age and educational level. *Acta Neurologica Belgica, 90,* 207–217.

Crowe, S. F. (1998). Decrease in performance on the *Verbal Fluency Test* as a funtion of time: Evaluation in a young healthy sample. *Journal of Clinical and Experimental Neuropsychology, 20,* 391–401.

Delis, D. C., Kaplan, E., & Kramer, J. H. (2001). *The Delis-Kaplan Executive Function System.* San Antonio: The Psychological Corporation.

Fabbro, F., (2001). The bilingual brain: Bilingual aphasia. *Brain and Language, 79*(2), 201–210.

Gierski, F., & Ergis, A-M. (2004). Verbal fluency: Theoretical considerations and new approaches. *L'Anee Psychologique, 104,* 331–360.

Heaton, R. K., Miller, S. W., Taylor, M. J., & Grant, I. (2004). *Revised comprehensive norms for an expanded Halstead-Reitan Battery.* Lutz, FL: Psychological Assessment Resources, Inc.

Jones, S., Laukka, E. J., & Backman, L. (2006). Differential verbal fluency deficits in the preclinical stages of Alzheimer's disease and vascular dementia. *Cortex, 42,* 347–355.

Kennedy, M. R. T., Wozniak, J. R., Muetzel, R. L., Mueller, B. A., Choiou, H.-H., Pantekoek, K., et al. (2009). White matter and neurocognitive changes in adults with chronic traumatic brain injury. *Journal of the International Neuropsychological Society, 15*(1), 130–136.

Kertesz, A. (2006). *Western Aphasia Battery - Revised (WAB-R).* San Antonio TX: The Psychological Corporation.

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

Loonstra, A. S., Tarlow, A. R., & Sellers, A. H. (2001). COWAT metanorms across age, education and gender. *Applied Neuropsychology, 8,* 161–166.

Lorenzen, B., & Murray, L. (2008). Bilingual aphasia: A theoretical and clinical review. *American Journal of Speech-Language Pathology, 17,* 299–317.

Marrero, M. Z., Golden, C. J., & Espe-Pfeifer, P. (2002). Bilingualism, brain injury, and recovery: Implications for understanding the bilingual and for therapy. *Clinical Psychology Review, 22*(3), 465–80.

Mitrushina, M., Boone, K., Razani, J., & D'Elia, L. (2005). *Handbook of normative data for neuropsychological assessment* (2nd ed.). New York: Oxford University Press.
Paradis, M. (Ed.). (1995). *Aspects of bilingual aphasia.* New York: Pergamon (Elsevier Science Ltd.).
Spreen, O., & Benton, A. L. (1969). *Neurosensory Center Comprehensive Examination for Aphasia.* Victoria: Neuropsychology Laboratory, University of Victoria.
Stuss, D. T., Alexander, M. P., Hamer, L., Palubo, C., Dempster, R., Binns, M., Levine, B., & Izukawa, D. (1998). The effects of focal anterior and posterior brain lesions on verbal fluency. *Journal of the International Neuropsychological Society, 4,* 265–278.
Thurstone, L. L., & Thurstone, T. G. (1962). *Primary Mental Abilities.* Chicago: University of Chicago Press.

Verbal Intelligence Quotient

► Verbal IQ

Verbal IQ

Rael T. Lange
British Columbia Mental Health and Addiction Services & University of British Columbia
Vancouver, BC, Canada

Synonyms

VIQ; Verbal Intelligence Quotient

Definition

Verbal IQ is a score derived from the administration of selected subtests from the Wechsler Intelligence Scales, designed to provide a measure of an individual's overall verbal intellectual abilities. The Verbal IQ score is a measure of acquired knowledge, verbal reasoning, and attention to verbal materials.

Current Knowledge

Wechsler Intelligence Scales (WIS): The WIS family of tests are some of the most widely used test batteries to assess general intellectual ability in adults aged 16 years or higher (Wechsler Adult Intelligence Scale; WAIS), children aged 6–16 years (Wechsler Intelligence Scale for Children; WISC), and children aged 2–7 years (Wechsler Preschool and Primary Scale of Intelligence; WPPSI). Since the original development of these tests (WAIS, 1955; WISC, 1949; WPPSI, 1967), all three batteries have been revised on several occasions. The most recent revisions were published in 2002 (WPPSI-III), 2003 (WISC-IV), and 2008 (WAIS-IV). The predecessor to the original WAIS, WISC, and WPPSI batteries was the Wechsler Bellevue Scale (1939 [Form 1], 1946 [Form 2]), designed to measure intellectual abilities in a broader age range of individuals (Form 1, 7–69 years; Form 2, 10–69 years).

History: The concept of Verbal IQ was first introduced by David Wechsler at the time of the development of the Wechsler–Bellevue Scale. Early measures of intelligence (e.g., ► Stanford–Binet) emphasized the notion of a general factor of intelligence (*g*) that was believed to be responsible for how an individual would perform on a variety of tasks. However, Wechsler emphasized the need to measure two broad types of abilities that should be analyzed separately to make inferences about an individual's performance. Wechsler introduced the Verbal IQ and Performance IQ scores in an effort to differentiate between the contributions of verbal and nonverbal intellectual abilities toward the measurement of *g*.

Evolution: Although the Verbal IQ score has been a prominent feature of the WIS since the development of the Wechsler–Bellevue Scale the diagnostic utility of IQ scores has been questioned for some time. Numerous factor analytic studies have supported the multidimensionality of the WIS, whereby *at least* three cognitive constructs are measured by these scales, rather than the two originally conceptualized constructs defined by Verbal IQ and Performance IQ scores. The importance of the multidimensionality of the WIS was quickly recognized by researchers who developed statistical methods that enabled factor-based interpretation of WIS scores not included in the original manuals (e.g., Atkinson, 1991). Factor-based interpretation was eventually adopted by the publisher of the WIS and was first included in the WISC-III and then later in the WAIS-III (and to some degree the WPPSI-III). The addition of new subtests to the WISC-III (i.e., Symbol Search) and WAIS-III (i.e., Symbol Search and Letter Number Sequencing) resulted in a four factor scoring system, defined by a Verbal Comprehension Index (VCI), Perceptual Organization Index (POI), Freedom from Distractibility Index (FDI; WISC-III)/Working Memory Index (WMI; WAIS-III), and Processing Speed Index (PSI). The index scores were initially introduced as an "alternative" system for scoring and interpretation that coexisted with the traditional IQ scores, which remained unchanged. However, in the recent

Verbal IQ. Table 1 Core Subtest Composition of Verbal IQ

	IN	DSP	VO	AR	CO	SI	WR	RV
WB-F1	•	•	•	•	•	•		
WB-F2	•	•	•	•	•	•		
WAIS	•	•	•	•	•	•		
WAIS-R	•	•	•	•	•	•		
WAIS-III	•	•	•	•	•	•		
WISC	•		•	•	•	•		
WISC-R	•		•	•	•	•		
WISC-III	•		•	•	•	•		
WPPSI	•		•	•	•	•		
WPPSI-R	•		•	•	•	•		
WPPSI-III[a]	•							•
WPPSI-III[b]	•		•				•	

Note: WB-F1 = Wechsler Bellevue-Form 1; WB-F2 = Wechsler Bellevue-Form 2; WAIS = Wechsler Adult Intelligence Scale; WISC = Wechsler Intelligence Scale of Children; WPPSI = Wechsler Preschool and Primary Scale of Intelligence; IN = Information; DSP = Digit Span; VO = Vocabulary; AR = Arithmetic; CO = Comprehension; SI = Similarities; WR = Word Reasoning; RV = Receptive Vocabulary.
[a]Ages 2:6–3:11.
[b]Ages 4:0–7:3.

publication of the WISC-IV and WAIS-IV, the Verbal IQ and Performance IQ scores were excluded and only the Full Scale IQ score was retained. The exclusion of the Verbal IQ and Performance IQ scores represents a significant deferment from the Wechsler scale tradition, with test interpretation largely focused on the Index scores. It is important to highlight, however, that the most recent edition of the WPPSI-III has retained the traditional Verbal IQ and Performance IQ score structure.

Subtest Composition: The core subtests used to derive Verbal IQ vary slightly across the WIS batteries and revisions. The subtests used to derive Verbal IQ across the WIS batteries and revisions are presented below. Only the core subtests (not supplementary subtests) are shown Table 1.

Cross References

- ▶ Full Scale IQ
- ▶ Intelligence
- ▶ Performance IQ
- ▶ Stanford-Binet Intelligence Scale and Revised Versions
- ▶ Verbal Comprehension Index
- ▶ Wechsler Adult Intelligence Scale (All Versions)
- ▶ Wechsler Intelligence Scale for Children
- ▶ Wechsler Preschool and Primary Scale of Intelligence

References and Readings

Atkinson, L. (1991). Some table for statistically based interpretation of WAIS-R Factor Scores. *Psychological Assessment, 3*(2), 288–291.

Kaufman, A. S., & Lichtenberger, E. O. (2006). *Assessing adolescent and adult intelligence* (3rd ed.). Hoboken, NJ: Wiley.

Sattler, J. M. (2008). *Assessment of children: Cognitive foundations* (5th ed.). San Diego, CA: Sattler Press.

Tulsky, D. S., Saklofske, D. H., & Ricker, J. H. (2003). Historical overview of intelligence and memory: Factors influencing the Wechsler Scales. In D. S. Tulsky, et al. (Eds.), *Clinical interpretation of the WAIS-III and WMS-III* (pp. 7–41). San Diego, CA: Academic Press.

Tulsky, D. S., Saklofske, D. H., & Zhu, J. (2003). Revising a standard: An evaluation of the origin and development of the WAIS-III. In D. S. Tulsky, et al. (Eds.), *Clinical interpretation of the WAIS-III and WMS-III* (pp. 43–92). San Diego, CA: Academic Press.

Verbal Mediation

Ashley de Marchena
University of Connecticut
Storrs, CT, USA

Synonyms

Inner speech; Private speech

Definition

Verbal mediation is private speech that facilitates learning and problem solving. Speech produced via verbal mediation can be either subvocal or uttered aloud; in either case, the speech is intended for the speaker, not an outside listener. Further, although verbal mediation can be accessible to conscious awareness, it is often automatic and implicit. Verbal mediation strategies increase with the development of fluent language. Although a verbal process, verbal mediation also improves performance on visuospatial and motor tasks. This process was identified early in the study of learning and memory.

Current Knowledge

Examples of verbal mediation. The act of labeling is a simple form of verbal mediation. For example, in an

early study of verbal mediation, memory for nonsense shapes was facilitated when the shapes were given names. The grammatical structure of language also facilitates learning and memory. In a paired-associates task, where participants are given pairs of words to memorize, memory is enhanced if the words are connected through a sentence. For example, the pair "elephant-glass" is learned more efficiently when it is part of the sentence "the elephant stepped on the glass." This finding may seem counterintuitive, because when a full sentence is given there is, in fact, more information to be remembered.

In addition to serving as a memory aid, verbal mediation enhances generalization in learners. The act of verbal mediation tends to direct learners' attention away from the specific stimuli being presented and toward the relationships between stimuli, which promotes generalization and leads learners to be less stimulus-bound. For example, imagine that someone is presented with three boxes of different sizes: small (box A), medium (box B), and large (box C), and taught to find a hidden object under the large box (box C). Where will this person look if the small box (box A) is replaced with an extra-large box (box D)? When verbal mediation is *not* used, participants tend to continue to select box C, as that is the specific stimulus previously associated with the hidden object. When verbal mediation *is* used, box D, the largest box available, is typically selected, suggesting that the participant generalized the relationship between the items to the new set of items (e.g., "the hidden object is under the biggest box").

Verbal mediation and neuropsychological testing. Creators of tests intended to measure purely visuospatial processing must be careful to design stimuli that do not naturally evoke verbalizations. For patients who have difficulty encoding information, the administrator can "test the limits" of their encoding abilities by explicitly giving them a verbal mediation strategy to use, for example in a paired-associates task.

Cross References

- ▶ Compensatory Strategies
- ▶ Learning
- ▶ Memory
- ▶ Metacognition
- ▶ Paired-Associate Learning
- ▶ Problem Solving
- ▶ Testing the Limits

References and Readings

Goss, A. E. (1964). Verbal mediation. *Psychological Record, 14*, 363–382.
Pyles, M. K. (1932). Verbalization as a factor in learning. *Child Development, 3*, 108–113.

Verbal Paraphasia

▶ Semantic Paraphasia

Verbal Selective Reminding Test (VSRT)

▶ Selective Reminding Test

Versional Movements

▶ Conjugate Gaze

Vertebrobasilar System

Elliot J. Roth
Northwestern University
Chicago, IL, USA

Definition

The posterior cerebral circulation, consisting of two vertebral arteries and one basilar artery, is known as the vertebrobasilar system, contributing about 20% of the brain's blood supply.

Current Knowledge

These arteries arise from the subclavian arteries, near the shoulders, ascend through the neck and into the skull, giving off a number of smaller arteries including the posterior inferior cerebellar artery (PICA), uniting as the singular basilar artery, which ultimately bifurcates into two posterior cerebral arteries. The vertebrobasilar

system perfuses the occipital cerebral cortex, cerebellum, thalamus, midbrain, pons, and medulla. Strokes that result from occlusion of these vessels are less common than anterior or middle circulation strokes. Basilar artery occlusion is fatal in 75% and survivors usually have severe disability. Blockage of vertebral arteries or their smaller branches results in a variety of findings depending on the specific location and vessel involved, but these can include quadriplegia, hemiplegia, ataxia, dysphagia, dysarthria, vertigo, visual or gaze abnormalities, cranial nerve palsies, and others. When strokes involve the brain stem, cortical deficits such as cognitive dysfunction and aphasia, are conspicuously absent. However, occipital cortex involvement can cause various visual, visuospatial and cognitive deficits, and at times, visual hallucinations.

Cross References

- ▶ Basilar Artery
- ▶ Brainstem Strokes
- ▶ Circle of Willis
- ▶ Posterior Cerebral Artery

Vertigo

Kerry Donnelly
University at Buffalo/SUNY
Buffalo, NY, USA

Definition

Vertigo refers to the subjective impression that either one's body is moving or turning (when it is not) or that the things in one's environment are spinning or moving (when they are not). It is a symptom and not a disease. Vertigo should be distinguished from dizziness, with which it is frequently confused. The latter is usually described as feelings of lightheadedness, faintness, or unsteadiness. Normally, one's sense of equilibrium requires a constant interaction between various peripheral and central mechanisms, including the labyrinthine structures of the inner ear (utricle, saccule, and semicircular canals), vestibular nuclei, vision, and proprioceptive feedback.

Vertigo usually results from damage to one or more of these systems, their connections, or their failure to work in concert with one another. Associated symptoms may include nausea, vomiting, nystagmus, feeling faint or signs of pallor, and difficulty walking. Benign paroxysmal position vertigo (BPPV) is a common and often easily treatable condition resulting from temporary displacement of otolithic crystals in the inner ear.

Cross References

- ▶ Vestibular System

References and Readings

Ropper, A. H., & Brown, R. H. (2005). *Adams and Victor's principles of neurology.* New York: McGraw Hill.

Troost, B. T. (2004). Dizziness and vertigo. In W. G. Bradley, R. B. Daroff, G. M. Fenichel, & J. Jankovic (Eds.), *Neurology in clinical practice* (Vol. I, pp. 233–245). Philadelphia: Butterworth/Heinemann.

Vestibular Dysfunction

Jennifer Sue Kleiner
University of Arkansas for Medical Sciences
Little Rock, AR, USA

Definition

The term vestibular dysfunction refers to damage to the vestibular system resulting in symptoms such as impaired balance and nausea.

Current Knowledge

Vestibular Dysfunction

Injury or dysfunction of the vestibular system will most commonly result in symptoms of dizziness or vertigo, problems with balance or equilibrium, nausea, vomiting, and/or nystagmus. It is important to distinguish ordinary dizziness from true vertigo. While dizziness can result from any number of conditions, true vertigo is more pathognomonic of vestibular system dysfunction. Vertigo involves the perception of movement. In some cases, patients perceive themselves to be spinning around in space, in others they will report that their room, the ceiling, or whatever environment they are in seems to be spinning around them. In such instances, nausea is not

uncommon. Symptoms of vestibular dysfunction can result from either peripheral (inner ear) or central (vestibular nuclei) pathology, or involvement of the vestibulocochlear nerve itself.

Problems affecting the inner ear are the most common cause of vestibular symptoms. One of the more common conditions causing such symptoms is *benign paroxysmal positional vertigo* (BPPV). It is thought to be caused by the small calcium crystals normally located in the utricle becoming dislodged and collecting in the semicircular canals causing abnormal stimulation in response changes in position of the head, such as tilting back the head, turning in bed, or getting up from the bed. Meniere's disease is another relatively frequent cause of dizziness and vertigo that is believed to result from disturbances in the inner ear. An abnormal buildup of fluids is thought to be the proximal cause of the symptoms, which commonly include auditory symptoms as well, such as loss of acuity and tinnitus. The symptoms can be relatively mild and brief or severe and chronic, the latter being very debilitating.

In addition to peripheral mechanisms, any condition that impacts the vestibular nuclei in the brain stem can also result in the vestibular symptoms described above. Conditions such as tumors, strokes, and multiple sclerosis are known for impacting any part of the central nervous system, including the brain stem. One type of tumor that is specific to the vestibulocochlear nerve is an acoustic neuroma. Originating from the Schwann cells surrounding the eighth cranial nerve, the tumor is typically found at the juncture of the pons, medulla, and cerebellum. Because it affects the cochlear portion of the nerve as well, hearing loss and tinnitus are also typically reported and in fact usually represent the earliest symptoms of the tumor. As is the case with any disorder affecting the brain stem, one should be alert for other symptoms that might help identify the location of the lesion. This might include motor or somatosensory long tract findings, cerebellar signs, or indications of other cranial nerves or their nuclei.

Typical tests for diagnosing vestibular dysfunction include electronystagmography (ENG), rotation table tests, and computerized dynamic posturography (CDP). ENG assesses functionality of the vestibulo-ocular reflex by measuring nystagmus and other eye movements; this is accomplished by attaching electrodes to the skin around the eyes and analyzing input from the electrodes. Rotation tests also utilize electrodes attached to the skin around the eyes to measure eye movements while the head is in motion. This is accomplished either by having the examinee move their head in certain directions while focusing on a fixed point, or by sitting in a computerized, motorized chair. CDP assesses motor control and balance under varying conditions. This assessment examines the relationships of the visual, somatosensory, and vestibular systems by requiring the examinee to focus on a fixed point while standing on a computerized, motorized platform. The Dix-Hallpike maneuver is another procedure used by neurology to help differentiate peripheral versus central causes of vertigo. Here, the patient is asked to lie supine on an examining table with his/her head extending slightly over the edge. With the head turned to one side, while being supported it is allowed to drop slightly below the level of the table, while observing for nystagmus or subjective reports of vertigo. Unlike with peripheral lesions in which there may be a few seconds delay, with central lesions the response, if present, is essentially instantaneous and does not abate over repeated trials.

Cross References

- ▶ Vertigo
- ▶ Vestibular System

References and Readings

Highstein, S. M., Fay, R. R., & Popper, A. N. (Eds.). (2004). *The vestibular system.* Berlin: Springer.

Ropper, A. H., & Brown, R. H. (2005). *Adams and Victor's principles of neurology.* New York: McGraw Hill.

Vestibular Nuclei

Randall E. Merchant
Virginia Commonwealth University Medical Center
Richmond, VA, USA

Definition

The vestibular nuclei function in the maintenance of equilibrium and posture, the perception of head position and acceleration, as well as general muscle tone.

Current Knowledge

The vestibular nuclei function in conjunction with the cerebellum to maintain equilibrium and posture, convey perception of head position and acceleration, and modify

muscle tone. Bipolar neurons of the vestibular ganglion (also called Scarpa's ganglion) receive input through peripheral branches coursing from specialized receptor cells in the semicircular canals and the utricle and saccule. Axons from the vestibular ganglion come together with axons projecting from the auditory neurons to form the vestibulocochlear nerve (cranial nerve VIII). The vestibular neurons project to the four vestibular nuclei, that is, superior, inferior, medial, and lateral, which lie under the floor of the fourth ventricle in the pons and rostral medulla.

The lateral vestibular nucleus receives input from the semicircular canals and utricle. Its neurons form the lateral vestibulospinal tract, which extends the length of the spinal cord, and end in the medial parts of the ventral horn of the spinal gray. Tonic excitation of those neurons affects motor neurons that innervate gravity-opposing muscles in the limbs. The medial and superior vestibular nuclei receive input from the semicircular canals. The medial vestibulospinal tract arises from medial vestibular nucleus, and these axons make connections in the cervical region of the spinal cord with motor neurons that innervate muscles of the neck. This pathway is important in mediating the reflex movements of the neck that help stabilize the position of the head in space. Ascending fibers from the superior and medial vestibular nuclei go to the motor nuclei of the oculomotor, trochlear, and abducens cranial nerves which supply the muscles of the eyes. This pathway also mediates the vestibule-ocular reflex in which eye movements are adjusted automatically for changes in head position. Ascending pathways also relay information via nuclei of the thalamus to the cerebral cortex. The inferior vestibular nucleus receives input from the semicircular canals, saccule, utricle, as well as the vermis of the cerebellum. This nucleus appears to be a site where vestibular inputs are integrated with inputs from other sensory systems as well as inputs from the cerebellum. Axons from the inferior vestibular nucleus project into the vestibulospinal and vestibuloreticular pathways. Finally, there also exist commissural projections from the contralateral vestibular nuclei to principally the superior and medial vestibular nuclei which have inhibitory influences on contralateral vestibular neurons. They also are important in vestibular compensation, a process by which reflexes and postural control that are impaired because of unilateral loss of vestibular receptor function through trauma or disease are gradually restored.

Skull fractures that pass through the internal auditory meatus can sever the eighth cranial nerve and result in rapid unilateral removal of the function of one labyrinth. When this occurs, one experiences acute symptoms including extreme dizziness, nausea and vomiting, deviation toward the side of the lesion when walking, and a brisk spontaneous nystagmus. Eventually, there is adaption to having only one vestibular labyrinth through vestibular compensation that begins almost immediately. This appears to be a learned modification in the reflexes such that the unbalanced inputs from the vestibular system are ignored and visual and proprioceptive inputs are relied upon completely.

Cross References

- ▶ Cranial Nerves
- ▶ Eye Fields
- ▶ Vestibular System
- ▶ Vestibulocochlear Nerve

Vestibular Schwannoma

▶ Acoustic Neuroma

Vestibular System

Jennifer Sue Kleiner
University of Arkansas for Medical Sciences
Little Rock, AR, USA

Definition

The vestibular system is responsible for equillibrioception, including detection of motion and positional changes, sense of balance, and spatial orientation.

Current Knowledge

The end organs of the vestibular system, which is closely connected to the auditory system, are located in the inner ear. The vestibular system is responsible for equillibrioception, including detection of motion and positional changes, sense of balance, and spatial orientation. It plays a major role in such basic functions as standing upright, coordination and movement, and in maintaining

one's balance and equilibrium. This is accomplished by detecting the position of the head relative to gravitational forces and and its motion, which then lead to compensating, reflexive adjustments of the trunk and/or limbs. This system also contributes to the adjustment of heart rate and blood pressure, muscle tone, and limb position.

The vestibular system portion of the inner ear is comprised of several components – the saccule, utricle, and the semicircular canals. The saccule and utricle contain small calcium crystals (otoliths) encased within a gel-like medium, which respond to changes in linear motion or position of the head in relation to gravity. Cilia-lined hair cells contained within these structures detect the shifting of the crystals that trigger a response in the nerve cells.

The canals consist of solid semicircular structures oriented in three different planes, horizontal/lateral, anterior/superior, and posterior/inferior. As is in the cochlea, these are filled with endolymph and contain motion-sensitive hair cells. As the head moves through different angles in space, the endolymph in one of more of the canals shifts in a "push-pull" fashion, stimulating these hair cells. The speed as well as the direction of the motion of the head will cause different hair cells to be deflected in a particular direction and with a given amplitude, resulting in either a depolarization or hyperpolarization of the attached nerves. These changes in the baseline firing rate of the vestibular neurons are conveyed to the vestibular nuclei, which are located in the dorsolateral portion of the brain stem adjacent to the fourth ventricle, although fibers appear to project directly to one of the more primitive portions of the cerebellum, the flocculonodular lobe. The vestibular nuclei themselves have complex, direct, and indirect interactive connections with the cerebellum, spinal cord, other parts of the brain stem, as well as the cortex that allow not only one to be consciously aware of one's orientation and movement through space, but also to provide a mechanism for automatic (reflexive or unconscious) motor adjustments to help maintain balance and equilibrium.

One such well-known phenomenon is the vestibulo-ocular reflex. If, while looking at a book or a computer screen, you turn your head from side to side, the text or image you see stays stationary. This ability to accommodate to such movements of the head while reading is known as vestibulo-ocular reflex. It is accomplished by direct connections between the vestibular nuclei and the brainstem nuclei controlling the extraocular muscles via the medial longitudinal fasciculus. Thus, if the head is moved to the right, the right oculomotor nucleus stimulates the medial rectus muscle of the right eye causing it to shift proportionally to the left, and the left abducens nucleus produces a similar reaction in the left eye by stimulating the lateral rectus. Another example of a vestibular reflex at work is when one might trip or unexpectedly step into a depression while walking. In either case, this results in a sudden shift in the position or motion of the head, followed by a cascade of changes in the flexor and extensor muscles of the extremities and of the trunk to help insure one does not fall. These reflexive actions are largely the result of an interaction between the vestibular nuclei, the spinal cord, and the cerebellum.

Damage or excessive stimulation to the vestibular system can result in vertigo, dizziness, postural instability or problems with balance, nystagmus, and nausea (See Vestibular Dysfunction for a more thorough review).

Cross References

- ▶ Vestibular Dysfunction
- ▶ Vestibular Nuclei
- ▶ Vestibulocochlear Nerve

References and Readings

Highstein, S. M., Fay, R. R., & Popper, A. N. (Eds.). (2004). *The vestibular system.* Berlin: Springer.

Wilson-Pauwek, L., Akesson, E. J., Stewart, P. A., & Spacey, S. D. (2002). *Cranial nerves in health and disease.* Hamilton, ONT: B.C. Decker, Inc.

Vestibulocochlear Nerve

John E. Mendoza
Tulane University Medical Center
New Orleans, LA, USA

Synonyms

Eighth cranial nerve

Definition

The cranial nerve that subserves both hearing and vestibular functions. It is essentially a double nerve that enters the brainstem laterally at the pontomedullary junction.

Current Knowledge

The cochlear portion of this nerve is responsible for carrying auditory information from the hair cells of the organ of Corti in the cochlea, within the inner ear, to the dorsal and ventral cochlear nuclei in the medulla. The vestibular portion of the nerve is derived from nerve receptors in the three semicircular canals, the utricle, and the saccule. The semicircular canals represent three different planes or orientations in space and respond to angular acceleration and deceleration. The utricle responds to gravitational forces and horizontal linear acceleration. The saccule responds to linear acceleration in the dorsal–ventral plane. Together these responds provide information regarding the orientation of the head in space and the movement of one's body (head) through space (both the direction of movement, as well as the sense of movement). As the fibers for the vestibular system enter the brainstem, a few course directly to the flocculonodular lobe of the cerebellum, while most synapse in the vestibular nuclei of the medulla and pons.

