O

Object Recognition

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

Synonyms

Visual object recognition

Definition

Visual object recognition is a complex process that includes the capacity to distinguish separate objects in the visual environment, the ability to recognize objects over a wide variety of viewing conditions, the means of categorizing and discriminating between objects of similar and dissimilar classes of objects, and the identification of an object's significance. Recognition, therefore involves the visual processing and perception of viewed objects, the retrieval of stored visual representations in memory, and access to semantic/conceptual information. For reviews on the current knowledge of object recognition, see (Devinsky et al. 2008; Pessoa et al. 2008) from which the following discussion was derived.

Current Knowledge

Neuropsychology of Object Recognition

An understanding of the neural mechanisms of visual object recognition came initially from early lesion studies in humans. Disorders of object recognition were first described by Lissauer in 1890, which he classified into two types, apperceptive agnosia and associative agnosia (cited in Pessoa et al. 2008). Apperceptive agnosia (or visual form agnosia) is the inability to recognize, copy, match, or discriminate simple visual stimuli despite presumed normal acuity, though sensory loss is difficult to assess in these patients. Impairment is thought to be in the early perceptual processing of visual attributes necessary for object recognition (e.g., deficits in the perception of shape and contour). Serino and colleagues (Serino et al. 2014) have proposed that more basic visual functions (e.g., line orientation processing) are impaired in severe forms of apperceptive agnosia. This is a relatively rare disorder. Patients have typically suffered diffuse posterior cerebral damage after stroke, anoxia, or carbon monoxide poisoning.

Patients with associative agnosia have difficulty recognizing visually presented objects but are able to recognize objects by other sense modalities. Unlike patients with apperceptive agnosia, associative agnosics can copy, describe

J. S. Kreutzer et al. (eds.), *Encyclopedia of Clinical Neuropsychology*,
https://doi.org/10.1007/978-3-319-57111-9

the attributes of objects, and make similarity judgments between objects. Impaired recognition in these patients is believed to be due to their inability to match what they see visually to stored visual representations of objects. Object agnosia may or may not be accompanied by prosopagnosia (see below). Bilateral infarction of the posterior cerebral arteries involving the inferior temporo-occipital junction and subjacent white matter is the most common cause of associative agnosia, although cases with unilateral lesions in this region have also been described.

Prosopagnosia is a selective deficit in the ability to recognize familiar faces or to learn and recognize new faces. These patients can identify the components that make up a face (e.g., eyes, nose, mouth) and identify that a face is a face but are unable to identify specific faces. It is a specific visual deficit as patients can recognize familiar individuals by other sense modalities (e.g., voice, perfume) and non-facial cues (e.g., clothing, body shape). Many of these patients also have difficulties distinguishing specific types of objects in a class (e.g., a turkey versus a peacock) but can distinguish between classes of objects (e.g., bird versus cat). Prosopagnosia results from lesions in the mesial occipitotemporal visual association cortex (lingual and fusiform gyrus) or the underlying white matter. In most autopsied cases, the damage to this area was bilateral. Cases of prosopagnosia and right unilateral lesions have also been described. For example, Konen and colleagues (Konen et al. 2011) present a case study of an individual with severe object agnosia and prosopagnosia following a lesion to the right lateral fusiform gyrus.

Experimental lesion studies in monkeys, like clinical studies of brain damage in humans, have demonstrated deficits in visual object recognition following lesions to the inferior temporal cortex.

Electrophysiology and Functional Neuroimaging of Object Recognition

Studies in Nonhuman Primates

Electrophysiological studies in nonhuman primates have demonstrated that visual object recognition is mediated, in large part, by the ventral visual pathway. Early models of this pathway posited that neuronal tuning specificity and invariance is processed serially in a bottom-up hierarchy of cortical areas. The initial cortical processing of visual information is characterized as low level and involves the extraction of elementary properties or features of the stimulus including luminance, color, edges, and contours. Low-level processing occurs in the early visual cortical areas (modules in V1 and V2, area V4). Intermediate-level processing combines aggregates of elementary features into progressively more complex representations such as object shape and occurs in an area of the monkey's inferior temporal cortex. Neurons within the anterior inferior temporal cortex (TE) extract complex visual information. For example, TE neurons have been found to be selectively tuned to biologically relevant stimuli (i.e., faces); they exhibit response invariance (or maintained selectivity) for object shape across changes in stimulus size, distance in depth, and illumination, and experience can affect the magnitude of neuron response. In addition to feed-forward projections, there are reciprocal feedback projections from higher-order to lower-order cortical areas and with cortical areas beyond the modality-specific visual system. The feedback projections are thought to reflect the top-down facilitation of object recognition (Pessoa et al. 2008).