The vestibulocochlear nerve can be affected by various neuropathological processes, including tumors, infections, and strokes. If the cochlear portion of the nerve is damaged, reductions in or loss of hearing as well as tinnitus may result. If the vestibular portion of the nerve is affected, the patient may experience vertigo and/or unsteadiness of gait.

Cross References

- ▶ Auditory System
- ▶ Vestibular System

References and Readings

Wilson-Pauwek, L., Akesson, E. J., Stewart, P. A., & Spacey, S. D. (2002). *Cranial nerves in health and disease*. Hamilton, ON: B.C. Decker, Inc.

Vibration, Perception of

▶ Pallesthesia

Vibratory Sense

▶ Pallesthesia

Victoria Symptom Validity Test

Eric S. Hart
University of Missouri Center for Health Care Quality
Columbia, MO, USA

Synonyms

VSVT

Description

The Victoria Symptom Validity Test (VSVT) is a two-alternative forced-choice, computer-administered assessment measure used to determine the validity of reported cognitive impairment (Slick, Hopp, Strauss, & Thompson, 1997). Interpretation of VSVT performance is based on binomial probability theory, which considers the probability of a particular response occurring by chance alone.

The VSVT is comprised of 48 items presented in three separate blocks (16 items per block). Stimuli (a five-digit number) in each block are first presented during a study trial, followed by a retention trial in which the examinee views a blank screen for a brief period, increasing from 5 to 15 s from the first to the third block. Next, examinees view two sets of stimuli presented on either side of a computer screen – a five-digit number from the study trial and a foil – and are asked to choose the item shown during the study trial. Presentation of the target stimuli and foil is counterbalanced and pseudo-randomized. In order to increase sensitivity by manipulating the perception of difficulty among test items, there are *Easy* and *Hard* trials, which are scored separately; each block contains an equal number of *Easy* and *Hard* items (Strauss, Sherman, & Spreen, 2006). *Easy* trials are those where the studied stimuli and foil share no common numbers. *Hard* trials share the same numbers, but the middle digits (i.e., second and third or third and fourth) are transposed. Slick et al. (1997) found that, although *Hard* trials appear more difficult, non-compensation-seeking individuals with head injury do not perform significantly worse on these items compared to *Easy* trials.

Scoring is based on the number of correct responses overall and within each section. Z-scores are generated based on consideration of the probability of chance-level performing (50% correct). For example, a Z-score of 0 represents performance in which half of the items are

passed successfully. Z-scores are further converted into *p* values to determine the likelihood that a particular response pattern was obtained by chance alone; higher scores (e.g., $z > 1.65$) suggest greater-than-chance performance, while lower scores (e.g., $z < 1.65$) suggest worser-than-chance performance. An important scoring dimension of the VSVT is the addition of the *Questionable* qualifier to describe scores that occur near the chance level, which is an expansion of traditional dichotomous classification systems (e.g., valid and invalid) commonly found in symptom validity tests (Thompson, 2002). Additional sources of interpretation include *bias*, referring to a tendency to use one hand more than the other to respond to account for possible motoric conditions or perceptual difficulty, and *mean response* latency (Strauss, Sherman, & Spreen, 2006).

Historical Background

The VSVT was originally published in 1997 (Slick et al., 1997) and was modeled after the Hiscock and Hiscock (1989) Forced-Choice Test, which similarly employed a two-alternative forced-choice paradigm and a set of five-digit numbers as target and foil objects. Based on their research, Slick and colleges (1994) developed the VSVT as a modification of Hiscock's test by reducing the number of items and manipulating item difficulty to increase sensitivity to feigned memory impairment, as described above. Subsequent research on the VSVT has focused on cross validation and the use of cutoff scores (e.g., Grote, Kooker, Garron, Nyenhuis, Smith, & Mattingly, 2000). In comparison with other measures in use at the time of its development, the VSVT was considered to be more psychometrically sound due to enhancements such as the distinction between *Easy* and *Hard* trials and the *Questionable* qualifier, which increased sensitivity without affecting administration time or specificity (Thompson, 2002).

Psychometric Data

Internal consistency reliability has been found to be 0.82 for *Easy* items, 0.87 for *Hard* items, and 0.89 for the entire set (Slick et al., 1996). Test–retest reliability has been found to be low for control samples (0.53–0.54), yet higher for compensation-seeking participants (0.56–0.84) (Slick et al., 1997). A variety of studies have supported the validity of VSVT performance as a sensitive indicator of motivation. Divergent and convergent validity have also been supported with other effort measures, such the MMPI-2 validity scales (Slick et al., 1996). The VSVT has also been validated for use with Spanish-speaking populations (Vilar-Lopez et al., 2007).

Clinical Uses

The growing interest in forensic neuropsychology and concerns regarding the potential impact of primary and secondary gain on neuropsychological test performance have prompted the development of empirically supported tests of symptom validity. Although estimates of the prevalence of poor effort on neuropsychological testing vary depending on the population and reason for referral, Mittenberg, Patten, Canyock, and Condit (2002) concluded that malingering occurred between 38.5% and 41.24% in a sample of individuals reporting mild traumatic brain injury who were involved in litigation. These data highlight the need for reliable and valid measures for assessing the validity of reported impaired cognition. In a survey of practicing neuropsychologists, the VSVT was rated as one of the top five of available symptom validity tests in terms of accuracy in detecting suboptimal effort (Sharland & Gfeller, 2007).

Early research on the VSVT supported the sensitivity of the instrument in detecting dissimulation in a traumatic brain injury population (Slick et al., 1994). More recent research has found the VSVT to be sensitive to incomplete effort in non-litigating clinical populations (epilepsy surgery patients), in which no motivation to feign impairment would be expected (Loring, Lee, & Meador, 2005). The VSVT has been utilized to document a relatively low prevalence of intentional feigning in a mixed clinical sample not involved in litigation (Loring et al., 2007). It has also been found sensitive to simulated ADHD and Reading Disorder (Frasier et al., 2008). It has been suggested that the VSVT likely meets Daubert standards, which determines the admissibility of scientific evidence in legal proceedings (Thompson, 2002).

Cross References

- ▶ Computerized Assessment of Response Bias
- ▶ *Daubert v. Merrell Dow Pharmaceuticals (1993)*
- ▶ Effort
- ▶ Fake Bad Scale (FBS)
- ▶ Hiscock Forced-Choice Test
- ▶ Malingering

References and Readings

Brandt, J. (1988). *Malingered amnesia: clinical assessment of malingering an deception.* New York: Guilford.

Frazier, T. W., Frazier, A. R., Busch, R. M., Kerwood, M. A., & Demaree, H. A. (2008). Detection of simulated ADHD and reading disorder using symptom validity measures. *Archives of Clinical Neuropsychology, 23*(5), 501–509.

Greve, K. W., & Bianchini, K. J. (2002). Using the wisconsin card sorting test to detect malingering: an analysis of the specificity of two methods in nonmalingering normal and patient samples. *Journal of Clinical and Experimental Neuropsychology, 24*(1), 48–54.

Grote, C. L., Kooker, E. K., Garron, D. C., Nyenhuis, D. L., Smith, C. A., & Mattingly, M. L. (2000). Performance of compensation seeking and non-compensation seeking samples on the Victoria Symptom Validity Test: Cross validation and extension of a standardized study. *Journal of Clinical and Experimental Neuropsychology, 22*, 709–719.

Hiscock, M., & Hiscock, C. K. (1989). Refining the forced choice method for the detection of malingering. *Journal of Clinical and Experimental Neuropsychology, 11*, 967–974.

Loring, D. W., Larrabee, G. J., Lee, G. P., & Meador, K. J. (2007). Victoria Symptom Validity Test performance in a heterogenous clinical sample. *Clinical Neuropsychologist, 21*(3), 522–531.

Loring, D. W., Lee, G. P., & Meador, K. J. (2005). Victoria Symptom Validity Test performance in non-litigating epilepsy surgery candidates. *Journal of Clinical and Experimental Neuropsychology, 27*, 610–617.

Mittenberg, W., Patton, C., Canyock, E. M., & Condit, D. C. (2002). Baserates of malingering and symptom exaggeration. *Journal of Clinical and Experimental Neuropsychology, 24*, 1094–1102.

Sharland, M. J., & Gfeller, J. D. (2007). A survey of neuropsychologists' beliefs and practices with respect to the assessment of effort. *Archives of Clinical Neuropsychology, 22*(2), 213–223.

Slick, D., Hopp, G., Strauss, E., Hunter, M., & Pinch, D. (1994). Detecting dissimulation; Profiles of simulated malingerers, traumatic brain-injury patients, and normal controls on a revised version of Hiscock and Hiscock forced choice memory test. *Journal of Clinical and Experimental Neuropsychology, 16*, 472–481.

Slick, D., Hopp, G., Strauss, E., & Spellacy, F. (1996). Victoria Symptom Validity Test: efficacy for detecting feigned memory impairment and relationship to neuropsychological tests and MMPI-2 validity scales. *Journal of Clinical and Experimental Neuropsychology, 18*, 911–922.

Slick, D., Hopp, G., Strauss, E., & Thompson, G. B. (1997). *VSVT: Victoria Symptom Validity Test (Version 1.0)*. Odessa, FL: Psychological Assessment Resources.

Slick, D. J., Tan, J. E., Strauss, E., Mateer, C. A., Harnadek, M., & Sherman, E. M. S. (2003). Victoria Symptom Validity Test scores of patients with profound impairment. Nonlitigant case studies. *Clinical Neuropsychologist, 17*, 390–394.

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

Thompson, G. B. III (2002). The Victoria Symptom Validity Test: an enhanced test of symptom validity. *Journal of Forensic Neuropsychology, 2*, 43–67.

Tan, J. E., Slick, D. J., Strauss, E., & Hultsch, D. F. (2002). How'd they do it? Malingering strategies on symptom validity tests. *Journal of Forensic Neuropsychology, 2*, 43–67.

Vilar-Lopez, R., Santiago-Ramajo, S., Gomez-Rio, M., Verdejo-Garcia, A., Llamas, J. M., & Perez-Garcia, M. (2007). Detection of malingering in a Spanish population using three specific malingering tests. *Archives of Clinical Neuropsychology, 22*(3), 379–388.

Vigilance

Stephen Correia, Ronald A. Cohen
Brown University
Providence, RI, USA

Definition

Vigilance (from Latin, *vigil;* awake) is conceptualized as a special case of the broader psychological construct of sustained attention. Sustained attention refers to the ability to consciously or semiconsciously focus on tasks over extended periods of time whereas vigilance may be defined more narrowly as a person's preparedness to detect infrequent and unpredictably occurring events or signals over prolonged periods of time. Vigilance is not easy for humans because it is difficult to sustain attention over long periods of time. The distinction made here between vigilance and sustained attention is not always made clear in the scientific literature and the two terms are often used interchangeably. Vigilance is typically measured using tests that require an individual to detect infrequent targets and discriminate them from "noise" or distractor stimuli and to do this over extended periods of time. Such tasks usually require the subject to either make or inhibit a response to the target.

Historical Background

The temporal nature of attention was discussed in the writing of Williams James, and has been considered to be an important aspect of attending by psychologists throughout the twentieth century. Most people can relate to this aspect of attention in everyday experience. When a teacher or parent instructs a child to pay attention, they are essentially telling them to sustain their attention focus to some task, be it an assignment or something being said. Formal investigation of attention was a natural outgrowth of research directed at understanding the mechanisms of information processing and the factors that impact it. Experimental work on vigilance per se accelerated in the 1950s when psychologists and engineers working on factors that influence signal detection in military and

occupational contexts, realized that it was essential to account for performance characteristics over time. The requirement for vigilance by radar operators as they monitored for signals of aircraft, missiles, or other relatively rare events was a catalyst for these efforts. Mackworth's research in the 1960 (Mackworth, 1964, 1965, 1968; Mackworth & Taylor, 1963) focused explicitly on these skills in that the experimental designs generally required a participant to keep watch for subtle signals over prolonged periods of time. The state of readiness to detect such signals was conceptualized as *vigilance level* (i.e., the participant's overall ability to detect signals), and *vigilance decrement* (i.e., decline in performance over time). Since then, our understanding of the construct of vigilance has been extended to the more general construct of sustained attention. Research in this area has advanced considerably, and has lead to the development and validation of a number of tests of sustained attention and vigilance. These advances, along with the development of functional neuroimaging techniques have fostered considerable research into the neuronal circuitry of vigilance and sustained attention.

Current Knowledge

Constructs for measurement; top-down vs. bottom-up; arousal: The ability to maintain vigilance can be conceptualized as being governed by "bottom-up" and "top-down" mechanisms (Sarter et al., 2001). These two mechanisms are thought to overlap and interact with one another to achieve optimal attentional performance. In bottom-up views, attentional functions are driven by the specific characteristics of the target stimulus and its sensory context. The ability to detect and process targets is governed mainly by sensory salience of the targets and the degree to which they trigger successively higher cortical areas, for example from initial ability to process the target in primary visual cortex to temporal cortical regions for object identification and parietal regions for object location. Top-down perspectives emphasize knowledge-driven mechanisms that facilitate neuronal processing of the relevant stimuli to promote discrimination between targets and distractors and to bias the subject to particular spatial locations where a target is likely to appear. Thus, top-down mechanisms invoke knowledge about the likely location, modality, and probability of a target to optimize performance on sustained attention tasks.

In a similar vein to the top-down vs. bottom-up distinction, Barkley (2006) differentiates between sustained attention and vigilance mechanisms that are maintained by external rewards ("contingency-shaped or context-dependent") vs. maintained by internal motivational mechanisms ("goal-directed persistence"). In the former case, attention is maintained primarily by the external context; "its origins lie in the nature of those immediate contingencies operating within the task or setting and the individual's contact with them" (p. 317) for example a schedule of intermittent reinforcement. In the latter case, sustained attention is under the control and guidance of internal representations such as goals, intentions, or plans. Motivation may also play a role, particularly in situations where vigilance is directed toward detection of dangerous or highly rewarding targets. According to Barkley, in the case of internally mediated attention requires self regulation, behavioral inhibition, and resistance to distracting stimuli that may arise from external or internal sources. Barkley maintains that externally-maintained and internally-maintained sustained attention cannot be distinguished behaviorally by the casual observer. The difference becomes apparent, however, when sources of immediate reinforcement are removed from the task or context: Vigilance performance that is mediated by internal representations should not diminish, whereas performance that is mediated by external contingencies should decline. Barkley suggests that patients with attention-deficit hyperactivity disorder experience a breakdown in goal-directed persistence whereas contingency-shaped or context-dependent attention functions remain intact. Barkley proposes that this is a reason why children with ADHD can easily focus on rewarding tasks such as playing video games but have difficulty with tasks that are contingent on internal representations such as doing homework.

There are a number of factors that can impact vigilance ability. Obviously, wakefulness and alertness are key requisites and vigilance drops off quickly during drowsiness. As noted already, motivation is a key factor in vigilance performance. It has been long known that there an inverted U-shaped distribution characterizes the relationship between stress and sustained attention performance (Yerkes & Dodson, 1908). That is, mild degrees of stress may actually improve performance on cognitive tests including tests of sustained attention, whereas excessive stress can result in performance decrements. Vigilance can be influenced by cognitive aspects of the stimuli such as signal modality (e.g., visual vs. auditory), intensity, and the duration of the stimuli and the amount and type of background noise. Also, the rate of target events, their probability, and how regularly they occur are also aspects of stimuli that may impact performance on vigilance tasks.

An important aspect of vigilance or sustained attention is the ability to release attention or switch attentional focus to other targets when necessary. DeGangi and Porges (1990) propose that release of attention can be influenced by physical or mental fatigue, the presence and rate at which new stimuli occur, arousal, and the strength of the stimulus. A recent report involving a lesion study in primates and a functional magnetic resonance imaging (fMRI) in humans highlights the importance of the prefrontal cortex (PFC) in mediating top-down switching of attentional focus (Rossi et al., 2009). In these experiments, it appeared that the frontal eye fields as well as middle and inferior PFC are critical for attentional switching. In the humans, the intraparietal sulcus was activated in addition to the PFC areas during attentional switching tasks.

Circuitry: Human functional neuroimaging studies have shown that performance on tasks of vigilance and sustained attention, including those that are guided by internal representations, is associated with activation of frontal (anterior cingulate and dorsolateral prefrontal) and parietal cortical areas, mostly in the right hemisphere. This finding has been consistent irrespective of the modality of stimuli used in these tasks. There is recent evidence that the fronto-parietal component of this network is critical for initiation and adjustment of attentional control, whereas the cingulate lateral frontal (opercular) component is important for maintaining a stable cognitive set over the course of a task (Dosenbach et al., 2008). Overall, there is high correspondence between the results of functional neuroimaging studies and studies of patients with brain damage and neuronal degeneration as evidenced by increased errors on vigilance tasks (e.g., reduction in the number of hits, increased susceptibility to distracting stimuli, increased reaction time). The reason for the lateralized finding in these studies is unclear but one possibility is a lateralized specialization in sustained attention ability. Additionally, or alternatively, this finding could be related to the types of tasks used in functional neuroimaging studies of vigilance and sustained attention. The right hemisphere dominance for vigilance seen in human studies has not been addressed adequately in animal studies. Electrophysiological studies suggest that sustained attention including vigilance may be subserved, at least in part, by recurrent activity in corticothalamic circuits involving apical dendrites of pyramidal neurons in cortical layers 5 and 6 (LaBerge, 2005).

Neurotransmitters: Sarter et al. (2001) provide a model of the neuroanatomical and neurochemical underpinnings of sustained attention and vigilance based on knowledge obtained from human and animal studies. They propose that there is a right hemisphere dominance for sustained attention and that an "anterior attention system" (Posner & Petersen, 1990) exerts top-down mediation of attentional processing in posterior cortical and sensory regions. The anterior attention system activates the basal forebrain and promotes release of acetylcholine to cortical regions. These cortical inputs modulate cognitive function generally and this likely includes sustained attention. The acetylcholine inputs to posterior cortex help to mediate frontal attentional systems and may also facilitate bottom-up sensory processing. Cholinergic activity in the brain stem and basal forebrain also plays a critical role in determining sleep vs. wakeful states. Noradrenergic projections from the locus ceruleus to basal forebrain regions and thalamus appear to be important in bottom-up processing of attention arousing stimuli. Motivational effects impacting sustained attention may be governed by immediate or delayed reward and may be mediated by the dopamine system.

Future Directions

The development of radiotracers for cholinergic and other neurotransmitters and receptors for use in human studies should permit testing to confirm or disconfirm the hypothesis that vigilance is related to cholinergic activity in the cortex. Continued studies involving non-human primates, human structural and fMRI studies, and electrophysiological studies, including multimodal analysis of such data, may continue to elucidate the functional neurocircuitry of sustained attention generally and vigilance more narrowly and the contingencies that moderate them.

Cross References

- ▶ Attention
- ▶ Focused Attention
- ▶ Sustained Attention
- ▶ Working Memory

References and Readings

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65–94.

Barkley, R. A. (2006). A theory of ADHD. In *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (3rd ed., pp. 297–334). New York: Guilford.

DeGangi, G., & Porges, S. (1990). *Neuroscience foundations of human performance.* Rockville, MD: American Occupational Therapy Association.

Dosenbach, N. U., Fair, D. A., Cohen, A. L., Schlaggar, B. L., & Petersen, S. E. (2008). A dual-networks architecture of top-down control. *Trends in Cognitive Sciences, 12*(3), 99–105.

Garavan, H., Ross, T. J., Murphy, K., Roche, R. A., & Stein, E. A. (2002). Dissociable executive functions in the dynamic control of behavior: Inhibition, error detection, and correction. *Neuroimage, 17*(4), 1820–1829.

Hancock, P. A., & Warm, J. S. (2003). A dynamic model of stress and sustained attention. *Human Performance in Extreme Environment, 7*(1), 15–28.

Hester, R., D'Esposito, M., Cole, M. W., & Garavan, H. (2007). Neural mechanisms for response selection: Comparing selection of responses and items from working memory. *Neuroimage, 34*(1), 446–454.

Kelly, A. M., & Garavan, H. (2005). Human functional neuroimaging of brain changes associated with practice. *Cerebral Cortex, 15*(8), 1089–1102.

Kubler, A., Murphy, K., Kaufman, J., Stein, E. A., & Garavan, H. (2003). Co-ordination within and between verbal and visuospatial working memory: network modulation and anterior frontal recruitment. *Neuroimage, 20*(2), 1298–1308.

LaBerge, D. (2005). Sustained attention and apical dendrite activity in recurrent circuits. Brain Research. *Brain Research Reviews, 50*(1), 86–99.

Lawrence, N. S., Ross, T. J., Hoffmann, R., Garavan, H., & Stein, E. A. (2003). Multiple neuronal networks mediate sustained attention. *Journal of Cognitive Neurosciences, 15*(7), 1028–1038.

Mackworth, J. F. (1964). Performance decrement in vigilance, threshold, and high-speed perceptual motor tasks. *Canadian Journal of Psychology, 18*, 209–223.

Mackworth, J. F. (1965). Decision interval and signal detectability in a vigilance task. *Canadian Journal of Psychology, 19*, 111–117.

Mackworth, J. F. (1968). Vigilance, arousal, and habituation. *Psychological Review, 75*(4), 308–322.

Mackworth, J. F., & Taylor, M. M. (1963). The D' Measure of signal detectability in vigilance-like situations. *Canadian Journal of Psychology, 17*, 302–325.

McGrath, J. J., & O'Hanlon, J. (1967). Temporal orientation and vigilance performance. *Acta Psychologica (Amsterdam), 27*, 410–419.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*, 167–202.

Oken, B. S., Salinsky, M. C., & Elsas, S. M. (2006). Vigilance, alertness, or sustained attention: Physiological basis and measurement. *Clinical Neurophysiology, 117*(9), 1885–1901.

Porges, S. W. (1984). Physiologic correlates of attention: A core process underlying learning disorders. *Pediatric Clinics of North America, 31*(2), 371–385.

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

Rossi, A. F., Pessoa, L., Desimone, R., & Ungerleider, L. G. (2009). The prefrontal cortex and the executive control of attention. *Experimental Brain Research, 192*(3), 489–497.

Sarter, M., Givens, B., & Bruno, J. P. (2001). The cognitive neuroscience of sustained attention: Where top-down meets bottom-up. *Brain Research Reviews, 35*(2), 146–160.

Valley, V., & Broughton, R. (1983). The physiological (EEG) nature of drowsiness and its relation to performance deficits in narcoleptics. *Electroencephalography and Clinical Neurophysiology, 55*(3), 243–251.

Weinberg, W. A., & Harper, C. R. (1993). Vigilance and its disorders. *Neurol Clinics, 11*(1), 59–78.

Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit-formation. *Journal of Comparative Neurology and Psychology, 18*, 459–482.

Vigilance Tests

► Cancellation Tests

Vineland Adaptive Behavior Scales

Sara S. Sparrow
Yale University Child Study Center
New Haven, CT, USA

Synonyms

Vineland II

Description

The Vineland Adaptive Behavior Scales II (Vineland II) (2005, 2006, 2008) is the second revision of the venerable and internationally employed Vineland Social Maturity Scale (VSMS) (Doll, 1935), the first standardized adaptive behavior test. The first revision, the Vineland Adaptive Behavior Scales (ABS) was published in 1984 (survey and expanded forms) and 1985 (classroom edition). The ABS and the Vineland II are the most widely used adaptive behavior tests in the world. The Vineland II assesses an individual's development of personal independence and social responsibility by gathering information about day-to-day activities necessary to take care of oneself and to get along with others. There are four forms of the Vineland II, two using information obtained in a semi-structured interview with a parent or caregiver (2005) and two using a rating form completed by a teacher or parent/caregiver (2005; 2006).

For more information on each form see Table 1.

The Vineland covers four adaptive behavior domains: communication, daily living skills, socialization, and motor skills (the latter being optional for individuals

Vineland Adaptive Behavior Scales. Table 1 Vineland II forms

Form	Format	No. of Items	Age range
Survey	Interview	376	Birth to 90+
Parent/caregiver	Rating form	376	Birth to 90+
Teacher report	Rating form	221	3–21
Expanded report	Interview	601	Birth to 90+

over age 6). It also provides an Adaptive Behavior Composite Score. Three subscales – internalizing, externalizing, and other – comprise the optional Maladaptive Behavior Index that provides a measure of undesirable behaviors that may interfere with an individual's adaptive behavior. The optional Maladaptive Critical Items do not contribute to a subscale or composite, but provide a brief measure of more severe maladaptive behaviors that examiners may want to consider in the overall assessment of adaptive behavior. For more information on each domain and subdomain, see Table 2.

The Vineland II offers several derived scores. The adaptive behavior domains and the Adaptive Behavior Composite have standard scores (a mean of 100 and an sd of 15) that range from 20 to 160. The subdomains have scaled scores called v-scaled scores (mean of 15 and an sd of 3) and a range of scores from 1 to 24. The *v-scaled scores* allow for finer differentiation of performance for low-functioning individuals than is usually found in any other standardized tests. Other derived scores include percentile ranks, adaptive levels that are percentile-based (high, moderately high, adequate, moderately low, and low), stanines, and age equivalents (for subdomains only).

The Vineland II was standardized on 3,695 individuals from birth to 90+ years of age and was stratified on the basis of the 2001 US census on race/ethnicity, mothers' or individuals' education level, geographic region, and community size. In addition, data were collected on seven special populations for whom, research with the ABS suggests, diagnostic patterns are often found that may discriminate between groups. For the clinical groups, see Table 3.

Vineland Adaptive Behavior Scales. Table 2 Vineland II domains and subdomains

Domains	Subdomains
Communication	Receptive: How the student listens, and pays attention, and what he or she understands
	Expressive: What the individual says, how he or she uses words and sentences to gather and provide information
	Written: What the individual understands about how letters make words, and what he or she reads
Daily living skills	Personal: How the student eats, dresses, and practices personal hygiene
	Domestic: What household tasks the individual performs
	Community: How the individual uses time, money, telephone, computer, and job skills
Socialization	Interpersonal: How the student interacts with others
	Play and leisure time: How the student plays and uses leisure time
	Coping skills: How the student demonstrates responsibility and sensitivity to others
Motor	Gross: How the student uses arms and legs for movement and coordination
	Fine: How the student uses hands and fingers to manipulate objects
Maladaptive (optional)	Internalizing
	Externalizing
	Other

Historical Background

The ABS, (Sparrow, Balla, & Cicchetti, 1984a, b, 1985) represented a major revision of the venerable and internationally employed VSMS (Doll, 1935). The Vineland II (Sparrow, Cicchetti, & Balla, 2005, 2006, 2008) is the revision of the ABS, and has several features not found in the ABS. Not only were the norms brought up-to-date, but the age range was extended from birth to 19 years to birth to 90+ years. Many items were modified or added to reflect cultural changes and new research knowledge of developmental disabilities since the publication of the ABS. In addition, an extensive bias review was carried out by experts in many fields to assure as much culture or ethnic free content as deemed possible. Data from the standardization suggest that, contrary to IQ testing, adaptive behavior does not demonstrate a "Flynn Effect" or the general increase in IQ over time across cultures (Flynn, 2007).