It has been proposed more recently (Kravitz et al. 2013) that the ventral visual pathway is best characterized as a "recurrent and interactive occipitotemporal network." The network links early visual areas to the inferior temporal cortex via multiple visual processing routes. Along these routes, the neural representation of object features is utilized and constrained by a number of cortical and subcortical systems specialized for specific behavioral, cognitive, or affective functions, each mediating different forms of learning and memory. This framework contrasts with earlier depictions of the ventral visual pathway as largely serial bottom-up processing of an object representation and "more parsimoniously incorporates attentional, contextual, and feedback effects" on the processing of object features and qualities (Kravitz et al. 2013).

Human Studies

It is possible to record neuronal spiking activity in the human brain under special circumstances. For example, patients with medically refractory epilepsy may undergo depth electrode placement in the brain to help detect seizure foci. Single-neuron recordings in epileptic patients mostly focus on the areas of the medial temporal lobe (entorhinal cortex, parahippocampal gyrus, subiculum, and amygdala). Neurons in the human medial temporal lobe have been found to show high selectivity for specific complex visual stimuli, including faces, places, household objects, animals, scenes; and show selective response to either broad categories of objects (e.g., distinguish between faces and household objects) or to specific objects within those categories (e.g., distinguish between famous, known faces, and novel faces). Neurons in the medial temporal lobe show not only selectivity to stimulus features but also invariance to very different pictures of given faces, landmarks, and objects. In addition, recordings of neuronal activity in the human medial temporal have demonstrated that modulated responses in activity occur during visual recall and when distinguishing between novel and previously seen stimuli, suggesting an important role in memory processes. Despite evidence of high selectivity and invariance of individual neurons in the human medial temporal lobe, it is likely that a single neuron responds to more than a single visual object and that the object is represented by many neurons (Kreiman 2007; Quian Quiroga et al. 2005).

Functional neuroimaging studies in humans have revealed distinct cortical areas in the ventral object vision system by using positron emission tomography (PET) to measure increased regional cerebral blood flow and functional magnetic resonance imaging (fMRI) to measure increased blood oxygenation as indices of neural activity during visual processing and object recognition. Early visual areas (V1–V3) are activated by visual stimulation regardless of whether it is a recognizable object or an unrecognized and "scrambled" image. The lateral occipital complex (LOC) is highly sensitive to image scrambling (i.e., greater activation to intact visual stimuli than to scrambled images), shows some invariance to image position, but is not selective to specific objects. This suggests that the LOC involves general object shape extraction and is an intermediate stage between low-level visual processing and high-level processing that involves memory. Object selective areas in the ventral stream include V4, posterior inferior temporal cortex, and anterior inferior temporal cortex (Pessoa et al. 2008). These cortical areas exhibit invariance to visual cues and object transformations supporting the role these areas play in processing object information (Grill-Spector and Malach 2004). Functional imaging studies have also demonstrated specialized processing regions for specific categories of objects, including faces in the fusiform gyrus ("fusiform face area"); places and scenes in the parahippocampal gyrus ("parahippocampal place area"); human body parts, such as arms and legs in a region of the LOC ("extrastriate body area"); and letter strings and words in the left occipitotemporal and inferior occipital sulci (Pessoa et al. 2008; Reddy and Kanwisher 2006). Top-down facilitation of visual object recognition may be mediated in part by regions of the prefrontal cortex as evident by fMRI and magnetoencephalography (MEG) studies showing that the orbitofrontal cortex is active during successful object recognition (versus nonrecognition) and that it is active early in the processing of visual object information (Fenske et al. 2006). Studies suggest that general knowledge about objects (e.g., physical features and functional properties) is stored across a distributed cortical network of sensory and motor areas (Pessoa et al. 2008).