The ABS has been translated into many languages including Spanish, Dutch, French, Indonesian, Farsi, German, Hebrew, Italian, Arabic, and various African

Vineland Adaptive Behavior Scales. Table 3 Vineland II clinical samples

Attention deficit disorders
Autism nonverbal
Autism verbal
Emotional and behavioral disturbance
Deafness/hard of hearing
Learning disability
Mental retardation/intellectual disability (mild)
Mental retardation/intellectual disability (moderate)
Mental retardation/intellectual disability (severe/profound)
Visual impairment

languages. Many of these versions are being retranslated for the Vineland II.

Adaptive behavior is age-based and is defined by the standards of others. Adaptive behavior, in contrast to IQ, represents the typical performance rather than the potential or ability of the individual – what a person actually does as opposed to what a person is capable of doing. Adaptive behavior is modifiable and can be affected negatively or positively depending on intervention and/or life events.

Psychometric Data

The Vineland II represents state-of-the-art psychometric properties. Reliability estimates included Interrater and inter-interviewer and were from the low .70s to high .80's. Split-half reliability for the Adaptive Behavior Composite ranges from .93 to .97, while subdomains were in the .80s and .90s. Test–retest reliabilities ran mostly from the .80s to .90s.

Investigation for validity was conducted based on the content, structure, demographic characteristics, clinical groups, and the relationships with other variables such as Adaptive Behavior Assessment System II (ABAS II), WISC III, WAIS III, and BASC II.

Clinical Uses

There are generally five clinical uses of the Vineland II:

1. Diagnostic evaluations
2. Developmental evaluations
3. Program monitoring
4. Program planning
5. Research

Diagnostic Evaluations

The most common use of the Vineland II is to contribute to the mandated definition of mental retardation or intellectual disability. To make such a diagnosis, the individual being assessed must demonstrate a significant deficit in adaptive functioning as well as significantly delayed cognitive functioning. However, in recent years, the use of the Vineland II has been broadened to help aid in the diagnoses of many conditions, with or without intellectual disability. The Vineland II is the most frequently used adaptive measure in the diagnosis of individuals on the autism spectrum, both low and high functioning. The social deficits found in autism have been well documented in autism research.

Developmental Evaluations

Since the Vineland II starts at birth and has normative data for following normal growth ranging from each 30 days below age 2 to 1 month through age 5, it is ideal for performing developmental evaluations. Public Law 99–437 made the assessment of adaptive functioning a necessary part of evaluating children from birth through the age of 5 when determining their eligibility for special services.

Program Monitoring

The Vineland II is often used to measure change or progress of intervention programs since the improvement of an individual's ability to cope with everyday life is a goal of many, if not most, interventions. In addition, because of the excellent norm density and thus better ability to document even small progress, the Vineland II can be utilized much more frequently than other instruments, particularly cognitive measures (i.e., IQ).

Program Planning

All forms of the Vineland II can be useful in program planning since they document those behaviors the individual needs to acquire in an intervention program. However, the format of the expanded form was designed specifically for this purpose. In terms of intervention, Klin and his colleagues found, in a study of individuals with high-functioning autism and Asperger's syndrome, that interventions addressing adaptive behavior led to more

favorable outcomes than those focusing on autistic symptomatology (Klin, Saulnier, Sparrow, Cicchetti, Volkmar, & Lord, 2007).

Research

Since the publication of the ABS in 1984, several thousand research studies have been conducted using the ABS and, more recently, the Vineland II, as both independent and dependent variables. Some of the diagnostic groups studied include all forms of intellectual disability, all forms of autism (both high and low functioning), many genetic disorders, post-traumatic brain injury, children who are gifted, children who are precocious readers, children exposed to violence, and children with attention deficit disorder, hearing impairment, visual handicap, and emotional and behavioral disturbance.

Cross References

► Vineland Social Maturity Scales

References and Readings

Doll, E. A. (1935). A genetic scale of social maturity. *The American Journal Of Orthopsychiatry, 5*, 180–188.

Klin, A., Saulnier, C. A., Sparrow, S. S., Cicchetti, D. V., Volkmar, F. R., & Lord, C. (2007). Social and communication abilities and disabilities in higher functioning individuals with autism spectrum disorders: The Vineland and the ADOS. *Journal of Autism and Developmental Disorders, 37*, 748–759.

Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1984a). *The Vineland adaptive behavior scales: Interview edition, survey form.* Circle Pines, MN: American Guidance Service.

Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1984b). *The Vineland adaptive behavior scales: Interview edition, expanded form.* Circle Pines, MN: American Guidance Service.

Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1985). *The Vineland adaptive behavior scales: Classroom edition.* Circle Pines, MN: American Guidance Service.

Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales: Second edition (Vineland II), survey interview form/ caregiver rating form.* Livonia, MN: Pearson Assessments.

Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2008). *Vineland adaptive behavior scales: Second edition (Vineland II), The expanded interview form,* Livonia, MN: Pearson Assessments.

Vineland II

► Vineland Adaptive Behavior Scales

Vineland Social Maturity Scales

Sara S. Sparrow
Yale University Child Study Center
New Haven, CT, USA

Synonyms

VSMS

Description

The Vineland Social Maturity Scales (VSMS), published by Edgar Doll in 1935, measures social maturity or social competence in individuals from birth to adulthood. The test is no longer available but represents an important historical contribution to the assessment of adaptive behavior or those behaviors that lead to personal independence and social responsibility. Doll classified eight categories of items on the VSMS (Doll, 1935): self-help general, self-help dressing, self-help eating, communication, self-direction, socialization, locomotion, and occupation. Although there is some difference of opinion as to whether Doll's categorization is the best, the perception of adaptive behavior as multidimensional has survived from one generation to the next. The VSMS has 117 items using Stanford Binet year-scale format in the record booklet. Each age level, however, does not measure all the eight categories resulting in limited item density for all the categories.

This said, it should be noted that most of these concepts remain crucial to the definition of adaptive behavior, now more than 70 years later. For example, the current Vineland II and its predecessor, the Vineland Adaptive Behavior Scales, embody the self-help categories under a single rubric, daily living skills; locomotion is dually categorized into gross and fine motor skills, and socialization remains as arguably the most critical of the areas of adaptive behavior in many diagnostic areas, not least among them, the definition of autism and autistic-like behaviors. While Doll restricted the application of adaptive behavior to the area of mental retardation or intellectual deficit, the current concept of adaptive functioning has seen relevance in every area of development and diagnosis (e.g., autism spectrum disorders, mental retardation/intellectual disability, traumatic brain injury; specific forms of developmental disorders, such as Prader–Willi syndrome, fragile X

syndrome; other genetic disorders; giftedness; and normal human development).

The administration of VSMS does not require the participation of the individual whose adaptive behavior is being assessed but only requires a respondent who is familiar with the individual's behavior. This "third-party" method of administration yields a valid measurement of the day-to-day activities that cannot be adequately measured through direct administration of tasks. The semi-structured interview method also allows assessment of individuals who will not or cannot perform on command in a direct administration situation, such as infants, individuals with severe or profound mental retardation, individuals with severe emotional disturbances, and individuals with physical disabilities.

Historical Background

The publication of the *Vineland Social Maturity Scale*, by Edgar Doll, in 1935 (Doll, 1935) represented a landmark event in the field of psychological assessment of mental retardation or intellectual disabilities. Prior to this publication, the classification of an individual as having mental retardation was based solely on significantly delayed cognitive development as measured by IQ tests. However, Doll noted that ability assessments of individuals with mental retardation are incomplete without valid estimates of adaptive behavior. According to Doll, the primary focus of assessment of individuals with mental retardation should be on their capacity for maintaining themselves and their affairs. Doll's concern was to identify the relationship between mental deficiency and social competence, which he defined as "the functional ability of the human organism for exercising personal independence and social responsibility" (Doll, 1953, p. 10). In his criteria of mental deficiency, Doll (1940) listed social incompetence as first and most important. Because the immediate occasion for suspicion of mental deficiency is a social circumstance, Doll wrote, no mental diagnosis is complete if it does not begin with a sound estimate of social competence and end with a prediction of social competence following prognosis or treatment.

The *Vineland Social Maturity Scale* was translated into many languages and for many years was employed throughout the world to assess individuals with cognitive delays or other handicapping conditions. It is still in use in some parts of the world but has mainly been replaced by its revision: the Vineland Adaptive Behavior Scales (Sparrow, Balla, & Cicchetti, 1984a, b) and the Vineland Adaptive Behavior Scales II (Sparrow, Cicchetti, & Balla, 2005, 2008).

Psychometric Data

When Doll developed the VSMS in 1935, he was ahead of his time in employing appropriate psychometric procedures and in standardizing the test on a sample of individuals from Vineland, New Jersey. The VSMS was the first standardized measure of adaptive behavior. Although, Doll's sample was not a national representative one, it represented for that time a sophisticated understanding of how psychological tests should be developed.

Clinical Uses

Traditionally, the VSMS was used along with a cognitive assessment to help make a diagnosis of mental retardation or intellectual disability. It has also been used extensively to assess the development of activities of everyday life in individuals who are difficult to test directly because of some handicapping condition but not necessarily intellectually impaired.

Cross References

► Vineland Adaptive Behavior Scales

References and Readings

Doll, E. A. (1935). A genetic scale of social maturity. *The American Journal of Orthopsychiatry, 5*, 180–188.

Doll, E. A. (1940). The social basis of mental diagnosis. *Journal of Applied Psychology, 24*, 160–169.

Doll, E. A. (1953). *Measurement of social competence.* Circle Pines, MN: American Guidance Service Inc.

Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1984a). *The Vineland adaptive behavior scales: Interview edition, survey form.* Circle Pines, MN: American Guidance Service.

Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1984b). *The Vineland adaptive behavior scales: Interview edition, expanded form.* Circle Pines, MN: American Guidance Service.

Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1985). *The Vineland adaptive behavior scales: Classroom edition.* Circle Pines, MN: American Guidance Service.

Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales: Second edition (Vineland II), survey interview form/ caregiver rating form.* Livonia, MN: Pearson Assessments.

Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2008). *Vineland adaptive behavior scales: Second edition (Vineland II), The expanded interview form.* Livonia, MN: Pearson Assessments.

VIQ

► Intelligence
► Verbal IQ

Viral Encephalitis

► Herpes Simplex Encephalitis

Visceral Nervous System

► Autonomic Nervous System

Visual Agnosia

Giulia Righi, Michael J. Tarr
Brown University
Providence, RI, USA

Short Description or Definition

Visual agnosia is a neurological deficit that results in impairments in the perception and recognition of complex visual stimuli such as common objects or faces, while low-level visual processes and the memory systems remain intact. The primary cause of these deficits is damage in the lateral part of the occipital lobes, and/or in the ventral portion of the temporal lobes.

Categorization

Visual agnosias can be divided into two main types: apperceptive visual agnosias and associative visual agnosias. This distinction was first put forth by Lissauer (1890), who suggested a pathological difference between (1) the inability to correctly perceive an object as a coherent whole because of perceptual deficits, and (2) the inability to ascribe meaning to an object despite an accurate perception of that object because of deficits in accessing the stored object representations. He dubbed the former as "apperceptive," and the latter as "associative."

Neuropsychology and Psychology of Visual Agnosia

The earliest documented cases of visual agnosia date back to the late-1800s. Lissauer was the first scientist to formally report a patient with visual agnosia. He suggested that the ability to recognize objects could be affected by brain damage independently from low-level visual perception. Moreover, he proposed that the visual recognition of objects consisted of two separate stages: the apperceptive stage and the associative stage. During the apperceptive stage, a sensory representation of an object is created, while during the associative stage, knowledge connected to that sensory representation is retrieved so that a visual stimulus can be recognized (Lissauer, 1890). The idea of two distinct processing stages led Lissauer to propose that brain injury could affect either of the two processes independently, giving rise to distinct symptomologies and two different forms of agnosia (Lissauer). This distinction is still used as the general framework for understanding agnosias in modern neuropsychology.

Apperceptive visual agnosias include different types of perceptual impairments that cause the misperception of objects despite intact low-level visual processes (Farah, 1990). For example, patients diagnosed with apperceptive agnosia may demonstrate difficulties in grouping sets of local features that represent full objects or the components of a visual scene (Farah & Feinbergh, 2006). Apperceptive agnosias are most commonly caused by large lesions encompassing the lateral portion of the occipital lobes and extending into the temporal lobes. Common causes of these types of lesions are stroke, anoxia, and carbon monoxide poisoning (c.f. Ghadiali, 2004). Because of the heterogeneity of deficits found in apperceptive agnosia patients, Farah (1990, 2004) has proposed several subtypes: a narrow definition of apperceptive agnosia, ventral simultagnosia, and a perceptual categorization deficit.

According to Farah (1990, 2004), pure apperceptive agnosia is a deficit in general shape perception and discrimination. Apperceptive agnosic patients show reasonably intact performance on tasks measuring visual acuity, color vision, and motion detection, while they are greatly impaired on tasks requiring them to recognize, match, and copy visual stimuli as simple as Xs and Os, or as

complex as common objects (Farah, 1990). These patients will usually exhibit large diffuse lesions in the occipital lobes.

Ventral simultagnosia was first defined as the inability to process more than one object, or more than one part of a complex object, at any one time (Wolpert, 1924, as reported in Farah, 1990). In contrast to pure apperceptive agnosics, these patients can recognize objects and use shape information, but they report not "seeing" more than any one element at the time, regardless of where the objects are in the visual field (i.e. it is not specific to a side of space, as in spatial neglect). For example, when presented with complex visual scenes, these patients are able to recognize individual objects in a scene, but cannot make sense of a scene as a whole. It has been suggested that simultagnosia is a by-product of a reduction in visual attention, resulting in a functionally reduced visual field (Michael & Henaff, 2004). Simultagnosia generally arises from large bilateral lesions extending from the occipital lobes to the temporal or parietal lobes (Farah, 1990, 2004).

Finally, "perceptual category deficits" comprises a third subtype of apperceptive agnosia (Warrington and colleagues, as reported in Farah, 1990). Patients exhibiting this impairment have difficulties in recognizing objects in unconventional views, despite being able to do so when the objects are presented in their canonical orientation. The few case studies of patients with this sort of deficit report lesions to the "right posterior quadrant of the brain, especially the right posterior inferior parietal lobe" (Farah, 1990, 2004). More recently, Mulder and colleagues (1995) observed patients with similar deficit with a similar left-lateralized lesion, suggesting that the side of the lesion might not be diagnostic of this deficit.

Unlike the majority of patients with apperceptive agnosias, patients with associative visual agnosia can "see" and perceive objects and their parts, but they cannot assign meaning to them. These deficits are usually associated with damage to the inferior-temporal occipital junction and the ventral temporal cortex. The lesions can be caused by traumatic brain injury, infarction of the posterior cerebral artery, and less frequently by tumors, hemorrhages, and demyelination (Ghadali, 2004).

Similar to apperceptive visual agnosias, several subtypes of the general syndrome have been identified on the basis of the different kinds of selective impairments observed across the patient population. Patients afflicted with associative visual agnosia can present either an impairment in recognizing many different categories of objects, or they can present recognition impairments that are either specific to a single object category (i.e. faces or letters) or that seem to affect general subcategories of objects much more so than others (i.e. man-made objects or living things).

The general form of associative agnosia can be defined as an impairment in recognizing, naming, matching, and at times, but not in all cases, drawing objects from many different categories, despite intact recognition using modalities other than vision, and normal low-level visual perception (Farah, 1990, 2004). These deficits are restricted to the visual modality, as these patients can recognize objects if presented to them via a different sensory modality. This general impairment is usually caused by large bilateral lesions encompassing portions of both the occipital and temporal lobes (Farah & Feinberg, 2006).

There have been many case studies reporting patients who show difficulties in recognizing specific object categories (De Renzi & di Pellegrino, 1998). Such case studies have given rise to a taxonomy of category-specific visual agnosias. The most prominent of these selective impairments has been termed "prosopagnosia" (Bodamer, 1943), in reference to a deficit in visual recognition that is mostly or entirely specific to faces. Prosopagnosic patients are unable to recognize familiar people, including family members, from their faces, and have difficulty in learning and remembering new faces. The lesions found in these patients are either right-lateralized or bilateral and include either or both lateral occipital areas and ventral temporal lobe areas. There have also been reports of selective impairments specific to visual letter and visual word recognition. In contrast to patients suffering from face recognition deficits, these patients show left-lateralized lesions in occipito-temporal areas. Farah and colleagues have found that the maximum overlap across patients with letter and word deficits is a lesion site in the left parahippocampal gyrus, fusiform gyrus, and lingual gyrus (Farah & Feinberg, 2006). Finally, there are several case studies reporting patients with impaired visual recognition for objects in categories that are associated by their meaning or function rather than by visual similarity. For example, Warrington and Shallice (1984) described a double dissociation between patients showing a deficit in recognizing living things, but normal recognition of non-living things, and patients showing a deficit in recognizing non-living things, but normal recognition of living things.

Across the literature, there are many instances in which patients show more than one of these object-selective impairments. For example, there are patients who show deficits on face recognition *and* word recognition, but have

relatively spared object recognition. Alternatively, some patients show impaired word and object recognition, but relatively spared face recognition. Such co-occurrences are consistent with a systematic pattern of lesion sites, such that a single selective impairment usually results from uni-lateral lesions, while two or more impaired categories are usually the result of bilateral lesions (Farah & Feinberg, 2006).

The nature of associative agnosia is still somewhat controversial in that it is unclear whether the deficit is a disorder of perception, a disorder of semantic memory, or possibly a disorder across both domains. One account has proposed that "pure" associative agnosias are the result of damaged stored visual representations in the absence of perceptual deficits, such that when a new stimulus is processed by an intact visual system, there is no stored representation available for matching it with the sensory input (Riddoch & Humphreys, 2003). However, Farah and colleagues have proposed that perceptual-level deficits may exist in such patients, because they typically exhibit very slow, feature-by-feature strategies in processing visual stimuli, even when they successfully perform a task. Such findings hint that both perception and memory are functioning abnormally in these patients (Farah & Feinberg, 2006).

Evaluation

Case studies of visual agnosic patients employ a variety of tasks to determine the specificity of the deficit encountered. Some of the most widely used tests used are the Birmingham Object Recognition Test (BORB; Riddoch & Humphreys, 1993), the Visual and Space Perception Battery (VOSP; Warrington & James, 1991), the Graded Naming Test (McKenna, 1997), and the Pyramid and Palm Trees test (Howard & Patterson, 1992).

The BORB is the most comprehensive of these measures, as it is intended to diagnose any visual object recognition impairment, including both the apperceptive and associative types of visual agnosia. The BORB contains behavioral tests to measure low-level visual perception, object perception, and retrieval of semantic knowledge about objects. The VOSP contains a series of tests designed to assess object and space perception. The Graded Naming Test is designed to measure patients' ability to name objects taken from many different categories from line drawing representations. The Pyramid and Palm Trees test is designed to assess object recognition in patients who may also have verbal impairments.

Treatment

There is no direct treatment for visual agnosias. However, patients can benefit from rehabilitation teaching them alternative strategies to compensate for their specific deficits, and also from repetitive training to attempt to recover some of the impaired functions (Ghadiali, 2004).

Cross References

- ▶ Prosopagnosia
- ▶ Ventral Visual Pathway
- ▶ Visual Object Agnosia
- ▶ 'What System'

References and Readings

De Renzi, E., & di Pellegrino, G. (1998). Prosopagnosia and alexia without object agnosia. *Cortex, 34*, 403–415.

Farah, M. J. (1990). *Visual agnosia.* Cambridge, MA: MIT Press.

Farah, M. J. (2004). *Visual agnosia* (2nd ed.) Cambridge, MA: MIT Press.

Farah, M. J., & Feinberg, T. E. (2006). *Visual Object Agnosia, in Patient-based approaches to cognitive neuroscience.* Farah & Feinberg editors, Cambridge, MA: MIT press.

Ghadiali, E. (2004). Agnosia. *Advances in Clinical Neuroscience and Rehabilitation, 4*, 18–20.

Howard, D., & Patterson, K. (1992). *The pyramids and palm trees test.* Bury St Edmunds, UK: Thames Valley Test Company.

Lissauer, H. (1980). Ein Fall von Seelenblindheit nebst einem beitrage zue Theorie derselben. *Archiv fur Psychiatrie und Nervenkrankheiten, 21*, 22–270.

McKenna, P. (1997). *The category-specific names test.* Hove, East Sussex, UK: Psychology Press.

Michael, F., & Henaff, M. A. (2004). Seeing without the occipito-parietal cortex: simultagnosia as a shrinkage of the attentional visual field. *Behavioral Neurology, 15*, 3–13.

Mulder, J. L., Bouma, A., & Ansink, B. I. J. (1995). The role of visual discrimination disorders and neglect in the perceptual categorization deficits in right and left hemisphere damage patients. *Cortex, 31*, 487–501.

Riddoch, M. J., & Humphreys, G. W. (1993). *Birmingham Object Recognition Battery (BORB).* Hove, East Sussex, UK: Lawrence Erlbaum.

Riddoch, M. J., & Humphreys, G. W. (2003). Visual Agnosia. *Neurologic Clinics of North America, 21*, 501–520.

Warrington, E. K. (1984) *Recognition memory test.* Windsor, UK: NFER-Nelson.

Warrington, E. K., & James, M. (1991). *Visual Object and Space Perception Battery (VOSP).* Bury St Edmunds, UK: Thames Valley Test Company.

Warrington, E. K., & Shallice, T. (1984). Category-specific semantic impairments. *Brain, 107*, 829–854.

Visual Amnesia

► Visual Object Agnosia

Visual Analog Scale

Tamara Bushnik
NYU Langone Medical Center
New York, NY, USA

Synonyms

VAS

Definition

Visual analog scale (VAS) is a method of measuring a subjective construct believed to exist along a continuum. VAS typically takes the form of a straight line of a specific length, usually 100 mm, with extreme descriptors at either end. Individuals rate the characteristic or attitude of interest by placing a mark at a point somewhere along the line that represents their subjective experience. For example, a pain VAS could ask "in the past 7 days, how severe has your pain been?" with possible ratings from "no pain" to "very severe pain." Once the individual has placed a mark along the VAS continuum, the distance from one end of the line to the mark is measured and expressed in millimeters. This method has been used with aphasic individuals to obtain a measure of depression, with stylized "smiley" and "sad" faces at either end.

Cross References

► Response to Intervention

References and Readings

Stern, R. A., & Bachman, D. L. (1991). Depressive symptoms following stroke. *American Journal of Psychiatry, 148*, 351–356.

Wewers, M. E., & Lowe, N. K. (1990). A critical review of visual analogue scales in the measurement of clinical phenomena. *Research in Nursing & Health, 13*, 227–236.

Visual Angle

Joan Swearer
University of Massachusetts Medical School
Worcester, MA, USA

Synonyms

Visual subtense

Definition

Visual angle is a dimension used to indicate the size of visual stimuli subtended at the eye without having to specify actual stimulus size or distance and is used to specify intraocular dimensions.

Current Knowledge

Visual angle predicts the amount of space that an image will subtend on the retina and describes the relative location of separate retinal images. It is also used to specify the size of spatial frequency gratings. It is formed by incoming light rays at the nodal point of the eye and is dependent on the size of the stimulus, its distance from the observer, and whether or not it is viewed in the frontal plane. In a simplified model, visual angle is formed from the light rays from two points (in height, width, or depth) of a viewed object as they enter the eye and is proportional to the angle projected onto the retina. The size of the subtended image is thus determined by the visual angle. An object viewed from different distances will have different retinal sizes, as will two equally sized objects viewed at different distances. Objects of different sizes will subtend the same visual angle if positioned at appropriate distances from the viewer (De Valois & De Valois, 1988; O'Shea, 1991).

Visual angle subunits are minutes and seconds of arc: 1 degree = 60 min of arc (or arcmin) and 1 arcmin = 60 s of arc (or arcsec). Components used to calculate visual angle (θ) include the size of the stimulus object (S_0) at a specified viewing distance (D_0). Retinal image size (S_i) is based on an average image distance (D_i) of 17 mm from the lens of the eye to the retina. The geometrical formulas for the relations between visual angle, size, and distance are (Scharff, 2003):

$$S_0/D_0 = \tan\theta = S_i/D_i.$$

If the angle is not known:

$$\theta = 2 \arctan(S_0/2D_0);$$

for visual angles smaller than 10 degrees: $\theta = \arctan(S_0/D_0)$.

Cross References

► Fourier Transforms

References and Readings

De Valois, R. L., & De Valois, K. K. (1988). *Spatial vision*. New York: Oxford University Press.

O'Shea, R. P. (1991). Thumb's rule tested: Visual angle of the thumb's width is about 2 deg. *Perception, 20*, 415–418.

Scharff, L. V. (2003). Sensation and perception research methods. In S. F. Davis (Ed.), *Handbook of research methods in experimental psychology* (pp. 263–284). Malden: Blackwell.

Visual Area 6

► Dorsomedial Visual Area

Visual Component Processes

► Visual Modularity

Visual Cortex

Warren L. Felton
Virginia Commonwealth University Medical Center
Richmond, VA, USA

Synonyms

Brodmann's area 17; Calcarine cortex; Occipital cortex; Striate cortex

Definition

The visual cortex is located in the occipital lobe of the brain and is composed of the primary visual cortex (striate cortex, Brodmann's area 17) and the visual association areas (nonstriate visual cortex, Brodmann's areas 18 and 19) (Milner, 2006).

Current Knowledge

The visual cortex processes visual information including form, color, motion, and depth (Grill-Spector & Malach, 2004). Cortical visual impairment may result from cerebrovascular disease, trauma, tumor, infection, degenerative disease, toxin, medication, migraine, psychosis, and other causes. The characteristic defect is loss of visual field. Unilateral impairment of the visual cortex causes loss of the opposite hemifield in each eye, usually sparing the central ten degrees of visual field, and is termed a macular sparing homonymous hemianopia. A defect at the pole of the occipital cortex that impacts central visual field is referred to as a homonymous central hemiscotoma. Unilateral impairment above or below the calcarine fissure causes a homonymous inferior or superior quadrantanopia, respectively. Cortical blindness results from severe bilateral visual cortical insult. Some individuals with cortical blindness deny their visual loss, a condition called Anton's syndrome. Rarely, individuals with cortical blindness, unable to perceive light, may perceive other visual stimuli and are said to have blindsight.

A variety of uncommon conditions arise from damage to the visual cortex (Girkin & Miller, 2001). Cerebral dyschromatopsia is a form of loss of color perception. Visual agnosia is the loss of recognition of familiar objects. Prosopagnosia is the loss of facial recognition. Patients with visual cortical impairment may develop alexia, the inability to read, or agraphia, the inability to write, alone or in combination. Simultagnosia is the inability to recognize a complete image with preserved ability to recognize its parts. Optic ataxia is impaired hand eye coordination despite intact motor and cerebellar function. Ocular motor apraxia is the inability to initiate saccades (rapid eye movements) to command. Occurring in combination, simultanagnosia, optic ataxia, and ocular motor apraxia comprise Balint's syndrome. Cerebral akinetopsia is the inability to perceive motion. Visual perseveration occurs in different forms. Palinopsia is the immediate persistence or the delayed recurrence of a visual image. Cerebral polyopia is the perception of a single image as multiple images.

Visual hallucinations may occur as simple spots, flashing lights, or shapes, or complex images of persons or animals. Release visual hallucinations are associated with visual field defects. The Charles Bonnet syndrome refers to formed visual hallucinations. Occipital seizures cause a variety of visual phenomena, including visual hallucinations. Cerebral micropsia, macropsia, and metamorphopsia refer to the misperception of an image as too small, too large, and in distorted form, respectively. Visual disturbances associated with migraine are common and may occur without headache as acephalgic migraine or migraine equivalents. Scintillating scotoma is surrounded by sparkling lights and may form C-shaped zigzag lines, termed a fortification spectrum.

Cross References

- ▶ Brodmann's Areas of the Cortex
- ▶ Gray Matter
- ▶ Localization
- ▶ Neurologic Examination
- ▶ Occipital Lobe
- ▶ Visual Evoked Potentials

References and Readings

Edmond, J. C., & Foroozan, R. (2006). Cortical visual impairment in children. *Current Opinion in Ophthalmology, 17*, 509–512.

Girkin, C. A., & Miller, N. R. (2001). Central disorders of vision in humans. *Survey of Ophthalmology, 45*, 379–405.

Grill-Spector K., & Malach, R. (2004). The human visual cortex. *Annual Review of Neuroscience, 27*, 649–677.

Milner, A. D. (2006). *The visual brain in action* (2nd ed.). Oxford: Oxford University Press.

Visual Evoked Potentials

Flora Hammond[1], Lori Grafton[2]
[1]Indiana University
Indianapolis, IN, USA
[2]Carolinas Rehabilitation
Charlotte, NC, USA

Synonyms

Visual evoked responses (VER)

Definition

Visual evoked potentials (VEP) test how the visual system responds to stimuli, testing the visual pathway function from the retina to the occipital cortex of the brain. This test measures the electrophysiologic responses of the optic nerve, optic chiasm, optic radiations, and occipital cortex to visual stimuli (such as flashing lights or checkerboard pattern). The waveforms recorded during this test are reviewed for delays that might indicate an abnormality in function along the visual pathway.

Current Knowledge

VEP is a sensitive tool that may able to detect visual system dysfunction that was not appreciable on physical examination or magnetic resonance imaging (MRI). However, VEP is not specific in identifying the etiology of a problem for which additional clinical history and MRI may be needed. VEP is useful in assessing anterior visual conduction, such as optic nerve function, and may miss detecting a retrochiasmatic lesion (Huszar, 2006). VEP may be abnormal in optic neuritis, optic neuropathy, demyelinating disease, multiple sclerosis (MS), Friedreich's ataxia, vitamin B12 deficiency, neurosyphilis, migraine headaches, ischemic disease, tumor compressing the optic nerve, ocular hypertension, glaucoma, diabetes, toxic amblyopia, glaucoma, aluminum neurotoxicity, manganese intoxication, retrobulbar neuritis, and brain injury (Huszar, 2006). VEP may be used in acute and chronic phases of MS to reveal the presence of active or subclinical demyelinating plaques, which may play an important role in making the diagnosis of MS. VEP may be obtained in infants with suspected visual impairment to assess for abnormality along the visual pathway, which may be due to delayed visual maturation.

Cross References

- ▶ Evoked Potentials

References and Readings

Huszar, L. (2006). Clinical utility of evoked potentials. *eMedicine*. Retrieved July 9, 2007, from http://www.emedicine.com/neuro/topic69.htm

Visual Evoked Responses (VER)

► Visual Evoked Potentials

Visual Field Deficit

Joan Swearer
University of Massachusetts Medical School
Worcester, MA, USA

Synonyms

Visual field loss; Scotoma

Short Description or Definition

A visual field deficit refers to diminished or absent vision in circumscribed parts of the visual field.

Categorization

Visual field deficits are caused by lesions at different levels of the visual system. Lesions at the retinal level result in scotoma of the affected eye. Optic nerve lesions peripheral to the partial crossing of fibers at the optic chiasm usually cause visual field deficits for one eye only (i.e., unilateral or monocular, incongruent defect). Lesions of the chiasm, optic tract, lateral geniculate nucleus, optic radiations, and primary visual cortex produce deficits in the contralateral visual hemifield that are roughly congruent for both eyes (i.e., covering the same area when tested monocularly (Fahle, 2003)).

There are four general types of visual field defects. **Altitudinal defects** occur with partial damage to an optic nerve and consist of a deficit in part or all of the nasal and temporal fields limited to the upper quadrants or to the two lower quadrants of vision. **Central scotoma** is the partial or complete loss of vision at the center of the visual field caused by injury of the portion of the optic nerve carrying fibers from the macular region usually owing to bilateral injury to the occipital poles. *Paracentral* scotoma is a small defect in the paracentral visual field. **Hemianopsia** occurs from lesions of the optic chiasm and refers to a visual field deficit respecting the vertical midline. When lesions affect the decussating fibers from the nasal retina of each eye (which carry information from the temporal fields), it results in the loss of both temporal visual fields, referred to as *bitemporal hemianopsia*. *Binasal hemianopsia* results from lesions of the decussating fibers from the temporal retina of each eye. Quadrantanopia is the loss of vision in superior or inferior visual field quadrants and can be bitemporal (or homonymous). For example, a bitemporal superior quadrantanopsia is usually caused by pituitary adenomas impinging on the optic chiasm that can evolve into full hemianopsia with growth of the adenoma. **Homonymous** (identical side) **hemianopsia** results from a lesion in the optic tract or the optic radiation of one eye causing a deficit in the temporal field of that eye and in the nasal field of the other eye. Homonymous superior quadrantanopsia can occur with lesions in the anterior temporal lobe where the fiber bundles of the optic radiation are most separate. Superior quadrantanopsia also results from a lesion of the lingual gyrus of the primary visual cortex; inferior quadrantanopsia is the result of a lesion in the cuneus (Gilman & Newman, 2003).

Figure: Visual field deficits. The visual pathways carry information from the left visual field to the right visual cortex and from the right visual field to the left visual cortex. The figure depicts visual field defects (in the black areas) as patients with lesions at sites 1–6 experience them.

1. A lesion of the right optic nerve causes total loss of vision in the right eye.
2. A lesion of the optic chiasm causes bitemporal hemianopsia (loss of vision in the temporal halves of both visual fields).
3. A lesion of the optic tract causes contralateral homonymous hemianopsia (complete loss of vision in the opposite half of the visual field).
4. After leaving the lateral geniculate nucleus, the fibers representing both retinas mix in the optic radiation. A lesion of the optic radiation in the temporal lobe causes an upper contralateral quadrantanopsia (loss of vision in the upper quadrant of the opposite half of the visual field of both eyes).

5, 6. Partial lesions in the primary visual cortex lead to partial defects on the opposite side. A lesion in the upper bank of the calcarine sulcus (5) causes a contralateral inferior quadrant deficit. A lesion in the lower bank of the calcarine sulcus (6) causes a contralateral superior quadrant defect. A more extensive lesion affecting both banks of the calcarine sulcus would cause more extensive contralateral visual field loss. The central area of the visual field is

unaffected by cortical lesions (5 and 6), probably because the foveal region of the retina is represented so extensively that a single lesion is not likely to destroy the entire representation (Wurtz & Kandel, 2000).

Epidemiology

The most common visual field deficit is homonymous hemianopsia (65%), followed by quadrantanopsia and paracentral scotoma. Occipital cerebrovascular disease is the cause of most visual field defects (76.1%). Other causes include closed head trauma (11.3%), tumor (operated, 5.5%), hypoxia (3.9%), and others (Zihl, 2000).

Evaluation

Perimetry is used to test visual fields. Perimetry can be kinetic, where points of light are moved from the periphery inward until the patient sees them, or static where points of light are flashed in the visual field and the patient indicates when they are detected. Visual fields can be screened "at bedside" using a confrontation method. For example, with one eye covered and visual fixation on the examiner's nose, the patient indicates whether they detect movement of the examiner's finger in each of the visual field quadrants.

References and Readings

Fahle, M. (2003). Failures of visual analysis: Scotoma, agnosia, and neglect. In M. Fahle & M. Greenlee (Eds.), *The neuropsychology of vision* (pp. 179–258). New York: Oxford University Press.

Gilman, S., & Newman, S. W. (2003). *Essentials of clinical neuroanatomy and neurophysiology* (10th ed.) Philadelphia, PA: F.A. Davis.

Wurtz, R. H., & Kandel, E. R. (2000). Central visual pathways. In E. R. Kandel, J. H. Schwartz & T. M. Jessell (Eds.), *Principles of neural science* (4th ed., pp. 523–547). New York: McGraw-Hill.

Zihl, J. (2000). *Rehabilitation of visual disorders after brain injury.* East Sussex, UK: Psychology Press.

Visual Field Loss

► Visual Field Deficit

Visual Form Discrimination

Beth A. Jerskey
Alpert Medical School of Brown University
Providence, RI, USA

Synonyms

Benton visual form discrimination test; Visual form discrimination test (VFDT)

Description

This test assesses the ability to make fine visual discriminations. Designed in a multiple choice format, the Visual Discrimination Test (VDT) consists of 2 sample items and 16 test items that range depending on the level of difficulty. Each item has a target and four stimuli directly below the target, one of which is an identical match. The other three foils contain minor variations in placement, rotation peripheral elements, or distortion of one of the major figures. All stimuli are displayed simultaneously with no time limit. Scoring is based on a correct match (2-points), an incorrect match that includes an error involving a peripheral shape (1-point), or an incorrect match involving a major shape (0-points). Strengths of this test include its ease in administration and the relatively quick time in which it can be administered.

Psychometric Data

Normative data are relatively limited. In the original publication of this test, Benton and colleagues (1983) reported norms for 85 adults (ages 16–75). Lichtenberg et al. (1994) expanded the original norms to include a greater proportion of geriatric individuals. In addition, Caplan and Schultheis (1998) later increased the clinical usefulness of the test by providing T-scores, percentiles, and clinical descriptors. Based on the original sample, a cut-off score of 25 or above was considered normal (Benton, des Hamsher, Varney, & Spreen, 1994). Mendez et al. (1990) reported that 68% of normal subjects achieved scores of 30 or more and none scored below 23.

Lopez et al. (2005) using a heterogeneous sample of elderly individuals found that the total score reliability

was 0.74. An item analysis demonstrated that 15 of the items were within the established criteria, while 5 were found to be poor discriminators. Malina et al. (2001) in a small sample of individuals with acute brain injury found an internal consistency of 0.66. Campo and Morales (2003) reported that test–retest reliability was relatively "stable" across testing sessions with some practice effects noted in peripheral errors. These researchers also noted a significant influence of age and education, which is in contrast to the original published normative sample. Gender was not significant in either sample.

In addition to the standard VDT, two short forms of the test have been developed (i.e., 8 of the original 16 items) and have demonstrated internal consistencies of 0.62 and 0.63, respectively. The implementation of a decision rule (i.e., "if the short form score falls on or between 22 and 28, then the entire test should be administered") yields near perfect classifications of either normal or impaired (Iverson, Woodward, & Smith-Seemiller, 2000).

Caplan and Caffery (1996) demonstrated that the VDT could be used in memory recognition testing by showing the examinee the target designs immediately following the original presentation. Correctly identified targets positively correlated with education ($r = 0.33$) and negatively correlated with age ($r = -0.43$). Owing in the relatively small sample on which this was tested, more normative data are needed.

Clinical Uses

Nabors et al. (1996) demonstrated specificity and sensitivity to cognitive impairment in elderly examinees, and additional work has found it to be a valid test of visuospatial impairment of patients with Alzheimer's disease (Kaskie & Storandt, 1995; Mendez, Mendez, Martin, Symth, & Whitehouse, 1990). Stroke patients have been shown to perform poorer relative to controls on both the discrimination and recognition portions of the VDT (Axelrod & Ricker, 1995). Other patient samples include individuals with vascular dementia (Mast, MacNeill, & Lichtneberg, 2000), Parkinson's disease (Tang & Liu, 1993), aphasia (Varney, 1981), and head injury (Iverson, Woodward, & Smith-Seemiller, 2000; Wilde, Bocke, & Sherer, 2000).

Cross References

► Visual System
► Visuoperceptual

References and Readings

Axelrod, B. N., & Ricker, J. H. (1995). Clinical utility of a recognition paradigm of the visual form discrimination test. *Applied Neuropsychology, 2*(3–4), 150–154.

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

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

Campo, P., & Morales, M. (2003). Reliability and normative data for the Benton visual form discrimination test. *The Clinical Neuropsychologist, 17*(2), 220–225.

Caplan, B., & Caffrey, D. (1996). Visual form discrimination as a multiple-choice visual memory test: Illustrative data. *The Clinical Neuropsychologist, 10*, 152–158.

Caplan, B., & Schultheis, M. (1998). An interpretative table for the visual form discrimination test. *Perceptual and Motor Skills, 87*, 1203–1207.

Iverson, G. L., Slick, D., & Smith-Seemiller, L. (1997). Screening for visual-perceptual deficits following closed head injury: A short form of the visual form discrimination test. *Brain Injury, 11*(2), 125–128.

Iverson, G. L., Woodward, T. S., & Smith-Seemiller, L. (2000). Internal consistency and concurrent validity of two short forms of the visual form discrimination test. *Applied Neuropsychology, 7*, 108–110.

Kaskie, B., & Storandt, M. (1995). Visuospatial deficit in dementia of the Alzheimer type. *Archives of Neurology, 52*(4), 422–425.

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

Lichtenberg, P., Millis, S., & Nanna, M. (1994). Use of the visual form discrimination test with geriatric urban medical inpatients. *The Clinical Neurophysiologist, 8*, 462–465.

Lopez, M. N., Charter, R. A., Oh, S., Lazar, M. D., & Imperio, S. M. (2005). Psychometric properties of the Benton visual form discrimination test. *Applied Neuropsychology, 12*(1), 19–23.

Malina, A., Regan, T., Bowers, D., & Millis, S. (2001). Psychometric analysis of the visual form discrimination test. *Perceptual and Motor Skills, 92*(2), 449–455.

Mast, B. T., MacNeill, S. E., & Lichtenberg, P. A. (2000). Clinical utility of the normative studies research project test battery among vascular dementia patients. *The Clinical Neuropsychologist, 10*, 173–180.

Mendez, M. F., Mendez, M. A., Martin, R., Symth, K. A., & Whitehouse, P. J. (1990). Complex visual disturbances in Alzheimer's disease. *Neurology, 40*, 439–443.

Nabors, N. A., Vangel, S. J. Jr., & Lichtenberg, P. A. (1996). Visual form discrimination test with elderly medical inpatients. *Clinical Gerontologist, 17*, 43–53.

Tang, C., & Liu, Y. (1993). Impairment of visual form discrimination in Parkinson's disease. *Acta Psychologica Sinica, 25*, 258–263.

Varney, N. R. (1981). Letter recognition and visual form discrimination in aphasia alexia. *Neuropsychologia, 19*, 795–800.

Wilde, M. C., Bocke, C., & Sherer, M. (2000). Wechsler Adult intelligence scale - Revised block design broken configurations errors in nonpenetrating traumatic brain injury. *Applied Neuropsychology, 7*, 208–214.

Visual Form Discrimination Test (VFDT)

► Visual Form Discrimination

Visual Hallucination

G. Alex Hishaw, Steven Z. Rapcsak
Neurology Section, Sourthern Arizona VA Health Care System, University of Arizona
Tucson, Arizona, USA

Synonyms

Visual illusions

Short Description or Definition

Visual hallucinations can be defined as false sensory experiences that occur in the absence of an external stimulus. The spectrum of abnormal visual sensations can range from simple images of dots, lines, flashing lights, or geometric shapes to elaborate and vivid images of people, animals, objects, or scenes.

In contrast to hallucinations, the term visual illusion has been applied to situations in which an external stimulus is present but the perception of the image is altered in terms of shape, size, number, location, movement, or temporal duration. For example, an individual's face may seem stretched and distorted, with the abnormal visual image persisting after the person leaves the room.

Although the traditional distinction between hallucinations and illusions is firmly established in the neuropsychological literature, a strict taxonomic separation between these two entities may not be fully justified or relevant owing to similar etiologies, lesion correlates, and underlying pathophysiological mechanisms. Furthermore, distinguishing hallucinations from illusions can be difficult in clinical practice, and differentiating these conditions has limited implications for the medical evaluation or treatment of the patient. For these reasons, in this text, the term visual hallucination is used to refer to all abnormal visual sensations.

Categorization

A simplified taxonomy of visual hallucinations based on the perceptual characteristics of the abnormal sensory experience is provided in Table 1. This phenomenological approach is justified by accumulating evidence for the fact that the content of the hallucinations reflects the location of the underlying neurophysiological disturbance within cortical visual areas. For instance, intra-operative studies in patients with epilepsy have shown that electrical stimulation of primary visual cortex produces simple or "unformed" hallucinations of lines, dots, or geometric patterns, whereas stimulation of temporal lobe extrastriate visual areas produces complex or "formed" hallucinations of people, animals, or objects (Penfield & Perot, 1963). More recently, neuroimaging studies have demonstrated a link between the content of hallucinations and increased activation in functionally specialized regions within extrastriate visual cortex (Allen, Laroi, McGuire, & Aleman, 2008; ffytche et al., 1998; ffytche, 2008). In particular, during hallucinations, neural activity increases in the same cortical modules that are involved in processing the specific stimulus attribute during normal visual perception. Thus, the fusiform face area is activated during the hallucinatory experience of seeing faces, while a different ventral visual area specialized for processing color information becomes activated during hallucinations of colors. Taken together, these observations suggest that the common neurobiological basis of visual hallucinations is increased physiological activity in cortical visual areas, with the phenomenology of the hallucinations reflecting

Visual Hallucination. Table 1 Phenomenological classification of visual hallucinations

Simple or unformed hallucinations: dots, lines, flashing lights, color blobs, elementary geometric shapes
Complex or formed hallucinations: elaborate visual images of faces, people, animals, objects, or scenes
Micropsia: images appear smaller
Macropsia: images appear larger
Metamorphopsia: images appear distorted
Polyopia: multiplication of images
Oscillopsia: images appear to be moving back and forth
Allesthesia: lateral transposition of images
Palinopsia: abnormal persistence of images
Tessellopsia: images resembling brickwork, lattice, grid, or netting

the anatomical and functional organization of the visual system (ffytche & Howard, 1999; Santhouse, Howard, & ffytche, 2000).

Epidemiology

Visual hallucinations represent a common clinical problem owing to the fact that they can be encountered in a variety of ophthalmologic, neurologic, toxic-metabolic, and psychiatric disorders (Cummings & Mega, 2003; Tekin & Cummings, 2003) (Table 2). Despite the diversity of disease processes that can lead to visual hallucinations, there are striking similarities in terms of the content of these abnormal sensory experiences. Thus, visual hallucinations due to eye pathology may resemble hallucinations produced by central nervous system lesions, focal seizures, or toxic-metabolic states. The phenomenological overlap and stereotypical appearance of visual hallucinations across a wide range of disease etiologies suggest related physiological mechanisms and neural substrates.

Visual Hallucination. Table 2 Common causes of visual hallucinations

Ophthalmologic Disorders
Charles–Bonnet syndrome related to visual loss due to macular degeneration, glaucoma, cataracts, etc.
Neurologic Disorders
Focal hemispheric lesions involving geniculostriate visual pathways
Brainstem/thalamic lesions (peduncular hallucinosis)
Degenerative disorders, including Alzheimer's disease, dementia with Lewy bodies, Parkinson's disease
Epilepsy
Narcolepsy (hypnagogic hallucinations)
Migraine
Medical Disorders
Toxic-metabolic encephalopathy or delirium
Drug-induced hallucinations (e.g., anticholinergics, dopaminergic agents, hallucinogens)
Drug or alcohol withdrawal
Psychiatric Disorders
Schizophrenia
Mania
Depression
Post-traumatic stress disorder (PTSD)

Natural History, Prognostic Factors, Outcomes

The prognosis in patients experiencing visual hallucinations is primarily determined by the nature of the underlying disease process. Hallucinations in the setting of toxic-metabolic encephalopathy or delirium may resolve quickly following successful medical treatment of the underlying cause. Similarly, drug-induced hallucinations promptly abate once the offending agents are withdrawn. Hallucinations occurring in the context of seizures or migraines are typically brief in duration and may be eliminated with appropriate pharmacological management. Patients with eye pathology or focal brain lesions may experience recurrent visual hallucinations for an extended period of time, but spontaneous resolution is frequently observed. Hallucinations associated with neurodegenerative disorders may progress over time and show limited response to treatment.

Neuropsychology and Psychology of Visual Hallucinations

The main challenge for neuropsychological models of visual hallucinations is to explain the specific mechanisms by which the different disease processes and lesion sites listed in Table 2 produce pathologically increased neural activation within cortical visual areas. Although a unified theory is currently beyond our reach, a number of hypotheses have been offered to provide an answer to this important question. In this section, the authors briefly review various proposals regarding the neurobiological basis of visual hallucinations. It should be noted that the different theoretical accounts have several elements in common and therefore should not be considered mutually exclusive or incompatible.

Perceptual Release Theory

It has been proposed that the reduction or loss of normal afferent visual input may result in increased spontaneous activity and/or hyper-excitability within cortical visual areas through a process of disinhibition (Burke, 2002; Manford & Andermann, 1998; Vaphiades, Celesia, & Brigell, 1996). This mechanism might explain how visual impairment caused by eye disease or focal damage to geniculostriate visual pathways can both give rise to "release" visual hallucinations. In patients with lesions involving central

visual pathways, the hallucinations are typically restricted to the hemianopic field. Relatively small lesions confined to primary visual cortex are most likely to produce visual hallucinations, pointing to the critical role of preserved extrastriate cortical regions in the generation of abnormal visual experiences. Perceptual release phenomena may also explain visual hallucinations in normal individuals under conditions of sensory deprivation.

Defective Brainstem/Thalamic Modulation of Neural Activity in Cortical Visual Areas

Although not directly involving cortical visual areas, focal brainstem/thalamic lesions may also be associated with visual hallucinations. A typical example is the syndrome of peduncular hallucinosis following damage to the rostral midbrain and/or thalamus. It has been suggested that visual hallucinations in these patients are caused by abnormal brainstem regulation of thalamic inputs to visual cortex via the lateral geniculate nucleus and pulvinar (Manford & Andermann, 1998; Mocellin, Walterfang, & Velakoulis, 2006). Brainstem structures and thalamus may also influence neural activity in cortical visual areas by modulating the sleep–wake cycle via the ascending reticular activating system, including the regulation of state-dependent switches in the firing patterns of thalamic sensory relay nuclei during transitions from wakefulness to sleep (Behrendt & Young, 2004). The anatomical/functional overlap between neural systems controlling arousal and the transmission of visual information to cortex may explain the frequent association between visual hallucinations and sleep disturbance in individuals with brainstem or thalamic pathology. In fact, it has been proposed that peduncular hallucinosis and also the hypnagogic hallucinations experienced by individuals with narcolepsy reflect the intrusion of REM dream activity during wakefulness or when in a semi-wakeful state. Decreased alertness and sleep abnormalities are also common in patients with visual hallucinations occurring in the context of toxic-metabolic encephalopathy or delirium, and REM rebound has been implicated as a possible mechanism of visual hallucinations in alcohol and sedative withdrawal (Cummings & Mega, 2003).

Neurotransmitter Theories

Neurotransmitter theories (Collerton, Perry, & McKeith, 2005; ffytche, 2007; Manford & Andermann, 1998) are motivated in part by observations that anticholinergic medications, dopaminergic drugs, and serotonergic agents can all give rise to prominent visual hallucinations (Table 2). Ascending cholinergic, dopaminergic, and serotonergic pathways originate in various brainstem and subcortical nuclei and project diffusely to neocortex, including the cortical visual areas. Neuronal loss affecting these brainstem pathways occurs in several neurodegenerative disorders, including Parkinson's disease (PD), Alzheimer's disease (AD), and dementia with Lewy bodies (DLB). The consequent reduction in neurotransmitter levels and/or the altered balance between the different neurotransmitter systems may play a critical role in producing the visual hallucinations commonly observed in these conditions. In particular, it has been suggested that decreased cortical cholinergic activity may contribute to visual hallucinations in patients with PD, AD, and DLB (Collerton et al., 2005). In addition to the proposed cortical cholinergic deficit, the loss of brainstem cholinergic neurons may promote the occurrence of hallucinations by interfering with the normal transmission of information to cortical visual areas via thalamic relay nuclei and/or by a disruption of the sleep–wake cycle. Thus, cholinergic deficiency may contribute to visual hallucinations by several different neurophysiological mechanisms. Visual hallucinations in patients with focal brainstem lesions may also reflect damage to ascending cholinergic pathways and the subsequent loss of direct and indirect cholinergic modulation of neural activity in cortical visual areas. Although the authors have focused here on the cholinergic system, brainstem pathology involving ascending dopaminergic, serotonergic, and noradrenergic projections may also play a role in the pathogenesis of visual hallucinations (Behrendt & Young, 2004; Collerton et al.; Manford & Andermann).

Cortical Irritation

As noted earlier, direct electrical stimulation of cortical visual areas produces visual hallucinations and spontaneous discharges arising from these brain regions is the most likely mechanism of ictal hallucinations in patients with focal epilepsy. Changes in cortical excitability may also be responsible for visual hallucinations occurring during migraine attacks (Pietrobon & Striessnig, 2003).

It is important to emphasize that in many disease states, visual hallucinations may reflect the combination of several different underlying mechanisms. For instance, in neurodegenerative disorders (PD, AD, DLB), hallucinations may be attributable to cholinergic dysfunction, abnormal sleep regulation, the local accumulation of neuropathological

changes in cortical visual areas (e.g., Harding, Broe, & Halliday, 2002), and the side effects of the medications used to treat these disorders. Similarly, hallucinations in elderly individuals with Charles–Bonnet syndrome are more likely to occur under conditions of decreased arousal/drowsiness or when there is evidence of co-existing cognitive impairment. Thus "pure" hallucinatory syndromes may be relatively uncommon and in most patients multiple factors may be contributing to the generation of abnormal visual experiences.

It should also be readily apparent from our discussion that a comprehensive theoretical account of visual hallucinations produced by damage to a variety of brain regions requires an integrated neural network approach. The evidence reviewed here is consistent with the notion that hallucinations reflect both local increases in neural activity within striate or extrastriate cortex that determine the content of the abnormal sensory experiences and dynamic changes in network connectivity between visual cortical areas and other cortical (frontal and parietal) and subcortical (brainstem and thalamus) regions (Behrendt & Young, 2004; Collerton et al., 2005; ffytche 2007, 2008; Manford & Andermann, 1998; Mocellin et al., 2006). Understanding the complex functional interactions between the different components of this large-scale neural network will be critical not only for elucidating the neurobiological mechanisms of visual hallucinations but also for exploring the relationship between hallucinations, dreams, visual imagery, and normal conscious visual perception.

Evaluation

The evaluation of patients with visual hallucinations begins with the taking of a detailed medical history. This should include questions regarding the specific content of the hallucinations and also the gathering of information about frequency, duration, time/place of occurrence (e.g., at night in bed just before falling asleep), and possible environmental triggers (e.g., poor lighting conditions). It is important to keep in mind that many patients with hallucinations are reluctant to report or complain about their abnormal visual experiences. It should also be established whether patients demonstrate preserved insight into and appropriate emotional attitude toward their hallucinations. In each case, a determined search is made to identify any ocular pathology resulting in visual impairment. The complaint of recurrent visual hallucinations should initiate a thorough diagnostic work-up to identify the presence of any of the neurologic, medical, or psychiatric conditions listed in Table 2. A detailed review of medications is critical, as a large number of prescription and over-the-counter drugs have the potential for producing visual hallucinations. If a focal brain lesion, epilepsy, or sleep disorder is suspected as the underlying cause, then neuroimaging studies, EEG recordings, or polysomnography may be indicated.