Models of Visual Object Recognition

Evidence from neuropsychology, electrophysiology, and neuroimaging suggest that there are specialized processing regions for specific categories of visual objects (i.e., faces). A number of models have been proposed for face recognition and for prosopagnosia, a specific deficit in face recognition: (1) Face recognition is a biological necessity and unique behavioral capacity that requires dedicated neuronal systems that can be selectively damaged (e.g., Kanwisher and Yovel 2006). (2) Prosopagnosia is a deficit in the general capacity

to discriminate between very similar exemplars in a class of objects due to damage to neural representations of objects that are widely distributed and overlapping (e.g., Haxby et al. 2001). (3) Prosopagnosia is the result of injury to specialized neural systems that develop with experience (expertise) and are required when distinguishing between subtle differences within a category of complex visual material (e.g., Gauthier et al. 1999). (For reviews of quantitative and computational models of visual object recognition, see Biederman and Kalocsai 1997; Riesenhuber and Poggio 2000; Serre et al. 2007.)

Cross-References

- ▶ Prosopagnosia
- ▶ Ventral Visual Pathway
- ▶ Visual Agnosia

References and Readings

Biederman, I., & Kalocsai, P. (1997). Neurocomputational bases of object and face recognition. *Philosophical Transactions of the Royal Society of London: Biological Sciences, 352*, 1203–1219.

Devinsky, O., Farah, M. J., & Barr, W. B. (2008). Visual agnosia. In G. Goldenberg & B. L. Miller (Eds.), *Neuropsychology and behavioral neurology* (pp. 417–428). Amsterdam: Elsevier.

Fenske, M. J., Aminoff, E., Gronau, N., & Bar, M. (2006). Top-down facilitation of visual object recognition: Object-based and context-based contributions. In S. Martinez-Conde, S. L. Macknik, L. M. Martinez, J.-M. Alonso, & P. U. Tse (Eds.), *Progress in brain research* (Vol. 155, pp. 3–21). Amsterdam: Elsevier.

Gauthier, I., Tarr, M. J., Anderson, A. W., Skudlarski, P., & Gore, J. C. (1999). Activation of the middle fusiform "face area" increases with expertise in recognizing novel objects. *Nature Neuroscience, 2*, 568–573.

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

Haxby, J. V., Gobbini, M. I., Furey, M. L., Ishai, A., Schouten, J. L., & Pietrini, P. (2001). Distributed and overlapping representations of faces and objects in ventral temporal cortex. *Science, 293*, 2425–2430.

Kanwisher, N., & Yovel, G. (2006). The fusiform face area: A cortical region specialized for the perception of faces. *Philosophical Transactions of the Royal Society of London: Biological Sciences, 361*, 2109–2128.

Konen, C. S., Behrmann, M., Nishimura, M., & Katner, S. (2011). The functional neuroanatomy of object agnosia: A case study. *Neuron, 71*, 49–60.

Kravitz, D. J., Saleem, K. S., Baker, C. I., Ungerleider, L. G., & Mishkin, M. (2013). The ventral visual pathway: An expanded neural framework for the processing of object quality. *Trends in Cognitive Sciences, 17*, 26–49.

Kreiman, G. (2007). Single unit approaches to human vision and memory. *Current Opinion in Neurobiology, 17*, 471–475.

Pessoa, L., Tootell, R. B. H., & Ungerleider, L. G. (2008). Visual perception of objects. In L. Squire, D. Berg, F. Bloom, S. D. Lac, A. Ghosh, & N. Spitzer (Eds.), *Fundamental neuroscience* (3rd ed., pp. 1067–1090). Amsterdam: Elsevier.

Quian Quiroga, R., Reddy, L., Kreiman, G., Koch, C., & Fried, I. (2005). Invariant visual representation by single neurons in the human brain. *Nature, 435*, 1102–1107.

Reddy, L., & Kanwisher, N. (2006). Coding of visual objects in the ventral stream. *Current Opinion in Neurobiology, 16*, 408–414.

Riesenhuber, M., & Poggio, T. (2000). Models of object recognition. *Nature Neuroscience, 3*(supplement), 1199–1204.

Serino, A., Cecere, R., Dundon, N., Bertini, C., Sanchez-Castaneda, C., & Làdavas, E. (2014). *Cortex, 52*, 12–27.

Serre, T., Kreiman, G., Kouh, M., Cadieu, C., Knoblich, U., & Poggio, T. (2007). A quantitative theory of immediate visual recognition. In P. Cisek, T. Drew, & J. F. Kalaska (Eds.), *Progress in brain research* (Vol. 165, pp. 33–56). Amsterdam: Elsevier.