Treatment

When insight into the hallucinations is preserved, such as may be the case in individuals with Charles– Bonnet syndrome or occipital stroke, simple reassurance may be sufficient. These types of hallucinations may be time-limited and often resolve spontaneously. Withdrawal of medications that may cause hallucinations should always be attempted. Antipsychotic drugs may be required in situations where the hallucinations are associated with a delusional state or inappropriate emotional reaction, or when they occur in patients with psychiatric disorders. Anticonvulsants are effective in treating ictal hallucinations due to epilepsy, but these drugs may also have adjuvant benefit in patients with hallucinations due to neurodegenerative disorders. Cholinesterase inhibitors may have some modest benefit in reducing hallucinations in patients with AD, DLB, or PD. Stimulant medications have been used in the setting of narcolepsy, although drugs that block REM sleep may also be helpful. Occipital transcranial magnetic stimulation has been shown to cause complete cessation of visual hallucinatory symptoms, but it is unclear whether this technique is practical and/or safe to consider in long-term management (Merabet, Kobayashi, Barton, & Pascual-Leone, 2003).

Cross References

► Cortical Blindness

References and Readings

Allen, P., Laroi, F., McGuire, P. K., & Aleman, A. (2008). The hallucinating brain: A review of structural and functional neuroimaging studies of hallucinations. *Neuroscience and Biobehavioral Reviews, 32*, 175–191.

Behrendt, R-P. & Young, C. (2004). Hallucinations in schizophrenia, sensory impairment, and brain disease: a unifying model. *Behavioral and Brain Sciences, 27*, 771–830.

Burke, W. (2002). The neural basis of Charles-Bonnet hallucinations. *Journal of Neurology, Neurosurgery and Psychiatry, 73*, 535–541.
Collerton, D., Perry, E., & McKeith, I. (2005). Why people see things that are not there: A novel perception and attention deficit model for recurrent complex visual hallucinations. *Behavioral and Brain Sciences, 28*, 737–794.
Cummings, J. L., & Mega, M. S. (2003). Hallucinations. In *Neuropsychiatry and behavioral neuroscience* (pp. 187–199). New York: Oxford.
ffytche, D. H. (2007). Visual hallucinatory syndromes: Past, present, and future. *Dialogues in Clinical Neuroscience, 9*, 173–189.
ffytche, D. H. (2008). The hodology of hallucinations. *Cortex, 44*, 1067–1083.
ffytche, D. H., & Howard, R. J. (1999). The perceptual consequences of visual loss: "positive" pathologies of vision. *Brain, 122*, 1247–1260.
ffytche, D. H., Howard, R. J., Brammer, M. J., David, A., Woodruff, P., & Williams, S. (1998). The anatomy of conscious vision: An fMRI study of visual hallucinations. *Nature Neuroscience, 1*, 738–742.
Harding, A. J., Broe, G. A., & Halliday, G. M. (2002). Visual hallucinations in Lewy body disease relate to Lewy bodies in the temporal lobe. *Brain, 125*, 391–403.
Manford, M., & Andermann, F. (1998). Complex visual hallucinations: Clinical and neurobiological insights. *Brain, 121*, 1819–1840.
Merabet, L. B., Kobayashi, M., Barton, J., & Pascual-Leone, A. (2003). Suppression of complex visual hallucinatory experiences by occipital transcranial magnetic stimulation: a case report. *Neurocase, 9*, 436–440.
Mocellin, R., Walterfang, M., & Velakoulis, D. (2006). Neuropsychiatry of complex visual hallucinations. *Australian and New Zealand Journal of Psychiatry, 40*, 742–751.
Penfield, W., & Perot, P. (1963). The brain's record of auditory and visual experience. *Brain, 86*, 595–696.
Pietrobon, D., & Striessnig J. (2003). Neurobiology of migraine. *Nature Reviews Neuroscience, 4*, 386–398.
Santhouse, A. M., Howard, R. J., & ffytche, D. H. (2000). Visual hallucinatory syndromes and the anatomy of the visual brain. *Brain, 123*, 2055–2064.
Tekin, S., & Cummings, J. L. (2003). Hallucinations and related conditions. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology,* (4th ed., pp. 479–494), New York: Oxford University Press.
Vaphiades, M. S., Celesia, G. G., & Brigell, M. G. (1996). Positive spontaneous visual phenomena limited to the hemianopic field in lesions of central visual pathways. *Neurology, 47*, 408–417.

Visual Illusions

► Metamorphopsia
► Visual Hallucination

Visual Integration

► Closure

Visual Modularity

Ronald A. Cohen
Brown University
Providence, RI, USA

Synonyms

Visual component processes; Visual processing stages

Definition

Visual modularity is a conceptualization of visual function that maintains that the various properties that comprise visual perception (form, color, texture, motion, etc.) are the by-product of separate processes that occur in distinct cortical or subcortical regions of the brain (Calabretta & Parisi, 2005). These processes operate to a greater or lesser extent independent of each other, but are integrated to yield a uniform percept under normal conditions. These separate visual processes are thought of as modules, each operating with different computational characteristics that enable them to analyze and reconstruct visual input.

Historical Background

Visual modularity is an extension of a broader theoretical framework upon which philosophers, psychologists, cognitive scientists, and neuroscientists have approached the study of "mind" over the past century. In general systems theory, modularity is defined by the degree to which a system can be separated into individual components, recombined in an organized way by those components according to certain rules that govern their interrelationship. Principles of modularity have been integrated and employed into the sciences of mathematics, biology, cybernetics, and psychology. Modularity is most easily applied to cybernetics and the development of computer programs, where solutions to problems typically involve coupling a series of independent algorithms, each of which addresses an element of the problem. In biology, modularity reflects the view that an organism is composed on separate organ systems (e.g., cardiovascular, renal, pulmonary, etc.) which interact in an organized way. At a more basic level, cells are comprised component structures in modular fashion, while metabolic pathways

also contain modular processes involving specific biochemical cascades.

Modularity has been applied in similar fashion to epistemology by philosophers studying mind–body relationships, and more recently by psychologists and other scientists studying cognition. There has been greater contention over whether modularity is a suitable framework for explaining the "mind." Elements of this debate were apparent in the earliest work of neuropsychologists who attempted to understand brain–behavior relationships. On one hand, evidence emerged from case studies showing the effects of localized brain lesions affecting language and other cognitive functions. On the other hand, the work of Lashley and other investigators suggested that associative memory was not stored in a single location, but rather seemed to be distributed in the brain. Furthermore, intellectual capacity seemed to be a function of the activity of the entire brain acting in an integrated fashion and not the result of a specific localized brain area. The alternative theoretical perspective to modularity was a holistic perspective in which cognition was viewed as an emergent byproduct of the entire cortex rather than modules. This view had a strong influence on the development of associative theories of cognition, such as the parallel distributed processing framework (McClelland & Rumelhart, 1986).

Despite the fact that there continues to be a dialectic between these different views of cognition and brain behavior relationships, there is now an overwhelming evidence for the modularity of certain cognitive functions. Language provided one of the first examples of this with lesion studies showing that distinct brain regions were involved in receptive and expressive language (Wernicke, Broca), and there is little debate at this point in time about whether language functions are lateralized, or that certain language functions occur in particular cortical regions. The visual neurosciences have provided further support for modularity, as experimental studies of laboratory animals, functional brain imaging, and clinical findings from humans have yielded converging evidence that modular functions interact to enable visual perception, visual-motor integration, and other visual functions. Yet, within modules, there also continues to be evidence of non-modular associative processes that form the content of cognitive processes.

Current Knowledge

Clinical evidence that separate areas in the occipital, parietal, and temporal lobes were responsible for different visual functions came from some of the earliest studies of focal brain lesions (Poppelreuter, 1990). Neuroscientific support initially came from controlled laboratory studies showing that visual areas 17, 18, and 19 in the monkey have different functional architectures and receptive fields (Hubel & Wiesel, 1965, 1968). Subsequently, visual modularity has been demonstrated across all six of the visual cortical areas in primates (Kaas & Collins, 2003; Chalupa & Werner, 2004). Perhaps the most dramatic example of a broad level of modularity is the distinction that has been shown between the ventral (what) and dorsal (where) processing streams (Mishkin, Ungerleider, Macko, & Yantis, 2000; Ungerleider & Haxby, 1994). The dissociation of modular visual processing components for reactive, volitional, and memory-guided saccadic and smooth pursuit eye movements demonstrates the level of specificity that now exists with respect to modularity (Deubel, Gopher, & Koriat, 1999). There is now experimental regarding the modular interactions of brain systems involved in visual perception and higher order visual processing(Chalupa, 2003; Farah 2000).

Neuropsychology has recognized the modularity of visual functions almost from its beginning as a clinical science. Dissociations between visual perceptual, visual spatial, and visual motor syndromes have been described in hundreds of articles over the past century, driven in large part by efforts to localize cognitive functions. Examples include studies aimed at characterizing the basis of visual agnosia (Cogan, 1979; Damasio & Damasio, 1983; De Renzi, Scotti, & Spinnler, 1969; Goodwin, 2002; Landis, Regard, Bliestle, & Kleihues, 1988; Whiteley & Warrington, 1977), as well as other specific syndromes such as cortical blindness, Gertsmann syndrome, constructional apraxia, prosogpagnosia, alexia without agraphia, spatial topographic disturbances, and a variety of other visual disorders.

Future Directions

There remains a sizeable gap between knowledge of the mechanisms by which perception and higher order visual functions are accomplished in the visual system from experimental visual neuroscientific inquiry, and clinical application of this knowledge for the assessment of patients with brain disorders. Most widely used neuropsychological tests for assessing visual function are useful for distinguishing between general types of visual deficits (e.g., primary perception, visual-spatial, object and face recognition, and visual-motor ability). However, these methods are not amenable to the assessment of

visual modular functions organized based on rapid developments occurring in the visual neuroscience. The incorporation of functional brain imaging methods into clinical neuropsychological assessment provides one means by which a greater level of precision in clinical visual function analysis will become possible.

Cross References

- ▶ Dorsal Visual Pathway
- ▶ Prosopagnosia
- ▶ Ventral Visual Pathway
- ▶ Visual Agnosia
- ▶ 'What System'
- ▶ 'Where System'

References and Readings

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

Cogan, D. G. (1979). Visuospatial dysgnosia. *American Journal of Ophthalmology, 88*(3 Pt 1), 361–368.

Damasio, A. R., & Damasio, H. (1983). The anatomic basis of pure alexia. *Neurology, 33*(12), 1573–1583.

De Renzi, E., Scotti, G., & Spinnler, H. (1969). Perceptual and associative disorders of visual recognition. Relationship to the side of the cerebral lesion. *Neurology, 19*(7), 634–642.

Deubel, H., Gopher, D., & Koriat, A. (1999). Separate mechanisms for the adaptive control of reactive, volitional, and memory-guided saccadic eye movements. In D. Gopher & A. Koriat (Eds.), *Attention and performance XVII: Cognitive regulation of performance: Interaction of theory and application.* (pp. 697–721). Cambridge: The MIT Press.

Farah, M. (2000). *The cognitive neuroscience of vision.* New York: Wiley-Blackwell.

Goodwin, J. (2002). Disorders of higher cortical visual function. *Current Neurology and Neuroscience Reports, 2*(5), 418–422.

Haxby, J. V., Grady, C. L., Horwitz, B., Ungerleider, L. G., Mishkin, M., Carson, R. E., et al. (1991). Dissociation of object and spatial visual processing pathways in human extrastriate cortex. *Proceedings of the National Academy of Sciences of the United States of America, 88,* 1621–1625.

Hubel, D. H., & Wiesel, T. N. (1965). Receptive fields and functional architecture in two nonstriate visual areas (18 and 19) of the cat. *Journal of Neurophysiology, 28,* 229–289.

Hubel, D. H., & Wiesel, T. N. (1968). Receptive fields and functional architecture of monkey striate cortex. *Journal of Physiology, 195*(1), 215–243.

Kaas, J. H., & Collins, C. E., (Eds.). (2003). *The primate visual system.* Boca Raton, FL: CRC Press.

Landis, T., Regard, M., Bliestle, A., & Kleihues, P. (1988). Prosopagnosia and agnosia for noncanonical views. An autopsied case. *Brain, 111*(Pt. 6), 1287–1297.

Mishkin, M., Ungerleider, L. G., Macko, K. A., & Yantis, S. (2000). Object vision and spatial vision: Two cortical pathways. In S. Yantis (Ed.), *Visual perception: Essential readings.* (pp. 296–302). New York: Psychology Press.

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

Ungerleider, L. G., & Haxby, J. V. (1994). 'What' and 'where' in the human brain. *Current Opinion in Neurobiology, 4*(2), 157–165.

Whiteley, A. M., & Warrington, E. K. (1977). Prosopagnosia: a clinical, psychological, and anatomical study of three patients. *Journal of Neurology, Neurosurgery, and Psychiatry, 40*(4), 395–403.

Visual Neglect

Mark Mennemeier
University of Arkansas for Medical Sciences
Little Rock, AR, USA

Synonyms

Amorphosynthesis; Hemiagnosia; Hemiinattention; Left (or right) neglect; Neglect; Spatial neglect; Unilateral neglect; Visuospatial agnosia; Visuospatial neglect

Definition

Visual neglect refers to the failure of a patient to report, respond, or orient to external visual stimulation or mental images of objects and scenes that are positioned contralateral to the brain lesion, which caused neglect (i.e., contralesional)(Heilman, Watson, & Valenstein, 1985). Further, the failure is not due to a primary sensory or motor deficit such as hemianopia or paralysis. This entry describes neglect because of its effects on visual processing, with an emphasis on its assessment, types, spatial dimensions, causes, and treatment. It is important to note that what can be said about visual neglect can also be said about neglect in other sensory modalities. The reader can consult "▶ The Neglect Syndrome" in this volume for a more comprehensive discussion. Several text books on neglect are available for further reading (Karnath, Milner, & Vallar, 2002; Robertson & Halligan, 1999; Robertson & Marshall, 1993).

Current Knowledge

One of the more striking presentations of a patient with acute neglect following a right hemisphere stroke is a misalignment of the head and eyes in relation to the rest

of the body. The patient is often lying in the bed with his or her head turned almost completely to the right (or ipsilesional side), and the eyes are deviated rightward as if the person is searching for something on that side of the bed. Addressing the patient from his or her left side often fails to elicit a response. The examiner may straighten the patient's head, only to find it back in the same position moments later. Testing patients at this stage may be complicated by difficulties in getting them to orient to or "see" the test stimuli. Placing stimuli at the body midline may be too far left for the patient to acknowledge them. Misalignment of the eyes and head becomes less dramatic as the recovery progresses, but a slight rightward deviation of the eyes may continue to be observed well into the chronic stages of the stroke. These outward manifestations of brain injury upon gaze and head orientation reflect the dramatic way in which visual attention, perception, and mental representation can be biased in neglect.

Assessment

Tests for visual extinction, line bisection, target cancellation, drawing or copy tasks, and reading tasks are most frequently used to assess visual neglect. These tests have proved to have varying degrees of sensitivity in a large series of patients with right hemisphere lesions (Azouvi et al., 2002); however, the sensitivity of each test can vary dramatically according to how it is constructed and administered (Mennemeier, Rapcsak, Dillon, & Vezey, 1998), and test performance can vary according to lesion location (McIntosh & Milner, 2005). Visual neglect is most commonly observed on the contralesional side of the body midline, but can also occur ipsilateral to brain injury (i.e., ipsilesional neglect) (Kim et al., 1999).

Visual extinction to double simultaneous stimulation is tested initially by asking patients to report a visual stimulus presented individually to each visual hemifield. Next, they are asked to report the same stimulus delivered either to one visual hemifield or simultaneously to both hemifields. After confirming that the patient can detect individual stimulation of both visual hemifields, the failure to report stimulation delivered to one hemifield when both are stimulated simultaneously confirms extinction and indicates hemi-inattention to visual stimuli (Heilman et al., 1985).

The sensitivity of line bisection to biases in spatial orientation and length estimation makes it an excellent test for visual neglect. Line bisection has been studied extensively in both neglect patients and normal subjects (Jewell & McCourt, 2000). Visual neglect on line bisection is indicated by a mark placed on the ipsilesional side of the line's true center (Schenkenberg, Bradford, & Ajax, 1980). An error measuring more than 2% of the line's total length lies outside of the boundary for normal performance (Mennemeier, Vezey, Chatterjee, Rapcsak, & Heilman, 1997). Patients with neglect from right hemisphere injury are strongly biased toward the right (or ipsilesional) end of the line. Cuing patients to the opposite end of the line can reduce the size of the bisection error, but cuing is typically insufficient to overcome neglect or to reverse the direction of neglect error. Line bisection also involves estimating the length of the lines. Like spatial orientation, length estimation is biased in neglect. Longer line lengths (e.g., greater than 10 cm in length) are underestimated and shorter line lengths (e.g., less than 2 cm in length) are overestimated (Chatterjee, 1995; Tegner & Levander, 1991). The combination of biases in spatial orientation and length estimation may explain a paradoxical pattern of performance on line bisection known as the crossover effect, in which short lines are bisected on the contralesional side of the true center whereas long lines are bisected on the ipsilesional side of the true center (Mennemeier et al., 2005).

The cancellation test is one of the most sensitive assessments of visual neglect (Azouvi et al., 2002). In its simplest form, the patient is instructed to mark out target items that are distributed across a page in a pseudo-random fashion (Albert, 1973); however, the sensitivity of cancellation tests can be increased by embedding targets among distractors (Lezak, 1983). Similar to line bisection, the cancellation test elicits biases in spatial orientation. The severity of visual neglect on cancellation testing can range from missing only a few targets in the lower contralesional quadrant of the page to missing all but a few targets that hug the ipsilesional edge of the page (see Fig. 1 ▶ Neglect Syndrome). Additionally, cancellation performance is determined, in part, by the total number of target items presented (Mennemeier et al., 1998). For example, a patient might not miss any targets on a page if only a small total number of target items are presented (e.g., 16), but they may begin to demonstrate neglect as the total number of target items increases (e.g., 32). Unlike line bisection, the cancellation test also requires sustained attention, visual searching, remembering items that have already been canceled, and response inhibition (Lezak, 1983). Patients with neglect have impairments of spatial working memory (McIntosh & Milner, 2005) and the added demands of searching for targets on may cause some patients to return to targets that have already been canceled and cancel them again. Additionally, search strategies on cancellation tests are

often disorganized in patients with brain injury relative to normal control subjects. Disorganized search may be indicative of poor executive planning, although it may not be specific to patients with neglect (Mark, Woods, Ball, Roth, & Mennemeier, 2004).

Drawing and copy tasks are also sensitive to visual neglect (Azouvi et al., 2002). Like cancellation they elicit biases in spatial orientation, executive planning, and spatial working memory. Drawing tests for visual neglect typically require patients to either copy a complex scene with details distributed across a page or to copy a single object, typically a symmetrical object such as a clock face or a daisy (see Fig. 2 ► Neglect Syndrome) (Lezak, 1983; Spreen & Strauss, 1991). Alternatively, a patient may be asked to draw a well-known object from memory (see Fig. 3 ► Neglect Syndrome). Neglect is indicated by omitting elements of a complex scene or details of a single object located in contralesional space. Patients often draw the picture on the ipsilesional side of the paper. Crowding of contralesional details into the ipsilesional side of a drawing is characteristic of drawings in patients with neglect. For example, when drawing a clock face patients may place all of the numbers from 1 to 12 on the ipsilesional side, or they may distort the clock face with uneven spacing and large gaps between numbers on the contralesional side. Patients with visual neglect often have difficulty rounding the contralesional side of the clock face or making objects like a daisy appear symmetrical.

Patients with left visual neglect may start reading sentences or paragraphs in the middle of the line or page (Chatterjee & Mennemeier, 1998; Lezak, 1983). When reading single words, they may either omit left-sided letters or insert letters (i.e., confabulation) such as reading the word "cowboy" as "boy" or reading the word "nut" as "peanut," respectively (Heilman et al., 1985). Reading tests are sensitive to the inability of some patients to orient their gaze to the contralesional side of the line, paragraph, or word. Confabulatory responses in reading may be attributable to attentional deficits and to bias in estimating the total number of letters or words presented (Chatterjee, 1995).

Types of Neglect

Perceptual neglect refers to neglect for external stimulation, such as that described above. Representational neglect refers to neglect for mental images of a scene or object (Bisiach & Luzzatti, 1978). The first cases of representational neglect to gain widespread attention described two patients who, when asked to describe a well-known piazza in Milan, the Piazza Del Dumo, only reported landmarks that would have appeared on their ipsilesional side. This was true even when the patients were asked to describe the Piazza from the opposite perspective. Similar phenomena have been described in other patients using other types of representational tasks. Perceptual and representational neglect are dissociable phenomena, and may represent different processes or levels of severity in neglect. Not everyone who has perceptual neglect also has representational neglect and representational neglect without perceptual neglect is relatively rare (Bartolomeo, D'Erme, & Gainotti, 1994; Coslett, 1997).

Neglect occurs in multiple frames of reference and in multiple dimensions of space (Chatterjee & Mennemeier, 1998). Visual information can be represented in different frames of reference, such as those centered on the viewer, the object, or the environment (Marr & Nishihara, 1978). A viewer-centered reference frame locates an object in space with regard to the viewer, such as their left or right side or whether the object is near or far, or above or below, the viewer. An object-centered reference frame highlights the spatial properties of the object such as its inherent left or right side, top or bottom, or front or back. An environment-centered reference frame locates objects in space with reference to local topography, an object's position relative to other objects, and environmental coordinates such as the horizon or gravitational vertical. Studies aimed at disassociating the viewer-, object-, and environment-centered reference frames indicate that visual neglect occurs in each frame of reference. Neglect with regard to the viewer's eye, head, and body midline is referred to as egocentric neglect. Neglect for the intrinsic properties of objects is referred to as allocentric neglect. Neglect with reference to environmental landmarks and gravitational coordinates is referred to as environment-centered neglect.

Visual neglect is most commonly described along a horizontal axis of space but neglect also occurs along a vertical axis, typically following bilateral lesions, and along a radial axis projecting away from the viewer (i.e., vertical and radial neglect, respectively). Neglect occurring within the limits of one's reaching distance, such as on bedside testing, is referred to as peri-personal neglect, while neglect for stimuli that are out of reach is referred to as extra-personal neglect.

Causes

The neuroanatomy of visual neglect does *not* appear to be distinct from neglect occurring within any other sensory

modality (McIntosh & Milner, 2005). Neglect is most commonly associated with lesions of the right hemisphere, and of the inferior parietal cortex in particular, but it also occurs following damage to the middle and inferior frontal and anterior cingulate cortices. Additionally, the superior temporal cortex of the right hemisphere has been postulated as critical lesion location for neglect (Karnath, Ferber, & Himmelbach, 2001). Subcortical lesions associated with neglect include regions of the thalamus, basal ganglia, and midbrain (Heilman, Watson, & Valenstein, 1994; Vallar & Perani, 1987). Lesions to similar areas of the left hemisphere can also produce neglect, which tends to be less severe and recovers more quickly than neglect after right hemisphere injury (Ogden, 1987). Visual information from the geniculostriate pathway arrives in the highest visual processing areas of the posterior–superior parietal and inferior temporal cortices via the dorsal and ventral processing streams, respectively. The dorsal stream mediates spatial processing, particularly as it relates to spatial movement and position, whereas the ventral stream plays a major role in perceptual representations of objects and faces. Surprisingly, tasks that are quite sensitive to deficits in dorsal stream processing, such as reaching between objects, can be spared in patients with neglect (McIntosh & Milner, 2005). Similarly, most neglect patients do not have difficulty with tasks sensitive to ventral stream processing, such as object recognition. These observations suggest that information processing within the dorsal and ventral streams may be well-preserved in neglect.

Treatments

Several treatments for neglect have specifically targeted visual processing systems (Chatterjee & Mennemeier, 1998; Mark, 2003; Pierce & Buxbaum, 2002). For example, prism glasses have been used to shift information from the neglected visual field toward the non-neglected field. Prisms initially cause patients to miss-reach for objects, but they eventually learn to correct their reach trajectory with practice (a process called prism adaptation). After removing the prism glasses, the beneficial effects of adaptation have been observed in both behavioral testing and activities of daily living. Prism adaptation may work by engaging the intact dorsal processing stream and the cerebellum in recovery (McIntosh & Milner, 2005). Other treatments have used optokinetic and caloric-vestibular stimulation to ameliorate neglect. Both techniques induce nystagmus – a type of eye movement characterized by alternating slow phase movements in one direction and fast phase movements in the other direction. Shifts in spatial attention are thought to accompany the slow phase of the nystagmus, thereby improving attention toward contralesional space. Treatment studies of neglect have also attempted to shift the orientation of attention toward contralesional space either by stimulating or suppressing activity of the superior colliculi. Each superior colliculus directs an orienting response to contralateral space. Additionally, each superior colliculus receives input from the contralateral eye and inhibits the other superior colliculus. Some studies delivered flashes of light to a patient's contralesional eye in order to stimulate the ipsilesional superior colliculus and facilitate an orienting response to contralesional space. Other studies patched a patient's ipsilesional eye in order to suppress activity of the contralesional superior colliculus. Both strategies demonstrated some short-term benefit, but the results have been mixed and the gains often fail to generalize to other tasks or beyond the experimental setting.

Cross References

- ▶ Hemispatial Neglect
- ▶ Inferior Parietal Area
- ▶ Neglect
- ▶ Neglect Syndrome

References and Readings

Albert, M. L. (1973). A simple test of visual neglect. *Neurology, 23*, 658–664.

Azouvi, P., Samuel, C., Louis-Dreyfus, A., Bernati, T., Bartolomeo, P., Beis, J. M., et al. (2002). Sensitivity of clinical and behavioural tests of spatial neglect after right hemisphere stroke. *Journal of Neurology, Neurosurgery, and Psychiatry, 73*, 160–166.

Bartolomeo, P., D'Erme, P., & Gainotti, G. (1994). The relationship between visuospatial and representational neglect. *Neurology, 44*, 1710.

Bisiach, E., & Luzzatti, C. (1978). Unilateral neglect of representational space. *Cortex, 14*, 129–133.

Chatterjee, A. (1995). Cross-over, completion and confabulation in unilateral spatial neglect. *Brain, 118*, 455–465.

Chatterjee, A., & Mennemeier, M. (1998). Diagnosis and treatment of spatial neglect. In R. B. Lazar (Ed.), *Principles of neurologic rehabilitation* (pp. 597–612). New York: McGraw-Hill.

Coslett, H. B. (1997). Neglect in vision and visual imagery: A double dissociation. *Brain, 120*, 1163–1171.

Heilman, K. M., Watson, R. T., & Valenstein, E. (1985). Neglect and related disorders. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (2nd ed., pp. 243–293). New York: Oxford University Press.

Heilman, K. M., Watson, R. T., & Valenstein, E. (1994). Localization of lesions in neglect and related disorders. In A. Kertesz (Ed.), *Localization and neuroimaging in neuropsychology* (pp. 495–524). New York: Academic Press.

Jewell, G., & McCourt, M. E. (2000). Psuedoneglect: A review and meta-analysis of performance factors in line bisection tasks. *Neuropsychologia, 38*, 93–110.

Karnath, H. O., Ferber, S., & Himmelbach, M. (2001). Spatial awareness is a function of the temporal not the posterior parietal lobe. *Nature, 411*, 950–953.

Karnath, H. O., Milner, A. D., & Vallar, G. (2002). *The cognitive and neural bases of spatial neglect.* Oxford, New York: Oxford University Press.

Kim, M., Na, D. L., Kim, G. M., Adair, J. C., Lee, K. H., & Heilman, K. M. (1999). Ipsilesional neglect: Behavioral and anatomic features. *Journal of Neurology, Neurosurgery, and Psychiatry, 67*, 35–38.

Lezak, M. D. (1983). *Neuropsychological assessment.* New York: Oxford University Press.

Mark, V. W. (2003). Acute versus chronic functional aspects of unilateral spatial neglect. *Frontiers in Bioscience, 8*, 72–89.

Mark, V. W., Woods, A. J., Ball, K. K., Roth, D. L., & Mennemeier, M. (2004). Disorganized search on cancellation is not a consequence of neglect. *Neurology, 63*, 78–84.

Marr, D., & Nishihara, H. K. (1978). Representation and recognition of the spatial organization of three-dimensional shapes. *Proceedings of the Royal Society of London B, 200*, 269–294.

McIntosh, R. D., & Milner, A. D. (2005). The neurological basis of visual neglect. *Current Opinion in Neurology, 18*, 748–753.

Mennemeier, M., Pierce, C. A., Chatterjee, A., Anderson, B., Jewell, G., Dowler, R., et al. (2005). Bias in attentional orientation and magnitude estimation explain crossover: Neglect is a disorder of both. *Journal of Cognitive Neuroscience, 17*, 1194–1211.

Mennemeier, M., Rapcsak, S. Z., Dillon, M., & Vezey, E. (1998). A search for the optimal stimulus. *Brain and Cognition, 37*, 439–459.

Mennemeier, M., Vezey, E., Chatterjee, A., Rapcsak, S. Z., & Heilman, K. M. (1997). Contributions of the left and right cerebral hemispheres to line bisection. *Neuropsychologia, 35*, 703–715.

Ogden, J. A. (1987). The neglected left hemisphere and its contribution to visuo-spatial neglect. In M. Jeannerod (Ed.), *Neurophysiological and neuropsychological aspects of spatial neglect* (1st ed., pp. 215–234). Amsterdam: North-Holland.

Pierce, S. R., & Buxbaum, L. J. (2002). Treatments of unilateral neglect: A review. *Archives of Physical Medicine and Rehabilitation, 83*, 256–268.

Robertson, I. H., & Halligan, P. W. (1999). *Spatial neglect: A clinical handbook for diagnosis and treatment.* Hove, East Sussex: Erlbaum.

Robertson, I. H., & Marshall, J. C. (1993). *Unilateral neglect: Clinical and experimental studies.* Hillsdale, NJ: Lawrence Erlbaum Associates.

Schenkenberg, T., Bradford, D. C., & Ajax, E. T. (1980). Line bisection and unilateral visual neglect in patients with neurologic impairment. *Neurology, 30*, 509–517.

Spreen, O., & Strauss, E. A. (1991). *Compendium of neuropsychological tests: Administration, norms, and commentary.* New York: Oxford University Press.

Tegner, R., & Levander, M. (1991). The influence of stimulus properties on visual neglect. *Journal of Neurology, Neurosurgery, and Psychiatry, 54*, 882–886.

Vallar, G., & Perani, D. (1987). The anatomy of spatial neglect in humans. In M. Jeannerod (Ed.), *Neurophysiological and neuropsychological aspects of spatial neglect* (pp. 235–258). Amsterdam: North Holland, Elsevier.

Visual Object Agnosia

Kerry Donnelly
University at Buffalo/SUNY
Buffalo, NY, USA

Synonyms

Monomodal visual amnesia; Visual amnesia

Definition

Visual object agnosia is a difficulty in recognizing objects presented visually and cannot be explained by primary visual defect, mental deterioration, disorder of attention, or a lack of familiarity with the object.

Current Knowledge

In visual object agnosia, the individual retains the ability to recognize the object through sensory modalities other than vision (e.g., by touch). Two main types of visual object agnosia have been identified: apperceptive and associative. *Apperceptive visual agnosia* involves a deficit presumed to lie in the production of a stable percept arising from an impairment of higher order visual perception and is thought to result from lesions of the secondary or unimodal visual association areas. *Associative visual agnosia* stems from the disruption of the post-perceptual stage of visual processing in which, meaning is attributed to the visual percept. Unlike in the apperceptive form, patients with associative visual agnosia may have little difficulty drawing the visually presented object, but can neither name it nor explain or demonstrate its use. The anatomical substrate of associative object agnosia is presumed to involve a disruption either in the secondary visual association cortices or in the connections between these areas and the tertiary or heteromodal cortices. *Prosopagnosia* is often considered a related disorder in which, the patient has difficulty in visual recognizing faces.

Cross References

► Apperceptive Visual Agnosia
► Associative Visual Agnosia
► Prosopagnosia

References and Readings

Bauer, R., & Demery, J. (2003). Agnosia. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (4th ed., pp. 236–295). NY: Oxford.

Cambier, J., Signoret, J. L., & Bolgert, F. (1989). Visual object agnosia: Current conceptions. *Revue Neurologique (Paris), 145*, 640–645.

Visual Object Recognition

▶ Object Recognition

Visual Perseveration

▶ Palinopsia

Visual Processing Stages

▶ Visual Modularity

Visual Subtense

▶ Visual Angle

Visual Synthesis

▶ Closure

Visual System

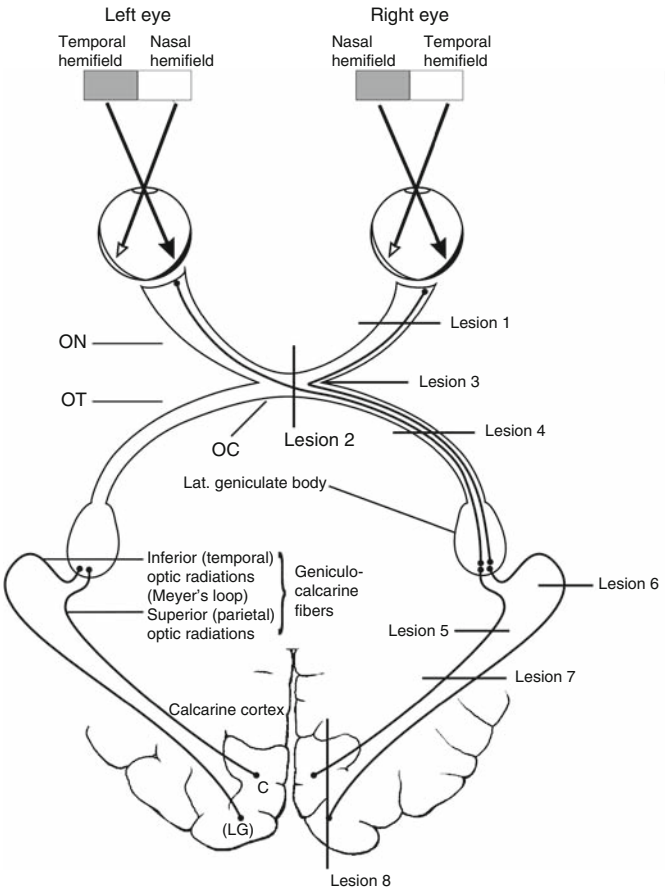
Maryellen Romero
Tulane University Health Sciences Center
New Orleans, LA, USA

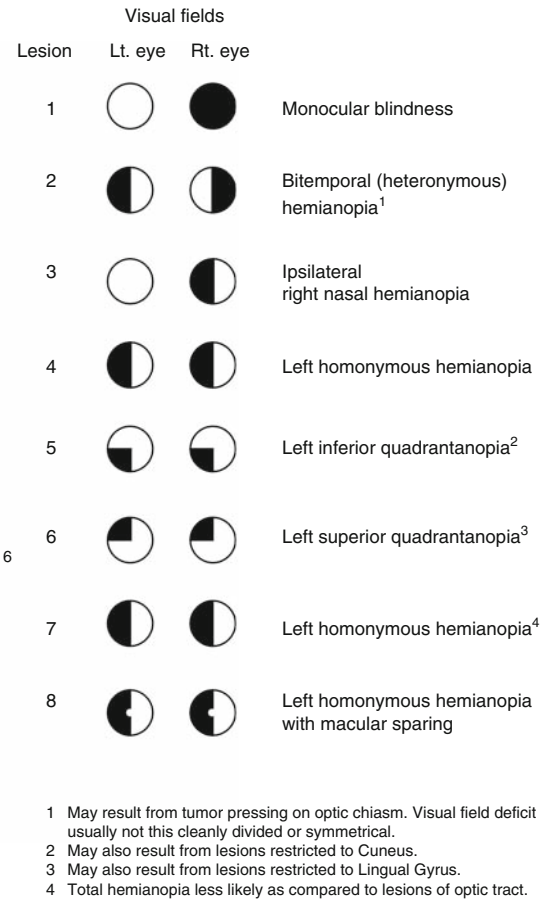
Structure

The human visual system comprises a complex collection of structures that transforms light into a rich palette of visual information that one experiences as the physical environment around us. The most recognizable element of the system is the eye, the peripheral organ of vision. Light rays are refracted by the cornea and projected through the pupil to the photoreceptive cells (rods and cones) of the retina at the back of the eye where the transformation of light energy into chemical messages begins. The rods are found with relatively greater frequency in the periphery of the retina and are better adapted to low-intensity light, such as night vision. The cones are more concentrated in the macula, particularly in the center of the macula or fovea, are better adapted to the perception of color and the sharper point-to-point vision necessary for fine discriminations (foveal vision). While other cells (horizontal cells) serve as interneurons, it is basically the rods and cones that transmit visual input to the bipolar cells that lie within the retina. These, in turn, synapse with the ganglion cells whose axons exit the eye through the optic disk to form the *optic nerves*.

As the optic nerves reach the level of the anterior hypothalamus on the ventral surface of the brain, there is a partial decussation in the *optic chiasm*. Here, the individual nerve fibers that emanated from the lateral (temporal) halves of the retinas remain ipsilateral, while those that were derived from the medial (nasal) portion of the retinas cross to the opposite side (Fig. 1). Due to the small opening (pupils) through which light rays reach the retina, light from the left side of the visual field (VF) strikes the temporal portion of the retina in the right eye and the nasal portion of the left eye (the converse being true of light from the right VF). Thus, the partial crossing of the optic nerves in the optic chiasm insures that the visual information from the left VF is sent to and processed by the right hemisphere, while that from the right VF is projected to the left hemisphere, consistent with the contralateral representation of the motor and somatosensory systems. Although they consist of exactly the same nerve fibers present in the optic nerves, once they exit the chiasm they are referred to as the *optic tracts*.

The optic tracts proceed posteriorly to synapse in *the lateral geniculates*, the thalamic relay nuclei for the visual system. Before reaching the thalamus, some fibers from the optic tract are diverted to the nuclei of the brain stem, which allow for reflex accommodations of the lens for near and distant viewing, of the pupils depending on light intensity, and the eye lids for protective blink responses. In contrast to the relative tight fiber bundles characteristic of the optic nerves and optic tracts, once leaving the lateral geniculates, the visual fibers spread out and form the *optic radiations* as they proceed to the medial aspect of the occipital lobes, the *primary visual cortex*





Visual System. Figure 1 Visual pathways and lesion effects. Abbreviations: C, cuneus; LG, lingual gyrus; OC, optic chiasm; ON, optic nerve; OT, optic tract

(Brodmann's area 17). Those portions of the optic radiations that course more ventrally actually take a bit of a forward turn in the temporal lobe before reaching the occipital cortex. This latter deviation is known as *Meyer's loop*.

As they reach the visual cortex, the optic radiations terminate on the superior and inferior banks of the *calcarine fissure* (sulcus). Those fibers representing visual information impacting on the *lower* portion of the retina (superior half of the VF) project to the inferior bank of this fissure or the *lingual gyrus*, while those whose ultimate origin was in the upper part of the retina (inferior VF) terminate on the superior bank (the *cuneus*). While some preliminary synthesis and differential processing of visual input can be said to take place in the eye itself with the various receptor and other types of cells present, it is in the occipital cortex that the final transformation of these bioelectrical impulses into what one experiences as vision takes place. While the primary visual cortex is essential for basic vision, the full richness or appreciation of visual experiences require the cooperation of adjacent cortical regions or the visual association areas of the occipital lobes.

Illness

As with other complex neural systems, damage to any part of the visual network can result in discrete or sometimes widespread visual dysfunction. At the retinal level, individuals who have lost cone-mediated vision are diagnosed as legally blind, whereas rod cell dysfunction leads to night blindness or difficulty with visual perception in low-light situations. Damage to the optic nerve can result in total blindness of the affected eye. *Amaurosis fugax* or transient monocular blindness result from a transient ischemic attack (TIA) involving the ophthalmic artery.

A pituitary tumor that exerts pressure on the center of the optic chiasm (where the nasal fibers from each are crossing) can result in a condition akin to tunnel vision where the lateral portions of both VF are affected, a syndrome known as *bitemporal hemianopsia* (see Fig. 1). Lesions involving the optic track or the lateral geniculate nucleus will typically result in the loss of vision in the field on the side opposite to that of the lesion (*homonymous hemianopsia*). While the same could theoretically occur following a lesion involving the optic radiations, because of their more widespread distribution, it is somewhat rare for all the visual fibers to be affected. A more likely scenario is that only some of the fibers will be damaged, resulting in a partial loss of vision in the contralateral VF. If such a lesion were to primarily impact the more ventral or temporal radiations, only the superior portion of the contralateral visual field would be impaired. Conversely, a deep parietal lesion involving the underlying radiations might result in an inferior defect. These conditions are respectively referred to as a *superior* or *inferior quadrantanopsia* (loss of vision in more or less a single quadrant of the VF). Should the primary visual cortex in one hemisphere be damaged, as might happen from an occlusion of one of the posterior cerebral arteries, again this would result in a contralateral homonymous hemianopsia, although some vision may be preserved in the very central portion of the affected field, a phenomenon known as *macular sparing*. If the occipital lesion were restricted to the lingual gyrus, as in the case of a lesion to the temporal radiations, the resulting deficit would be described as a superior quadrantanopsia. Similarly, a lesion affecting only the upper bank of the calcarine fissure (the cuneus) would produce an inferior quadrantanopsia. If the medial surfaces of both the right and left occipital cortices were damaged, such as might occur in strokes, the patient would be said to suffer from cortical blindness. However, especially in the acute stage of such a lesion(s), an interesting clinical finding might result. Even though totally blind, the patient may not express an awareness of being blind, insisting that he or she might see things that obviously they cannot. This is essentially an example of an anosognosia for their visual deficit, a condition known as *Anton's syndrome*. Finally, whenever a cortical lesion is involved, such as that which might affect the optic radiations, there is the possibility that *unilateral neglect* might either make the visual field defect appear to be worse than it actually is, or make the patient's functional deficit worse than might be expected given the nature of the visual loss.

The types of visual defects described above are typically the result of some type of structural damage to the central nervous system, most commonly as a result of vascular disease, tumor, or trauma. However, there are other conditions that can directly affect the eye itself. Some of the more common conditions might include trauma, glaucoma, cataracts, macular degeneration, diabetic retinopathy, or even prolonged vitamin A deficiencies.

Cross References

- ▶ Achromatopsia
- ▶ Anosognosia
- ▶ Color Agnosia
- ▶ Cortical Blindness
- ▶ Neglect Syndrome
- ▶ Retinopathy

References and Readings

Kandel, E. R., Schwartz, J. H., & Jessel, T. M. (1991). *Principles of neural science* (3rd ed.). Norwalk, CT: Appleton & Lange.

Mendoza, J. E., & Foundas, A. L. (2008). *Clinical neuroanatomy: A neurobehavioral approach.* New York: Springer.

Wilson-Pauwek, L., Akesson, E. J., Stewart, P. A., & Spacey, S. D. (2002). *Cranial nerves in health and disease.* Hamilton, ONT: B.C. Decker, Inc.

Visual Tracking

Uraina Clark
Neuropsychology, The Miriam Hospital, The Warren Alpert Medical School of Brown University
Providence, RI, USA

Definition

During visual tracking, the main objective of the observer is to keep the image of the fixated item on the fovea, the area of the retina where visual resolution is best. Normally, the observer tracks moving targets using a combination of smooth pursuit movements and small saccades. Smooth pursuit movements allow the observer to track a moving object, while saccades (derived from the French word for jerk or to pull) allow the observer to quickly redirect their gaze to the object of interest. During smooth pursuit, eye velocities are generated that approximate the velocity of the moving target object. When the eyes and target are in synch, the velocity of the target's retinal image is reduced to zero. If the eyes fall short of, or

overshoot the target, saccades are generated to reposition the image of the target object back on to the region of the fovea. During visual tracking, the vestibuloocular reflex and optokinetic movements permit the observer to compensate for head movements, which helps to stabilize the visual image on the retina.

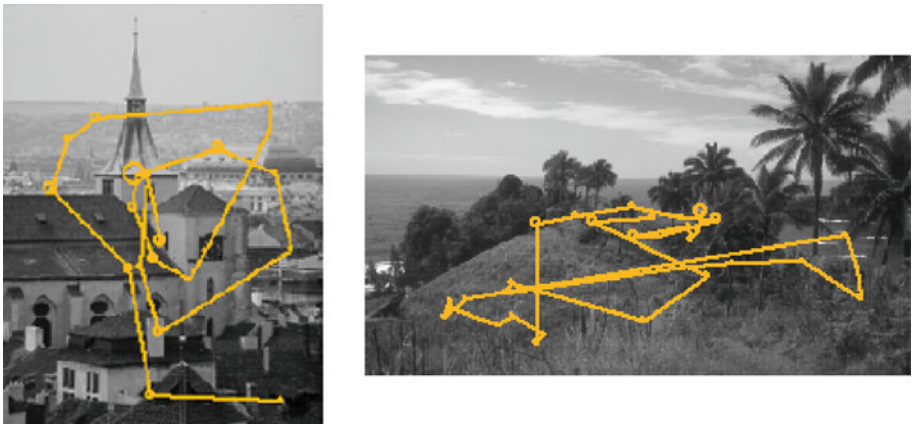
Eye tracking methodologies are commonly used to monitor and quantify eye movements in the laboratory setting (Fig. 1). Eye tracking can be used to study eye movements in response to both moving and stationary images. Because saccadic eye movements are thought to be closely linked to attention, they have received a great deal of examination in studies of attentional and perceptual processes. In the clinic setting, visual tracking and oculomotor scanning abilities are assessed using tests of visual attention and perception, such as the line bisection test and cancellation tests. Saccadic and smooth pursuit movements can also be assessed in the clinic by asking the patient to look back and forth between two widely spaced targets (e.g., the examiner's left and right index fingers) and by asking the patient to follow a moving target, as is done during a neurologic examination.

Current Knowledge

Smooth pursuit movements are significantly slower than saccades and typically occur at velocities of around 100°/s. This speed permits feedback from the vestibular and visual systems, which help to regulate the speed and duration of the movements. Generation of smooth pursuit movements involves a complex network of neural structures, including the cerebellum, vestibular nuclei, and cortical regions. Extrastriate regions sensitive to visual motion are implicated in the origination of smooth pursuit commands. Information from these regions is integrated with signals from the brain stem and frontal eye fields during smooth pursuit. Functional imaging studies have been used to explore neural regions associated with smooth pursuit; however, interpretation of some findings is clouded by the fact that visual tracking involves a combination of smooth pursuit movements and saccades. Activation related to smooth pursuit has been reported in the frontal eye fields, lateral occipito-temporal cortex, lingual gyrus, dorsomedial cuneus, and dorsal occipito-parietal cortex.

In patients with unilateral cerebral lesions, ipsidirectional smooth pursuit defects are noted. That is to say, smooth pursuit movements are impaired when tracking targets moving in the direction toward the side of the lesion. In general, cerebral pursuit defects are asymptomatic, but can often be observed clinically or in the laboratory setting. Patients with parietal and occipito-temporal lesions can show ipsidirectional pursuit defects, as can patients with lesions of the frontal eye fields, supplementary eye fields, or prefrontal cortex. Ipsidirectional defects in pursuit can also be observed following lesions of the posterior limb of the internal capsule, which affect descending cortical connections with brainstem nuclei.

Saccadic eye movements are extremely rapid; they can reach velocities of up to 700°/s. They are important in



Visual Tracking. Figure 1 The images depict examples of oculomotor scanning patterns captured over an 8 s viewing period using eye tracking (Clark et al., in press). The scan patterns shown were made by a healthy adult during a task in which the viewer was asked to examine the image and identify the type of scene viewed. Yellow circles represent fixations; yellow lines depict saccades. The size of the circles indicates length of fixation duration, with larger circles corresponding to longer duration times

visual tracking behaviors and play a primary role in visual exploration. They allow the observer to sample several bits of visual information from the environment, such as during scene viewing or reading. The neural circuitry that supports saccadic eye movements has been studied extensively and is quite intricate; it involves regions in the brain stem, subcortical structures, and several areas in the cerebral cortex. Primary cortical regions include the frontal eye fields, posterior parietal cortex, prefrontal cortex, and supplemental eye fields. The contribution of subcortical regions (i.e., the basal ganglia) to saccade generation and control is also well documented (see below). The frontal eye fields are involved in the generation of saccades made under volitional control, while posterior parietal regions are thought to be involved in the generation of automatic or reflexive saccades, which are saccades made to stimuli that occur suddenly in the environment (e.g., a person walking by or a load noise in the periphery). The prefrontal cortex supports saccades made based on spatial memory, as well as the suppression of reflexive saccades. The supplemental eye fields are noted to play a role in the temporal organization of saccades.

Bilateral cerebral lesions can cause significant disturbances in saccadic eye movements. For example, bilateral injury to the parietal lobes, such as in Balint's syndrome, can result in marked impairments in voluntary saccade generation. Most unilateral cerebral lesions do not result in saccadic dysfunction; however, unilateral frontal eye field lesions can cause delayed latencies when making voluntary saccades to the contralateral side (especially when the anterior limb of the internal capsule or white matter near the frontal horns are also affected), while vertical saccades remain intact. Patients with unilateral frontal eye field lesions spend less time scanning complex scenes on the side of the image that is contralateral to the lesion. Some consider the behaviors resulting from unilateral frontal eye field lesions to represent an exploratory or intentional type of hemineglect. Notably, some patients with unilateral frontal eye field lesions demonstrate mild signs of neglect on neuropsychological measures (e.g., line bisection and cancellation tests). Unilateral lesions of the posterior parietal cortex are associated with abnormalities in reflexive saccades, characterized by prolonged activation latencies. Typically, this abnormality is only seen in reflexive saccades made to the contralateral side, but in some patients the abnormality can be observed in both directions horizontally.

The basal ganglia are also implicated in saccade generation and control. They are noted to play a role in the temporal organization of saccades. Individuals with disorders affecting the basal ganglia (e.g., ▶ Parkinson's disease, ▶ Huntington's disease) often exhibit eye movement abnormalities. In individuals with Parkinson's disease, saccades can be small, jerky, and slow. Huntington's disease and Parkinson's disease patients also demonstrate deficits in generating memory guided saccades.

Cross References

- ▶ Balint's Syndrome
- ▶ Cancellation Tests
- ▶ Diplopia
- ▶ Dysconjugate Gaze
- ▶ Frontal Eye Fields
- ▶ Line Bisection
- ▶ Neurologic Examination
- ▶ Optokinetic Reflex
- ▶ Visual System

References and Readings

Barton, J. J. S. (2001). Brain damage and eye movements. In M. Behrmann (Ed.), *Handbook of neuropsychology* (2nd ed., Vol. 4, pp. 15–44). Amsterdam: Elsevier.

Clark, U. S., Neargarder, S., & Cronin-Golomb, A. (in press). Visual exploration of emotional facial expressions in Parkinson's disease. *Neuropsychologia*.

Hikosaka, O., Takikawa, Y., & Kawagoe, R. (2000). Role of the basal ganglia in the control of purposive saccadic eye movements. *Physiological Reviews, 80*(3), 953–978.

Visual Word Form Area

- ▶ Pure Alexia

Visual-Motor Function

Jessica Somerville Ruffolo
The Miriam Hospital
Providence, RI, USA

Synonyms

Constructional apraxia; Graphomotor function; Visuoconstruction; Visuospatial construction

Definition

Assessment of visual-motor function typically involves administering tasks that utilize the ability to organize and manually manipulate spatial information to make a design. Common visual-motor measures include assembling tasks (e.g., block or puzzle designs) and copying or drawing pictures. Visual-motor (constructional) function is a multifactorial construct requiring different cognitive abilities, such as perceptual and visuospatial skills, motor programming and coordination, and executive functioning (e.g., planning, organization, conceptualization). In addition, attention is required, and in some tasks (i.e., "draw-to-command" drawing tasks) receptive language and memory skills are also required. Disruptions in the *integration* of different aspects of brain functioning can also interfere with visual-motor performance.

Historical Background

Poppelreuter (1917) discussed "visual apraxia" symptoms, such as problems with reaching for objects, awkward object manipulation, and disturbed copying of designs, with an underlying assumption of impairment in the activation of the motor centers from visual information. The neuropsychological domain of visual-motor function was introduced by Kliest (1914/1918) as "constructional apraxia," defined as the inability to construct objects due to left-hemisphere dysfunction and problems with purposeful movements. Since then, numerous studies have shown that visual-motor deficits are not limited to left-hemisphere disturbance, but commonly arise from damage to the right hemisphere (e.g., Piercy, Hecaen, & De Ajuriaguerra, 1960; Villa, Gainotti, & De Bonis, 1986). Duensing (1953) first made the differentiation between an "ideational-apractic" (left-hemisphere) form of visual-motor disability and a "spatioagnostic" (right-hemisphere) form related to visual-spatial dysfunction. However, making distinctions based on lesion location and task performance is not always very clear-cut. Studies have shown that visuoperceptive impairment from lesions of either hemisphere can result in visual-motor disability (e.g., Dee, 1970). It has been theorized that a perceptuo-motor integrative mechanism in the left hemisphere mediates the motor aspect of visual-motor performance, irrespective of visuospatial disability (Benton & Tranel, 1993). Studies have also assessed intrahemispheric locus of lesion (anterior–posterior comparisons) and visual-motor disability. Results show a trend for more pronounced visual-motor deficit in posterior lesions (e.g., Black & Strub, 1976). However, lesions to the frontal lobes can also impact visual-motor performances due to disruptions in executive functioning (e.g., impaired organizational skills, impulsivity, conceptualization) and motor planning. Size of lesion has not been clearly found to be an important factor in predicting visual-motor performance (e.g., Benson & Barton, 1970).