Observer Reports and Ratings

Glen E. Getz
Department of Psychiatry, Allegheny General Hospital, Pittsburgh, PA, USA
Neuropsychology Specialty Care, LLC, Pittsburgh, PA, USA

Synonyms

Informant reports and ratings; Parent reports and ratings

Definition

Structured behavior rating scales to assess behavioral, emotional, cognitive, and academic functioning that are administered to parents, spouses,

teachers, or other individuals familiar with the client's functioning.

Current Knowledge

Utilization of behavior rating scales completed by parents, spouses, teachers, or other individuals who are familiar with how the client functions outside of the testing environment is a common practice utilized by clinical neuropsychologists. The use of observer rating scales has proven to be helpful in measuring behaviors at home, at school, and within the community. These methods can assess long-standing functioning or more recent skills following changes associated with an insult to the brain. Observer reports rely on information from people familiar with the client in order to assist in diagnosis or overall functioning and to obtain information on the patient's behavior in multiple settings. Behavior rating scales completed by informants are utilized to assist in establishing diagnoses, such as autism spectrum disorder, mental retardation, or attention deficit/hyperactivity disorder. They can also be utilized to assist in determining personality development or abilities associated with executive functioning. Ecologically valid rating scales can also assist in understanding cognitive ability in order to assist in determining whether full neuropsychological testing is necessary and which neuropsychological test should be administered. Some ratings scales are completed by clinicians as a way of capturing information (e.g., qualitative aspects of test behavior) that would not be reflected in test scores.

Cross-References

- ▶ AAMD Adaptive Behavior Scales
- ▶ Childhood Autism Rating Scales
- ▶ Conners Comprehensive Behavior Rating Scales™
- ▶ Self-Report Measures
- ▶ Vineland Adaptive Behavior Scales

References and Readings

Conners, C. K. (2009). *Conners* (3rd ed.). North Tonawanda: Multi-Health Systems.

Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2015). *Behavior rating inventory of executive function* (2nd ed.). Lutz: Psychological Assessment Resources.

Goldstein, S., & Naglieri, J. A. (2013). *Autism spectrum rating scales*. North Tonawanda: Multi-Health Systems.

Obsessive-Compulsive and Related Disorders

Amma A. Agyemang
Department of Physical Medicine and Rehabilitation, Virginia Commonwealth University Medical Center, Richmond, VA, USA

Definition

Obsessive-compulsive and related disorders describe a group of disorders in the *Diagnostic and Statistical Manual of Mental Disorders 5th Edition* that share the unifying symptoms of persistent, inflexible mental preoccupations and/or rituals or repetitive behaviors, consistent with behavioral disinhibition (American Psychiatric Association 2013). The group encompasses obsessive-compulsive disorder (OCD), body dysmorphic disorder, hoarding disorder, trichotillomania, and excoriation disorder. Neuropsychological research on OCD and related disorders points to dysfunction in the areas of executive functioning (e.g., organization), nonverbal memory, sustained attention, and processing speed. Some have argued that this group of disorders is caused by dysregulation in the basal ganglia, though the debate is ongoing.

See Also

- ▶ Body Dysmorphic Disorder
- ▶ Excoriation
- ▶ Hoarding
- ▶ Obsessive-Compulsive Personality Disorder

References and Readings

Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive–compulsive disorder: A meta-analysis. *Clinical Psychology Review, 33*(8), 1163–1171.

Abramowitz, J. S., & Jacoby, R. J. (2015). Obsessive-compulsive and related disorders: A critical review of the new diagnostic class. *Annual Review of Clinical Psychology, 11*, 165–186.

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders (DSM-5 ®)*. Washington, DC: American Psychiatric Association.

Snyder, H. R., Kaiser, R. H., Warren, S. L., & Heller, W. (2015). Obsessive-compulsive disorder is associated with broad impairments in executive function: A meta-analysis. *Clinical Psychological Science, 3*(2), 301–330.

Obsessive-Compulsive Personality Disorder

Cynthia Rolston
Department of PM&R, Virginia Commonwealth University-Medical College of Virginia, Richmond, VA, USA

Synonyms

Anankastic personality disorder

Definition

Obsessive-compulsive personality disorder (OCPD) is defined in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychiatric Association 2013) by a pattern of perfectionism, over-controlling behaviors of self and others, and extreme inflexibility around rules and routines, starting in early adulthood and present across contexts. Diagnostic criteria include:

- Preoccupation with details, rules, order, detracting from task intent.
- Rigidness, stubbornness, and perfectionism, interfering with task completion.
- Excessive devotion to work, limiting social and leisure activities.
- Inflexibility around morality, ethics, and values.
- Inability to discard worn, useless objects of no value.
- Reluctance to delegate tasks unless they will be done to exact specifications.
- Overly frugal, likely to hoard money.