Visual-motor function can be assessed with many different types of tasks, each with different requirements and cognitive demands. Some visual-motor tasks include assembling blocks or puzzles (e.g., WAIS Block Design and Object Assembly), some require copying designs (e.g., Beery-Buktenica Visual Motor Integration Test, Bender Visual Motor Gestalt Test), and others involve drawing pictures "from memory"/to command (e.g., "draw me the face of a clock"). In addition, these tasks can vary in complexity from very simple (e.g., copy a square) to more difficult (e.g., copying a complex geometric figure like the Rey-Osterrieth Complex Figure [ROCF]). Patients may perform differently based on the visual-motor task used, suggesting that all visual-motor tasks are not alike. As noted by Lezak (2004), some patients may experience difficulty in performing multiple visual-motor tasks, while others may make good block constructions but consistently produce poor drawings, and yet others may copy drawings well but are unable to do free drawing. Even with much variability between tests, most visual-motor tasks are conceptualized as measuring a similar function or construct. The variety of diverse tasks available to measure visual-motor ability varies widely in their cognitive demands (e.g., in regard to sustained attention, perception, executive function, motor skill). And often, different types of visual-motor tasks are used together clinically, as well as in research studies, to measure this heterogeneous construct. Reporting of impairment under the umbrella-term, "visual-motor" or "constructional" function can denote problems in performance on *any* type of visual-motor task used. This complicates interpretation of findings in neuropsychological assessment and across research studies.

It is not understood whether impairment in the performance of one type of visual-motor task (e.g., assembly task) should be considered separately from performance on another type of visual-motor task (e.g., graphomotor/drawing tasks), or even whether task complexity significantly impacts performance. To date, very few authors differentiate between subtypes of visual-motor tasks. Some studies have shown that performances in visual-motor ability can differ depending on whether an

assembly or drawing task is used (e.g., Dee, 1970). Therefore, it could be argued that clinicians and researchers should use different subtypes of visual-motor measures (e.g., assembly and drawing tasks, simple and complex tasks) in neuropsychological assessment batteries. Additionally, to assist with the interpretation of visual-motor test results, each underlying cognitive domain must be properly assessed with reliable and valid neuropsychological measures. Following this, interpretation of visual-motor ability depends on (1) analysis of patterns in performance between different visual-motor tests (which differ in complexity and cognitive demands) and (2) these performances are then compared to patterns of performance across other areas of brain functioning. A primary goal in visual-motor assessment is typically to determine whether impairments in visual-motor ability may be related to a particular cognitive deficit (e.g., in perception, visuospatial function, motor functioning, planning) or a combination of factors.

Current Knowledge

Although visual-motor deficit was once thought to be primarily related to impairments in praxis, the most common conception involves the execution of visuospatial skills. In fact, in the literature, some authors interchangeably refer to visual-motor tasks as visuospatial tasks, even though visual-motor ability involves more than just perceptual and spatial skills. Numerous studies have demonstrated that visual-motor function impairment may result from factors other than visuospatial deficit or from other damage than right, parietal lesions. Furthermore, cognitive processing models of drawing ability argue for a multicomponential framework, hypothesizing that various cognitive systems underlie the process of drawing, such as visual perception, visual imagery (but not in all cases), and graphic production (i.e., planning and action programming) (e.g., Guerin, Ska, & Belleville, 1999).

The concept of visual-motor function is complicated by poor agreement within studies that assess this cognitive domain. Early research on visual-motor function focused on understanding the relationship between local brain lesion site (e.g., right- vs. left-hemisphere injury), but no clear answers were concluded. Poor agreement within these studies may be due to different methodologies used by different researchers. Among many methodological concerns (e.g., the use of different patient populations, different exclusionary criteria) is the fact that very different visual-motor tasks were employed across studies. Tasks used in research on visual-motor function typically differ in administration technique, as well as in complexity level and cognitive demands. For example, studies may base their findings on subjects' ability to draw simple lines, simple geometric shapes (e.g., square, star, Greek Cross), or more demanding tasks such as WAIS Block Design or the ROCF. This variability between tasks can make it difficult to compare findings across studies.

It has also been suggested that inconsistencies in research on visual-motor function may be attributed to the fact that this construct is multifactorial and impairment may result from deficits in many different cognitive abilities. In addition, previous research has not always studied commonly-used or commercially-available neuropsychological measures, hindering the utility of the findings. Furthermore, the few studies that have explored underlying mechanisms behind visual-motor impairment, or have directly compared specific visual-motor tests for similarities and differences, have used select groups of patients, hindering the amount of variability in deficits across domains, and limiting generalizability, as well. For example, in a study assessing the concurrent and content validity of two commonly-used visual-motor tasks (ROCF and the Beery Visual-Motor Integration Test), it was found that both tasks shared considerable variance (Demsky, Carone, Burns, & Sellers, 2000). However, the patient population in this study was 6 to 11 year olds; it is not known whether these results may validly translate to adults or specific patient population groups.

The domain of visuospatial skills is typically assumed as a major factor in visual-motor function, though only a few studies have attempted to study this directly. The underlying cognitive components of visual-motor function were explored by Guerin, Belleville, and Ska (2002) in a sample of eight probable Alzheimer's patients using select simple and complex copying tasks. In this study, visual-motor performance was related to deficiencies in visual exploration and judgment of spatial relations. Contrary to expectation, however, graphical planning was not significantly related to visual-motor performance (these results were cautiously interpreted given the small sample size). A study by Angelini, Frasca, and Grossi (1992) found a significant relationship between visuospatial skills (e.g., Judgment of Line Orientation Test) and visual-motor abilities (i.e., Benton Visual Retention Test-copy); however, they suggested that visuospatial skills are insufficiently related to the cognitive demands of a visual-motor task. The authors stated that inspection of scattergrams showed that, in some cases, severe visual-motor impairment was evident

without comparable visuospatial deficit (and vice versa: visuospatial deficit without visual-motor impairment), suggesting that many factors are involved in visual-motor ability. Similarly, in an early study by De Renzi and Faglioni (1967) of right- versus left-hemisphere lesioned patients, visual-motor impairment was not consistently related to visuospatial deficit (especially for the left-hemisphere lesioned patients). The authors suggested that visual-motor impairment may often be attributed to other factors than visuospatial impairment, such as executive dysfunction or ideomotor apraxia (motor impairment).

Some investigators have examined the role of executive/frontal systems functioning in visual-motor assessment. These studies were conducted on select patient groups and/or with only one or two visual-motor function and executive measures. For example, Williams, Rich, Reed, Jackson, and LaMarche (1998) used factor analysis with a group of 50 medical patients to demonstrate the construct validity of the Visual Reproduction subtest of the Wechsler Memory Scale-Revised (WMS-R). They found that tasks believed to measure executive skills (Trail Making Test, Part B and category test) and tasks believed to measure visual-motor ability (WMS Visual Reproduction, Block Design) all loaded together on the same factor titled, "visual-perceptual-motor and conceptual skills." Using brain MRI, Price, Jefferson, Merino, Heilman, and Libon (2005) found a strong association between the amount of white matter abnormality (WMA) present in Alzheimer's and vascular dementia patients and greater impairment on measures of executive control and visual-motor/constructional abilities; WMA in this study was not related to incremental impairment in memory and language abilities. In another study (Nagahama, Okina, Suzuki, & Matsuda, 2008), dysfunction of the frontal-subcortical network (examined with SPECT) was related to impaired clock drawing in Lewy body dementia patients and was also found to be related to impaired performances on attentional and visual-motor tasks, such as copying a cube and block; however, this relationship was not found in patients with normal clock constructions. It was demonstrated by Libon et al. (2007) that frontotemporal dementia patients demonstrated impairment in both working memory/executive functioning and visual-motor/constructional functioning, but not declarative and semantic memory, and patients with Alzheimer's disease demonstrated the opposite pattern, suggesting a relationship between frontal systems (executive) functioning and visual-motor skills. Other studies have found significant relationships between executive measures and visual-motor tasks, such as the clock drawing test (e.g., Juby, Tench, & Baker, 2002; Libon, Malamut, Swenson, Sands, & Cloud, 1996; Royall, Mulroy, Chiodo, & Polk, 1999), the ROCF (Freeman et al., 2000; Ogino et al., 2008; Somerville, Tremont, & Stern, 2000), and Block Design (Bondi, Kaszniak, Bayles, & Vance, 1993). Simpler (less challenging) visual-motor tasks have not been examined in this regard. It has been argued by some (Royall, Cordes, & Polk, 1998) that simpler copying tasks depend less on executive functioning.

Other cognitive abilities, such as perceptual and motor skills, have also been found to impact visual-motor performance. For example, in a study of Alzheimer's disease patients by Huff et al. (1987), there was a strong relationship between visual discrimination (i.e., perceptual ability) and visual-motor function, and it was stated that the two domains "are clearly interdependent". Additionally, Dee (1970) examined unilaterally brain damaged patients and found that visual-motor impairment was closely associated with perceptual dysfunction (using a discrimination task). However, in some patients, like those with Huntington's disease, poor motor functioning can also impair visual-motor performance (Rouleau, Salmon, Butters, Kennedy, & McGuire, 1992). Furthermore, in a study of Parkinson's patients (Grossman et al., 1993), perceptual skills, motor skills, and executive functioning were all related to ROCF performance. To partial out the motoric requirement of visual-motor tasks, Boller et al. (1984) studied visuospatial and visual-motor tasks along with the Hooper Visual Organization Test (HVOT), which challenges visuospatial, perceptual, and organizational skills without requiring manual movement, in a sample of nondemented Parkinson's patients. Because their sample was impaired on both types of tasks, they concluded that impairments on visual-motor measures likely involve deficits in visuoperception and/or visual organization. Paul et al. (2001) also demonstrated that in a study of vascular dementia patients performance on the WAIS Block Design task accounted for 60% of the variance on the HVOT, suggesting a strong relationship between visual organizational skills and visual-motor ability.

To date, there are few current studies assessing the neuroanatomical correlates of visual-motor task performance and commonly-used visual-motor tests. In a recent study by Tranel (2008), the clock drawing task was studied with focal brain lesioned patients, and it was found that only 30 of the 133 patients were impaired in their clock drawing (16 were "borderline" impaired). Impaired clock performance was associated with right parietal cortical lesions and left inferior frontal-parietal lesions, with visuospatial errors being prominent with right-hemisphere damage and time setting errors associated

with left-hemisphere damage. Neural correlates of the Clock Drawing Test were also assessed in Alzheimer's patients with the use of PET (Lee et al., 2008), and results showed that poor clock performance was related to functional decline in the right hemisphere, particularly in the parietal cortex. In another recent study by Kim, Lee, Choi, Sohn, and Lee (2008), clock drawing performance and structural brain changes were assessed in dementia patients. It was found that cognitive and executive functioning was significantly correlated with clock performance, and increased white matter hyperintensity (particularly perventricular) and medial temporal lobe atrophy was inversely related to performance. It was suggested that executive dysfunction (due to frontal systems dysfunction/white matter abnormality) and memory impairment (due to medial temporal abnormality) are responsible for poor clock performance.

Future Directions

Interpretation of visual-motor task performance is currently complicated by the multifactorial nature of this construct and the wide variety of tasks and methods available to assess it. Therefore, future research would benefit from careful comparisons of commonly-used visual-motor tasks to examine similarities and differences, as well as to investigate the underlying neurocognitive domains of each type of task. To do so, different patient populations and normal controls could be evaluated with various visual-motor tests. and task performances could be compared (e.g., with correlational analyses, multiple regression, multivariate analysis of covariance, factor analysis) to better understand patterns of performance between tasks and between patient groups. Longitudinal case studies could also be used to study the development of visual-motor impairment in various disorders over time. There would also be benefit to researching the utility of classifying visual-motor tasks in terms of subcategories (e.g., assembly vs. drawing, simple versus complex) and how different subtypes differ in respect to cognitive demands and cerebral function. Another promising future direction includes studying the anatomical correlates utilized in performance of various visual-motor tests, as has been performed in some studies with the Clock Drawing Test.

A common trend in visual-motor function research includes assessing qualitative differences across various patient groups. Some research already suggests that *specific* visual-motor test performance may vary based on diagnosis. However, a comprehensive study of multiple visual-motor measures would shed light as to whether certain patient populations (e.g., ▶ Alzheimer's disease, ▶ Parkinson's disease) may produce different *patterns* of performance across various visual-motor tests. Studies thus far have been limited by factors mentioned above and also by the fact that using and studying multiple visual-motor tasks together is not always feasible. However, until more research exploring this confusing but important neuropsychological construct is conducted, it cannot be fully understood what underlying cognitive domains are being assessed with each visual-motor task and to what degree. Advancing our understanding of each of these tasks, and the visual-motor construct in general, would improve efficient neuropsychological assessment and interpretation of test results.

Cross References

▶ Apraxia
▶ Executive Functioning
▶ Visual-Spatial Ability
▶ Visuoperceptual

References and Readings

Angelini, R., Frasca, R., & Grossi, D. (1992). Are patients with constructional disorders different in visuo-spatial abilities? *Acta Neurologia, 14*, 595–604.

Benson, D. F., & Barton, M. I. (1970). Disturbances in constructional ability. *Cortex, 6*, 19–46.

Benton, A., & Tranel, D. (1993). Visuoperceptual, Visuospatial, and Visuoconstructive Disorders. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (2nd ed.). New York: Oxford University Press.

Black, F. W., & Strub, R. L. (1976). Constructional apraxia in patients with discrete missile wounds of the brain. *Cortex, 12*, 212–220.

Boller, F., Passafiume, D., Keefe, N. C., Rogers, K., Morrow, L., & Kim, Y. (1984). Visuospatial impairment in Parkinson's disease: Role of perceptual and motor factors. *Archives of Neurology, 41*, 485–490.

Bondi, M. W., Kaszniak, A. W., Bayles, K. A., & Vance, K. T. (1993). Contributions of frontal system dysfunction to memory and perceptual abilities in Parkinson's disease. *Neuropsychology, 7*, 89–102.

Dee, H. L. (1970). Visuoconstructive and visuoperceptive deficit in patients with unilateral cerebral lesions. *Neuropsychologia, 8*, 305–314.

Demsky, Y., Carone, D. A., Burns, W. J., & Sellers, A. (2000). Assessment of visual-motor coordination in 6- to 11-year olds. *Perceptual and Motor Skills, 91*, 311–321.

De Renzi, E., & Faglioni, P. (1967). The relationship between visuospatial impairments and constructional apraxia. *Cortex, 3*, 327–342.

Duensing, F. (1953). Raumagnostische und ideatorisch-apraktische Storung des gestalten den Handelns. *Dtsch. Z. Nervenhilk, 170*, 72–94.

Freeman, R. Q., Giovannetti, T., Lamar, M., Cloud, B. S., Stern, R. A., Kaplan, E., et al. (2000). Visuoconstruction problems in dementia: Contributions of executive systems functions. *Neuropsychology, 14*, 415–426.

Grossman, M., Carvell, S., Peltzer, L., Stern, M. B., Gollomp, S., & Hurtig, H. (1993). Visual Construction Impairments in Parkinson's Disease. *Neuropsychology, 7*, 536–547.

Guérin, F., Belleville, S., & Ska, B. (2002). Characterization of visuoconstruction disabilities in patients with probable dementia of Alzheimer's type. *Journal of Clinical and Experimental Neuropsychology, 24*, 1–17.

Guérin, F., Ska, B., & Belleville, S. (1999). Cognitive processing of drawing disabilities. *Brain and Cognition, 40*, 464–478.

Huff, F. J., Becker, J. T., Belle, S. H., Nebes, R. D., Holland, A. L., & Boller, F. (1987). Cognitive deficits and clinical diagnosis of Alzheimer's disease. *Neurology, 37*, 1119–1124.

Juby, A., Tench, S., & Baker, V. (2002). The value of clock drawing in identifying executive cognitive dysfunction in people with a normal Mini-Mental State Examination score. *Canadian Medical Association Journal, 167*, 859–864.

Kim, Y. S., Lee, K. M., Choi, B. H., Sohn, E. H., & Lee, A. Y. (2008). Relation between the clock drawing test (CDT) and structural changes of brain in dementia. *Archives of Gerontology and Geriatrics, 48*, 218–212.

Kliest, K. (1914/1918). Kriegsverletzungen des Gehirns in ihrer Bedeutung fur die Hirnlokalisation und Hirnpathologie. In O. von Schjerning (Ed.), *Handbuch der arztlichen Erfahrung im Weltkrieg 1914/18, Vol IV: Geistes- und Nervenkrankheiten.* Barth: Leipzig.

Lee, D. Y., Seo, E. H., Choo, I. H., Kim, S. G., Lee, J. S., Lee, D. S., et al. (2008). Neural correlates of the Clock Drawing Test performance in Alzheimer's disease: A FDG-PET study. *Dementia and Geriatric Cognitive Disorders, 26*, 306–313.

Lezak, M. L. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.

Libon, D. J., Malamut, B. L., Swenson, R., Sands, L. P., & Cloud, B. S. (1996). Further analyses of clock drawings among demented and nondemented older subjects. *Archives of Clinical Neuropsychology, 11*, 193–205.

Libon, D. J., Xie, S. X., Moore, P., Farmer, J., Antani, S., McCawley, G., et al. (2007). Patterns of neuropsychological impairment in frontotemporal dementia. *Neurology, 68*, 369–375.

Nagahama, Y., Okina, T., Suzuki, N., & Matsuda, M. (2008). Cerebral substrates related to impaired performance in the clock-drawing test in dementia with Lewy bodies. *Dementia and Geriatric Cognitive Disorders, 25*, 524–530.

Ogino, T., Watanabe, K., Nakano, K., Kado, Y., Morooka, T., Takeuchi, A., Oka, M., Sanada, S., & Ohtuska, Y. (2008). Predicting executive function task scores with the Rey-Osterrieth complex figure. *Brain and Development, 31*, 52–57.

Paul, R., Cohen, R., Moser, D., Ott, B., Zawacki, T., & Gordon, N. (2001). Performance on the Hooper Visual Organizational Test in patients diagnosed with subcortical vascular dementia: Relation to naming performance. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 14*, 93–97.

Piercy, M., Hécaen, & De Ajuriaguerra, J. (1960). Constructional apraxia associated with unilateral cerebral lesions: Left and right sided cases compared. *Brain, 83*, 225–242.

Poppelreuter, W. (1917). *Die psychischen Schadigungen durch Kopfschuss im Kriege 1914–1917. IV: Die optische Apraxie.* Barth: Leipzig.

Price, C. C., Jefferson, A. L., Merino, J. G., Heilman, K. M., & Libon, D. J. (2005). Subcortical vascular dementia: Integrating neuropsychological and neuroradiologic data. *Neurology, 65*, 376–382.

Rouleau, I., Salmon, D. P., Butters, N., Kennedy, C., & McGuire, K. (1992). Quantitative and qualitative analyses of clock drawings in Alzheimer's and Huntington's disease. *Brain and Cognition, 18*, 70–87.

Royall, D. R., Cordes, J. A., & Polk, M. (1998). CLOX: An executive clock drawing task. *Journal of Neurology, Neurosurgery, and Psychiatry, 64*, 588–594.

Royall, D. R., Mulroy, A. R., Chiodo, L. K., & Polk, M. J. (1999). Clock Drawing is sensitive to executive control: A comparison of six methods. *Journal of Gerontology, 54B*, 328–333.

Somerville, J. A., Tremont, G., & Stern, R. A. (2000). The Boston Qualitative Scoring System (BQSS) as a measure of executive functioning in Rey-Osterrieth Complex Figure performance. *Journal of Clinical and Experimental Neuropsychology, 22*, 613–621.

Tranel, D. (2008). Does the Clock Drawing Test have focal neuroanatomical correlates? *Neuropsychology, 22*, 553–562.

Villa, G., Gainotti, G., & De Bonis, C. (1986). Constructive disabilities in focal brain-damaged patients: Influence of hemispheric side, locus of lesion and coexistent mental deterioration. *Neuropsychologia, 24*, 497–510.

Williams, M. A., Rich, M. A., Reed, L. K., Jackson, W. T., & LaMarche, J. A. (1998). Visual reproduction subtest of the Wechsler Memory Scale-Revised: Analysis of construct validity. *Journal of Clinical Psychology, 54*, 963–971.

Visual-Perception

► Visuoperceptual

Visual-Spatial Ability

Farzin Irani
University of Pennsylvania
Philadelphia, PA, USA

Synonyms

Spatial processing; Visuospatial ability

Definition

Visuospatial ability is a component of visual perception that enables processing of the visual orientation or location of objects in space. The visuospatial or "*where*" system is functionally and neuroanatomically distinct from the visuoperceptual "what" system. Visuospatial information is processed by a "*dorsal stream*" *occipito-parietal pathway*, while a "ventral stream" occipito-temporal processes visuoperceptual information

(Ungerleider & Mishkin, 1982). Specifically, the visuoperceptual system gets input from "type M" retinal ganglion cells which project to the ventral layers of the lateral geniculate nucleus followed by superior occipital and parietal projections. Impairments in visuospatial abilities can result in deficits in visuospatial judgment, visual neglect, topographic disorientation, and Balint's syndrome (Capruso, Hamsher, & Benton, 2006).

Historical Background

Dr. Arthur Benton credits an early case report by Dr. Badal in 1888 for inciting interest in visuospatial impairments (for a more detailed review of historical figures see Benton, 1979). Dr. Badal reportedly described a patient with a disability in "the sense of space" who despite intact central visual acuity showed spatial disorientation. This was manifested by an inability to navigate her home or neighborhood, difficulty in locating objects in space, difficulty with serial reading despite intact reading of letters, numbers and familiar words, and difficulty in estimating size, distance, or location of objects despite being able to recognize the identity of objects. Subsequently other similar cases were also described with autopsies suggesting bilateral lesions of the angular and supramarginal gyrus that extended into adjacent occipital and temporal areas. Notably, in 1909 Balint added "psychic paralysis of gaze" to the clinical presentation of this subgroup of patients who presented with a constellation of symptoms that now carry his name, i.e., *Balint's syndrome*: (1) *simultanagnosia* or an inability to perceive more than one object or point in space at a time, (2) *ocular apraxia* or faulty visual scanning with an inability to project gaze voluntarily into the peripheral field and scan it despite full eye movements (Balint's "psychic paralysis of gaze"), and (3) faulty visual reaching, also known as *optic ataxia* or visuomotor apraxia (Victor & Ropper, 2001). Holmes (1918) also played an important role in identifying two subdivisions for visuospatial abilities – one involving disturbances in orientation, size, and distance estimation and the second involving disturbances in ocular fixation and the subsequent inability to find objects.

Current Knowledge

Visual neglect involves inattention to the visual space contralateral to the site of a brain lesion, typically right posterior hemisphere (usually parietal lobe), although frontal and left hemisphere lesions can also be involved. Neglect tends to be most apparent during the acute stages of brain trauma (e.g., immediately following a stroke), but inattention to visual stimuli might persist with time and subtleties are often detected by neuropsychological assessment. *Topographic disorientation* involves an impaired ability for revisualization or retrieval of previously established visuospatial knowledge which results in disorientation around familiar places (Lezak, Howieson, & Loring, 2004). Visual neglect and deficits in landmark recognition may also be contributory (Capruso et al., 2006). Topographic disorientation is usually caused by lesions in the dorsal convexity of the right parietal lobe. The constellation of symptoms for Balint's syndrome results in the impaired ability to prepare and guide visual attention to specific points in space, while coordinating arm movements towards specific objects. This syndrome is typically associated with bilateral lesions of the superior parietal lobe.

Lezak et al. (2004) describe a number of tests used to assess visuospatial abilities. Visuospatial judgment is typically assessed using the Benton Judgment of Line Orientation Test which measures the accuracy of angular orientation of lines (Benton, Varney, & Hamsher, 1978). Visual neglect is typically assessed using tests of line bisection (Schenkenberg, Bradford, & Ajax, 1980) and cancellation (Bells Test: (Gauthier, Dehaut, & Joanette, 1989)). Additional cancellation and copying tests that are part of the Behavioral Inattention Test examine tendencies to hemi-inattention and include measures of line crossing, star cancellation, letter cancellation, figure and shape copying, and representational drawing (Wilson, Cockburn, & Halligan, 1987). Clinical observation of patients while walking (e.g., bumping into furniture on one side), talking (e.g., addressing people on only one side), eating (e.g., only one side of the plate), dressing (e.g., putting on only one sock), and writing (e.g., copying half a sentence) can also be very informative when assessing for the presence of neglect. Topographic disorientation is more difficult to assess, but Dr. Lezak suggests asking the patient to locate prominent cities on a map of the country, draw a floor plan of their home or draw a map showing how to get from one familiar location to another (Lezak et al., 2004).

Future Directions

Computerized assessments of cognition are increasingly being used to collect reliable data on a range of cognitive domains that can be linked to specific brain systems. For instance, the NIH Toolbox for Assessment of

Neurological and Behavioral Function is a contract funded by the Institutes and Centers that comprise the NIH Blue print for Neuroscience Research (http://www.nihtoolbox.org/default.aspx). It will provide investigators with a brief, yet comprehensive measurement tool for assessment of cognitive function, emotional health, and sensory and motor function. While measures of visuospatial function are currently not included in the Toolbox initiative, other computerized neurocognitive batteries such as the Penn Computerized Neurocognitive Battery are available that include computerized measures of visuospatial ability such as the Computerized Judgment of Line Orientation (https://penncnp.med.upenn.edu/).

Cross References

- ▶ Balint's Syndrome
- ▶ Cancellation Tests
- ▶ Judgment of Line Orientation
- ▶ Line Bisection
- ▶ Neglect Syndrome
- ▶ Visuoperceptual

References and Readings

Benton, A. (1979). Visuoperceptive, visuospatial and visuoconstructive disorders. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology*. New York: Oxford University Press.

Benton, A., Varney, N., & Hamsher, K. (1978). Visuospatial judgment. A clinical test. *Archives of Neurology, 35*(6), 364–367.

Capruso, D. X., Hamsher, K., & Benton, A. L. (2006). Clinical evaluation of visual perception and constructional ability. In P. J. Snyder, P. Nussbaum, & D. Robins (Eds.), *Clinical neuropsychology – A pocket handbook for assessment* (2nd ed.). Washington: American Psychological Association.

Gauthier, L., Dehaut, F., & Joanette, Y. (1989). The Bells Test: A quantitative and qualitative test for visual neglect. *International Journal of Clinical Neuropsychology, 11*, 49–54.

Holmes, G. (1918). Disturbances of visual orientation. *British Journal of Ophthalmology, 2*(10), 506–516.

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

Schenkenberg, T., Bradford, D. C., & Ajax, E. T. (1980). Line bisection and unilateral visual neglect in patients with neurologic impairment. *Neurology, 30*(5), 509–517.

Ungerleider, L. G., & Mishkin, M. (1982). Two cortical visual systems. In D. J. Ingle, M. A. Goodale, & R. J. Mansfield (Eds.), *Analysis of visual behavior* (pp. 549–586). Cambridge: MIT press.

Victor, M., & Ropper, A. H. (Eds.). (2001). *Principles of neurology* (7th ed.). New York: The McGraw-Hill Companies.

Wilson, B., Cockburn, J., & Halligan, P. (1987). Development of a behavioral test of visuospatial neglect. *Archives of Physical Medicine Rehabilitation, 68*(2), 98–102.

Visual-Spatial Processing

▶ Spatial Processing

Visuoconstruction

▶ Visual-Motor Function

Visuomotor Ataxia

▶ Optic Ataxia

Visuoperception

▶ Visuoperceptual

Visuoperceptual

Farzin Irani
University of Pennsylvania
Philadelphia, PA, USA

Synonyms

Visual-perception; Visuoperception

Definition

Visuoperceptual ability is a component of visual perception that enables recognition of objects based on their form, pattern, and color. The visuoperceptual or "*what*" system is functionally and neuroanatomically distinct from the "where" system, which involves processing of visuospatial information. An occipito-temporal pathway known as the "*ventral stream*" is responsible for identifying "what" an object is, while a dorsal stream occipito-parietal pathway processes where information is located in space (Ungerleider & Mishkin, 1982). Specifically, the visuoperceptual system gets input from "type P" retinal

ganglion cells which project to the dorsal layers of the lateral geniculate nucleus followed by inferior occipital and temporal projections. Impairments in visuoperceptual abilities result in visual agnosias and deficits in form or pattern discrimination.

Historical Background

Dr. Arthur Benton recognized Hughlings Jackson as the first neurologist to identify visuoperceptual impairments in patients with brain disease (for a more detailed review of historical figures see Benton, 1979). Dr. Jackson reportedly described a patient in 1876 with a tumor in the posterior region of the right hemisphere that showed what he called "imperception" or a lack of recognition of familiar persons and places. Soon thereafter, in 1878 Munk's animal experimentation led to the description of "mindblindness" in dogs who had lost the ability to appreciate the meaning of many visual stimuli following bilateral ablation of the "upper convex" of the occipital lobe. He reportedly believed that the ablation had destroyed the "memory image" of earlier visual experiences. Subsequently, clinical reports of patients that showed "apperceptive" and "associative" types of mindblindness emerged (Lissauer, 1890). Lissauer's classic distinction suggested that the *apperceptive* type was linked with accurate perception of an object, while the *associative* type involved association of that perception with past experience. Nomenclature later changed following Freud's introduction of the term "agnosia" (literally, no knowledge) to refer to disorders of recognition. Teuber (1968) clarified the distinction between sensory and perceptual deficits by defining agnosia as "a normal percept stripped of its meanings"(Teuber, 1968). Soon thereafter, the term "visual object agnosia" became the preferred term for "mindblindness" and the terms "visual form agnosia" or "geometric form agnosia" were introduced to indicate impairment in discriminating forms of complex figures with preserved recognition of common objects. Since then, there has been a proliferation of clinical and research efforts to further understand visuoperceptual impairments as evident in visual agnosias, deficits in visual analysis, and synthesis as well as impairments in color perception.

Current Knowledge

Visual agnosias are typically associated with bilateral occipito-temporal lesions that damage the visual association cortices. There are several different subtypes of visual agnosias that have been identified including visual object agnosia, prosopagnosia, color agnosia, and simultanagnosia. Briefly, *visual object agnosias* involve a deficit in the recognition of common objects despite an intact ability to recognize the objects in other modalities (e.g., tactile) as well as intact visual acuity and language functions. *Prosopagnosia* (Greek prosopon, "face" and gnosis, "knowledge") involves an inability to recognize the identity of typically familiar faces. Apperceptive and associative subtypes have been identified for both of these subtypes of visual agnosias. *Color agnosia* has been more difficult to categorize but is associated with disproportional impairment in recognizing, naming, or using colors. Bauer (2006) describes c*entral achromatopsia* as an acquired deficit in color vision due to central nervous system disease, which results in an inability to match, discriminate, or name colors. He describes *color anomia* as a specific difficulty in naming colors, while *specific color aphasia* involves a disproportionate difficulty in the linguistic processing of colors. Finally, he identifies *color agnosia* as a residual category for patients who have difficulty appreciating the nature or name of colors.

In addition to visual agnosias, visuoperceptual deficits can also be seen in impairments in the ability to match complex patterns, discriminate unfamiliar faces, perform *visual analysis* by identifying overlapping or hidden figures, perform *visual synthesis* by mentally combining disparate parts into wholes and identifying or matching objects obscured by excessive shadowing or presentation at unusual angles.

The more commonly used visuoperceptual tests include Benton's Test of Facial Recognition which assesses the capacity to identify and discriminate photographs of unfamiliar faces and the Visual Form Discrimination Test, which assesses the capacity for complex visual form discrimination. The Hooper Visual Organization Test assesses the ability to conceptually reorganize fragmented visual stimuli. Other less frequently used tests in neuropsychological evaluations include the Visual Object and Space Perception Battery which assesses perception of letters, animals, silhouettes as well as different aspects of space perception. Gestalt completion tests such as the Closure Speed test are also infrequently used but assess perceptual closure capacity in incomplete pictures. Tests of unusual views of pictured objects assess the ability to identify familiar objects under distorting conditions or angles. The Perceptual Speed test assesses perceptual speed and accuracy when rapidly matching target figures under timed conditions. Tests of color perception

include Farnsworth's Dichotomous Test for Color Blindness (D-15), Lanthony's Desaturated 15 Hue Test (D15-d), Neitz Test of Color Vision, and the Color-to-Figure Matching Test.

Future Directions

Computerized assessments of cognition are increasingly being used to collect reliable data on a range of cognitive domains that can be linked to specific brain systems. For instance, the NIH Toolbox for Assessment of Neurological and Behavioral Function is a contract funded by the Institutes and Centers that comprise the NIH Blue print for Neuroscience Research (http://www.nihtoolbox.org/default.aspx). It will provide investigators with a brief, yet comprehensive measurement tool for assessment of cognitive function, emotional health, and sensory and motor function. While measures of visuoperceptual abilities are currently not included in the Toolbox initiative, other computerized neurocognitive batteries such as the Penn Computerized Neurocognitive Battery are available that include computerized measures of visuoperceptual learning and memory such as the Visual Object Learning Test (https://penncnp.med.upenn.edu/).

Cross References

- ▶ Agnosia
- ▶ Benton, Arthur (1909–2006)
- ▶ Color Agnosia
- ▶ Dorsal Visual Pathway
- ▶ Prosopagnosia
- ▶ Visual-Spatial Ability

References and Readings

Benton, A. (1979). Visuoperceptive, visuospatial and visuoconstructive disorders. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology.* New York: Oxford University Press.

Lissauer, H. (1890). Ein Fall von Seelenblindheit Nebst Einem Beitrage zur Theori derselben. *Archiv fur Psychiatrie und Nervenkrankheiten, 21*, 222–270.

Teuber, H. L. (1968). Alteration of percpetion and memory in man. In L. Wiskrantz (Ed.), *Analysis of behavioral change.* New York: Harper and Row.

Ungerleider, L. G., & Mishkin, M. (1982). Two cortical visual systems. In D. J. Ingle, M. A. Goodale, & R. J. Mansfield (Eds.), *Analysis of visual behavior* (pp. 549–586). Cambridge: MIT press.

Visuospatial Ability

- ▶ Visual-Spatial Ability

Visuospatial Agnosia

- ▶ Hemispatial Neglect
- ▶ Neglect
- ▶ Neglect Syndrome
- ▶ Visual Neglect

Visuospatial Construction

- ▶ Visual-Motor Function

Visuospatial Neglect

- ▶ Hemispatial Neglect
- ▶ Neglect
- ▶ Neglect Syndrome
- ▶ Visual Neglect

Visuospatial Processing

- ▶ Route Finding

Vocabulary

Susan Steffani, Leesa V. Huang
California State University
Chico, CA, USA

Synonyms

Lexicon; Semantics

Definition

Vocabulary is defined as all the words of a particular language, group, or field of knowledge. Most individuals have a larger receptive vocabulary (understanding of words) than expressive vocabulary (use of words). The developmental process begins around 12 months, with most early words containing one or two syllables. By 18 months, a child will have a lexicon of approximately 50 words, a majority of which are nouns (Owens, 2007). During the preschool years, it is hypothesized that children can determine the connection between a word and its referent after a single exposure to the word; this is known as fast-mapping. Vocabulary growth continues through the formative school years with an increase in specificity of definition and enhanced ability to discern all meanings of a word. New words are continually added throughout the adult years (Owens, 2007) with definitions of words being more abstract, exclusionary (i.e., what an entity is not), and includes personal biases and experiences.

Receptive and expressive language skills reflect crystallized abilities (i.e., acquired knowledge) and correlate highly with composite cognitive indices. In adults, vocabulary tends to be somewhat more resistant to the effects of neurological impairment than other skills. Thus, vocabulary is used to assess an individual's verbal ability, and performance is often considered to reflect a *rough* estimate of premorbid functioning. It is also important to note that measures of vocabulary are susceptible to intrasubtest scatter in which individuals may incorrectly answer easy items, but provide correct responses to more difficult ones. Word-retrieval failure or the indiscriminate loss of learned information may explain inconsistencies in performance.

Cross References

► Lexicon

References and Readings

Hiebert, E. H. & Kamil, M. L. (2005). *Teaching and learning vocabulary: Bringing research to practice.* Philadelphia, PA: Lawrence Erlbaum Associates.

Owens, R. E. (2007). *Language development: An introduction* (7th ed.). Boston, MA: Allyn & Bacon.

Vocational Assessment

Brittany J. Allen, Brick Johnstone
University of Missouri
Columbia, MO, USA

Synonyms

Occupational assessment; Vocational evaluation; Vocational testing

Definition

In the context of neuropsychology settings, Vocational Assessment refers to evaluations conducted for the purpose of informing employment issues (e.g., ability to return to work, appropriateness to continue working, recommended compensatory strategies, etc.). Such evaluations typically assess multiple domains, including cognition, behavioral functioning, sensory-motor skills, and emotional functioning (Kay & Silver, 1988). In other settings, such as career counseling, vocational assessment may refer to evaluations conducted for the purpose of providing career advice, such as suggesting a field of work in which to pursue employment based on vocational and lifestyle interests.

Current Knowledge

Vocational assessments are a common reason for neuropsychological referrals (Guilmette, Faust, Hart, & Arkes, 1990); they require a shift in emphasis from a "traditional" diagnostic interpretation to a focus on the vocational rehabilitation plan (Barisa & Barisa, 2001). Vocational evaluations integrate a comprehensive picture of the client including his/her premorbid background, neurological status, persisting impairments, prognosis, identified needs, and suggested compensatory strategies to accommodate weaknesses and accentuate strengths. Such assessments consider how deficits may interfere with daily functioning, in what ways they can be compensated for, and how impairments will specifically interfere with work-related duties. The nature and duration of vocational evaluations are tailored to the age of the client; for example, older clients who are near retirement participate in a shorter examination primarily targeting suspected problem areas (Lezak, Howieson, & Loring, 2004).

Neuropsychologists endeavor to ensure that vocational evaluations are ecologically valid, which can be a challenging task (Guilmette & Pinchot Kastner, 1998). Research on the ecological validity of psychological tests in predicting employment outcomes is often limited by examining outcome in an "employed versus unemployed" capacity, ignoring the variability that may be present in each of these categories (e.g., assistive employment, reduction in duties compared to premorbid employment, etc.), and by focusing on short-term work status (Chaytor & Schmitter-Edgecombe, 2003). Despite these limitations, there are relatively consistent findings that intellectual functioning, memory, and executive functioning skills are the most important variables in predicting employability (Kalechsteain, Newton, & van Gorp, 2003; Wen, Boone, & Kim, 2006), and that the ability of neuropsychological testing to predict return to work is significant, although moderate in magnitude (Chaytor & Schmitter-Edgecombe, 2003).

Cross References

- ▶ Disability
- ▶ Ecological Validity
- ▶ Vocational Counseling
- ▶ Vocational Rehabilitation

References and Readings

Barisa, M. T., & Barisa, M. W. (2001). Neuropsychological evaluation applied to vocational rehabilitation. *NeuroRehabilitation, 16*, 289–293.

Brown, D. G., Burnett-Stolnack, M., Hashimoto, N., Hier-Wellmer, S., Perlman, O. Z., & Seigerman, C. (1996). The relationship of neuropsychological status and productive outcomes following traumatic brain injury. *Brain Injury, 10*, 663–676.

Chaytor, N., & Schmitter-Edgecombe, M. (2003). The ecological validity of neuropsychological tests: A review of the literature on everyday cognitive skills. *Neuropsychology Review, 13*, 181–197.

Guilmette, T. J., Faust, D., Hart, K., & Arkes, H. R. (1990). A national survey of psychologists who offer neuropsychological services. *Archives of Clinical Neuropsychology, 5*, 373–392.

Guilmette, T. J., & Pinchot Kastner, M. (1998). The prediction of vocational functioning from neuropsychological data. In R. J. Sbordone & C. J. Long (Eds.), *Ecological validity of neuropsychological testing* (pp. 387–411). Boca Raton: St. Lucie Press.

Kalechstein, A. D., Newton, T. F., & van Gorp, W. G. (2003). Neurocognitive functioning is associated with employment status: A quantitative review. *Journal of Clinical and Experimental Neuropsychology, 25*, 1186–1191.

Kay, T., & Silver, S. M. (1988). The contribution of the neuropsychological evaluation to the vocational rehabilitation of the head-injured adult. *Journal of Head Trauma Rehabilitation, 3*, 65–76.

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

Wen, J. H., Boone, K., & Kim, K. (2006). Ecological validity of neuropsychological assessment and perceived employability. *Journal of Clinical and Experimental Neuropsychology, 28*, 1423–1434.

Vocational Counseling

Stephanie A. Kolakowsky-Hayner
Santa Clara Valley Medical Center, Rehabilitation Research Center
San Jose, CA, USA

Synonyms

Career counseling for individuals with disabilities; Rehabilitation counseling

Definition

Vocational counseling includes professional and personal counseling, that incorporates an additional focus on the theory and research of career development and the unique vocational needs of individuals with disabilities. The goal of vocational counseling is to assist and empower individuals with various disabilities (including neuropsychological) to achieve their career goals in the most integrated setting possible. Vocational counseling should be the central and guiding agent within the overall vocational rehabilitation process.

Current Knowledge

Vocational counseling for individuals with disabilities is, at its core, personal counseling – guided by the same fundamental theories, research, and practice methods as counseling in general. However, it is also particularly focused on theories and research of occupational choice, career development, and vocational behavior. Furthermore, it is informed by the unique vocational rehabilitation experiences and needs of individuals with disabilities.

Vocational counseling most often takes place within the context of state rehabilitation agencies or private supported rehabilitation agencies, although not exclusively. The goal of vocational counseling may differ somewhat, depending on the person, situation, or context in which

it is being delivered – but in almost all instances, its purpose is to assist and empower individuals with various disabilities to achieve their career goals in the most integrated setting possible.

Vocational counseling itself is often one of several functions within the overall vocational rehabilitation process for persons with disabilities – a process that may also include other functions such as case management, consultation, (re)training, advocacy, etc. However, no matter what other functions are required or utilized, counseling should be the central and guiding function throughout the vocational rehabilitation process.

Services offered as part of the vocational counseling process may include: diagnostic and psychological evaluation, vocational assessment, career exploration and decision making, personal/social adjustment counseling, group counseling, job attitude counseling, job-seeking skills training, postemployment/maintenance counseling, etc.

Because individuals with disabilities are people first, many of the same general career counseling theories and practices, as utilized for all people, can be applied. However, additional considerations are often warranted. A highly individualized approach should be maintained by the vocational counselor, focusing on the unique experiences and needs of each of their clients/consumers with disabilities. Issues to consider when tailoring the vocational counseling and planning process for any particular person include: the type of neuropsychological disability and its impact, the onset of disability (i.e., pre-career onset, midcareer onset, and/or progressive or episodic disabilities), functional limitations and residual capabilities, transferrable skills, (re)training/educational abilities, vocational interests and values, level of skills for independent living, access to transportation, family/personal support systems, etc.

Some of the unique concerns and needs of job seekers with disabilities, which may impact the vocational rehabilitation process include: architectural and environmental barriers, adjustment/acceptance of disability, lack of career role models with disabilities, delayed independence and employment-related experiences (if onset of disability was at a young age), being stereotyped with a "disability" label, social/interpersonal skills, attitudinal barriers, employment discrimination, etc.

For persons with neuropsychological disabilities, vocational decision-making and planning can at times be hampered by the bleak misconceptions offered by some mental health and medical professionals concerning prognosis and rehabilitation. In such cases, vocational counselors can play a particularly important role in assisting these individuals with (re)establishing self-image, attaining greater independence, empowerment, societal involvement, and purpose through the vocational counseling process.

A particularly important value, especially when vocational decision-making and planning occur, is that of shared responsibility between counselor and client/consumer. To as great a degree as is reasonably possible, the individual with a neuropsychological disability should be an equal partner in the vocational counseling relationship.

Cross References

- ▶ Rehabilitation Counseling
- ▶ Vocational Rehabilitation

References and Readings

Goodman, J., & Gillis, S. (2009). Vocational guidance requests within the international scene. *Career Development Quarterly, 57*(4), 335–341.

Kosciulek, J. (2004). Vocational rehabilitation counseling. *American Rehabilitation, 28*(1), 40.

Morris, P., & Lloyd, C. (2004). Vocational rehabilitation in psychiatry: A re-evaluation. *Australian and New Zealand Journal of Psychiatry, 38*(7), 490–494.

Ryder, B. (2003). Counseling theory as a tool for vocational counselors. *Journal of Visual Impairment and Blindness, 97*(3), 149.

Vocational Evaluation

- ▶ Vocational Assessment

Vocational Rehabilitation

Christopher Wagner
Virginia Commonwealth University
Richmond, VA, USA

Definition

Vocational rehabilitation (VR) is the process of assisting individuals with injuries, illnesses, or disabilities to identify career goals and possibilities, match individuals to best-suited career options, train them in required skills, place them in applicable and promising employment

positions, support them as they adjust to new demands, and assist them in advancing their careers.

Historical Background

VR services span public and private agencies. Public services are primarily provided through state-federal rehabilitation service delivery system, in which almost $3 billion is provided by the federal government but administered by state Departments of Rehabilitation Services (agency names may vary by jurisdiction). State-federal programs were initially formed to help returning injured soldiers after World War I, but have expanded dramatically to provide services to all citizens with physical, psychiatric, learning, or behavioral impairments. With shrinking budgets and growing demands, many states now prioritize services for those with the most severe disabilities.

Rationale or Underlying Theory

The essential rationale behind VR services is that employment problems are not an inevitable result of disease or disability, but that these conditions often create a mismatch between skills and opportunities. By guiding and training potential workers, and simultaneously working with community employers to accommodate workers with different needs, more individuals are able to participate in the workforce, helping communities and individuals alike.

Goals and Objectives

The goal of VR services is to support the efforts of individuals with disabilities in successfully obtaining and maintaining competitive employment that is suitable to their current needs, interests, and abilities. The goal is to maximize autonomy and participation in all aspects of community living.

Treatment Participants

Generally, there is a correlation between outcomes of VR services and employment statistics in general. Clients who are young, male, with higher education and greater past work experience tend to have somewhat better outcomes. Conversely, individuals who are older, women, with less than a high school education and of minority or underprivileged backgrounds tend to have less-successful outcomes. It is likely that this is more attributable to cultural factors than to direct effectiveness of VR services per se, although it is possible that rehabilitation services are more culturally attuned to well-educated, majority-status, younger men than other groups.

Treatment Procedures

On the supply side, VR services include providing consumers with vocational and personal adjustment counseling, vocational evaluation, career counseling, case management, training in employment and job seeking skills, educational services, job placement, supported employment, provision of reasonable job accommodations, and job retention and enhancement services. On the demand side, VR services include marketing to businesses, development of employer relationships, job development and staffing, development of workplace supports, reduction of barriers to accessibility, early employment assistance and consultation, and consultation related to problem resolution. Many businesses also require assistance in implementing the Americans with Disabilities Act, which requires the provision of reasonable accommodations and removal of architectural and communication barriers for persons with disabilities. Various services may be bundled in supported employment programs and additional services such as medical and psychiatric intervention may be offered to increase readiness for initial work or job stability thereafter.

Another established trend is empowerment of consumer choice in VR services. In many states, consumers now have real choices between public or private VR services through the ticket to work and other funding programs. Gradually the rigid distinctions between public and private programs are dissipating. Consumers are also required to be meaningfully involved in the design, execution, and evaluation of their rehabilitation plans in order to make them full partners in the rehabilitation process. Many people in leadership positions at all levels of rehabilitation policy and service delivery are themselves individuals with severe disabilities.

Consumer empowerment is also enhanced with the gradual decrease in the number of number and role of segregated employment settings such as sheltered workshops. These have given way to more community-based supported employment programs. Current trends also emphasize career development over the lifespan as opposed to point-in-time training and job placement. Career opportunities for VR professionals are very positive due to the aging of the workforce and the increases in disabilities associated with the aging process.

Efficacy Information

The efficacy of VR services is dependent on many factors: client interest and readiness, program elements included or neglected, counselor knowledge and skill, client–counselor working alliance, community and employer attitudes about including workers with disabilities in the workforce, and provider–employer relationships; current national and local economic conditions. Because the programs are multifaceted and community-based, little generalizable knowledge about the overall efficacy of practices is known. The recent federal partnerships with Industry program intended to package evidence-based employment practices and focused on working with businesses to support development of opportunities, reports successful placement of previously unemployed workers into competitive employment of between 53% and 73% (U.S. Department of Education, 2004).

Outcome Measurement

Employment outcome research does not have a standardized means of measuring outcomes. Client–counselor processes are sometimes measured with psychotherapy research instruments such as the Working Alliance Inventory. Outcome measurement is often more pragmatic than instrument-oriented. Outcome variables include number of hours worked, wages earned, income and benefits, job satisfaction, career advancement, and worker quality of life.

Qualifications of Treatment Providers

VR services are primarily provided by rehabilitation counselors, trained in graduate master programs accredited by the Council on Rehabilitation Education (CORE) and rehabilitation psychologists, trained in doctoral programs accredited by the American Psychological Association (APA). Rehabilitation counselors and psychologists are trained in a broad array of competencies, including medical and psychosocial aspects of disability and the provision of services to both persons with disabilities and businesses as described above. Well-prepared vocational counselors are also knowledgeable in such areas as current labor market demands, current business practices, disability-related legislation, disability benefits, payment systems for rehabilitation services, independent living, advocacy, and the current array of available services for consumers. VR services are complemented by various allied health professionals, such as occupational therapists, and physicians, such as those who specialize in occupational health or physical medicine.

Private sector VR services are typically provided by worker compensation or long-term disability insurance companies. These providers are typically very job placement-oriented. Many VR counselors provide expert witness testimony regarding the employability and earning capacity of plaintiffs in worker compensation or personal injury litigation.

Cross References

- ▶ Employment Specialist
- ▶ Occupational Therapy
- ▶ Rehabilitation Counseling
- ▶ Vocational Counseling

References and Readings

Homes, J. (2007). *Vocational rehabilitation.* Oxford, Great Britain: Blackwell.

Ross, J. (2007). *Occupational therapy and vocational rehabilitation.* Chichester, England: Wiley.

Rubin, W. E., & Rossler, R. T. (2008). *Foundations of the vocational rehabilitation process* (6th ed.). Austin, TX: Pro-Ed.

Wagner, C. C., Armstrong, A. J., Fraser, R. T., Vandergoot, D., & Thomas, D. F. (2006). Evidence-based employment practices in vocational rehabilitation. In K. Hagglund & A. W. Heinemann (Eds.), *Handbook of applied disability and rehabilitation research.* New York: Springer.

Wehman, P. H. (Ed.). *Journal of Vocational Rehabilitation.* Amsterdam, The Netherlands: IOS Press.

www.worksupport.com

Vocational Rehabilitation Counseling

▶ Rehabilitation Counseling

Vocational Specialist

▶ Employment Specialist

Vocational Testing

► Vocational Assessment

Voice Disorder

► Dysphonia

Voire Dire

Nathalie DeFabrique
Cook County Department of Corrections
Chicago, IL, USA

Synonyms

Qualifying an expert; Selection of jurors

Definition

The term "voir dire" is a phrase derived from French meaning to "speak the truth." It refers to the process by which possible jurors are questioned about their backgrounds and potential biases before being chosen to sit on a jury. When individuals are called to jury duty, they gather at the court house to form a pool of potential jurors. From there they are called in groups for specific criminal or civil trials. In these groups, they may be questioned by the judge and the attorneys for each side about their background, life experiences, and opinions. The questioning aims to determine the individual's ability to weigh the evidence justly and without bias. Each attorney attempts to select jurors who are most sympathetic to their side.

Cross References

► Cross-Examination
► Expert Witness

References and Readings

Brodsky, S. L., & Poythress, N. G. (1985). Expertise on the witness stand: A practitioner's guide. In C. P. Ewing (Ed.), *Psychology, psychiatry and the law: A clinical and forensic handbook.*
Cleary, G. P. (2007). Trial evidence foundations. James.
Kovera, M. B., Cidkinson, J. J., & Cutler, B. L. (2003). Voir dire and jury selection. In A. Goldstein (Ed.), *Handbook of psychology (Vol. 11) Forensic psychology.* New Jersey: Wiley.

Volitional Tremor

► Intention Tremors

Volume Element

► Voxel

Voluntary Motor Tract

► Cortical Motor Pathways

Von Recklinghausen's Disease

► Neurofibromatosis Type 1 (NF1)

Voxel

Robin Sekerak
Waikato District Health Board
Hamilton, New Zealand

Synonyms

Volume element

Definition

A voxel is a measurement of volume in a structure that is to be imaged. Each voxel represents a defined volume, and

can be localized by coordinates on a three-dimensional grid. Both CT and MRI scanners image a slab of tissue and describe it in a two-dimensional image on the computer screen. Each image is made up of a matrix of two-dimensional cells called pixels. Each pixel represents a volume of tissue, or *voxel*. The voxel has the same 2-D (*x*-axis, *y*-axis) size as the pixel, but the third dimension (*z*-axis) is equivalent to the slice thickness of the scan.

The color (tissue attenuation value) of each pixel is an average of the tissues represented in the voxel. If one tissue type alone is present, then it is a true representation. But if tissues of different density are present in the same voxel they are averaged, and can cause a *partial volume artefact*. Partial volume artefacts can be reduced by thinner slices, or smaller pixel size, reducing the overall size of the voxel.

Cross References

► Computed Tomography
► Magnetic Resonance Imaging
► Computed Tomography

References and Readings

Bushberg, J., Seibert, J., Leidholdt, E. M. Jr., & Boone, J. (2001). *The essential physics of medical imaging* (2nd ed. pp. 329, 371–372, 440, 457). Baltimore, MD: Lippincott Williams & Wilkins.

Bushong, S. (2004). *Radiologic science for technologists* (p. 432). Philadelphia: Elsevier Mosby.

Patel, P. (2005). *Lecture notes: Radiology* (2nd ed., pp. 8–9). Oxford: Blackwell Publishing.

Sloane, R. (2000). CT Tecchniques and Protocols. In R. M. Sloane, A. J. Fisher, P. J. Pickhardt, F. R. Gutierrez & D. M. Balfe (Eds.), *Body CT: A practical approach* (pp 1–2). New York, NY: McGraw-Hill.

Weishaupt, D., Koechli, V., Marincek, B., Froehlich, J., Nanz, D., & Pruessman, K. (2006). *How does MRI work: An introduction to the physics and function of magnetic resonance imaging* (p. 30). New York, NY: Springer.

Wolbarst, A. B. (2000). *Physics of radiology* (2nd ed., p. 95). Madison, WI: Medical Physics Publishing.

VRIN

► Variable Response Inconsistency Scale (VRIN, MMPI)

VSMS

► Vineland Social Maturity Scales

VSVT

► Victoria Symptom Validity Test

VTE

► Venous Thrombosis