Categorization

OCPD is classified with the Cluster C Personality Disorders in DSM-5.

Current Knowledge

Prevalence

Estimates of prevalence range from 2.1–7.9%, making OCPD one of the most commonly diagnosed personality disorders. Longitudinal twin studies suggest genetic contributions outweigh environmental factors in familial patterns (Gjerde et al. 2015).

Clinical Correlates

Persons with OCPD are likely to experience emotional dysregulation, higher levels of negative affect (particularly anger), poor emotional clarity, and rejection of emotional responses. There are high levels of overlap with obsessive-compulsive disorder (OCD), anxiety disorders, bipolar disorders, and hoarding. Among the anxiety disorders, generalized anxiety, social anxiety, and specific phobias are common. A recent factor analysis found two factors – perfectionism and preoccupation with details – explained much of the variability in a model connecting OCD and OCPD (Park et al. 2016).

Physiology and Neuropsychology

Unique neuroimaging patterns found in OCPD implicate underlying neurophysiologic mechanisms, including in the prefrontal cortex and precuneus (Coutinho et al. 2016). There is little research on the neurocognitive profiles of individuals with OCPD, although some recent work has found impaired mental flexibility and executive planning deficits (Fineberg et al. 2015a). Interestingly, certain cognitive abilities may be accentuated. Ansari and Fadardi (2016) found that persons with OCPD performed more accurately on visual performance tasks than controls.

Assessment and Treatment

Diagnosis is typically achieved through structured clinical interview (e.g., Structured Clinical Interview for DSM-5 Personality Disorders) and psychological assessment measures (e.g., Minnesota Multiphasic Personality Inventory, Millon Clinical Multiaxial Inventory). Specific to OCPD, Liggett et al. (2017) developed a 7-item scale to detect features related to DSM-5 criteria. Several modalities of psychotherapeutic intervention have been supported in the literature. Cognitive-behavioral therapy produced overall symptom reduction for patients with both OCD and OCPD, compared to those without OCPD (Gordon et al. 2016). Obsessive-compulsive behaviors were reduced after a single-session intervention of psychoeducation around stress and anxiety, paired with repeated exposure to experiences eliciting physiologic symptoms of anxiety (Timpano et al. 2016). Targeting specific interpersonal deficits may also be useful, as individuals with OCPD report high degrees of relationship distress (Cain et al. 2015). Medications are not typically prescribed unless the individual suffers from a comorbid condition, such as anxiety or depression.

See Also

- ▶ Hoarding
- ▶ Obsessive-Compulsive and Related Disorders
- ▶ Personality Disorders

References and Readings

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association Publishing.

Ansari, Z., & Fadardi, J. S. (2016). Enhanced visual performance in obsessive compulsive personality disorder. *Scandinavian Journal of Psychology, 57*(6), 542–546. https://doi.org/10.1111/sjop.12312.

Cain, N. M., Ansell, E. B., Simpson, H. B., & Pinto, A. (2015). Interpersonal functioning in obsessive–-compulsive personality disorder. *Journal of Personality Assessment, 97*(1), 90–99. https://doi.org/10.1080/00223891.2014.934376.

Coutinho, J., Goncalves, O. F., Soares, J. M., Marques, P., & Sampaio, A. (2016). Alterations of the default mode network connectivity in obsessive–compulsive personality disorder: A pilot study. *Psychiatry Research: Neuroimaging, 256*, 1–7. https://doi.org/10.1016/j.pscychresns.2016.08.007.

Fineberg, N., Day, G., Koenigswarter, N., & Reghunandanan, S. (2015a). The neuropsychology of obessessive-compulsive personality disorder: A new analysis. *CNS Spectrums, 20*(5), 490–499.

Fineberg, N. A., Kaur, S., Kolli, S., Mpavaenda, D., & Reghunandanan, S. (2015b). Obsessive-compulsive personality disorder. In K. A. Phillips & D. J. Stein (Eds.), (pp. 247–272). Arlington: American Psychiatric Publishing, Inc. https://doi.org/10.1093/med/9780198706878.001.0001.

Gjerde, L.C., Czajkowski, N., Røysamb, E., Ystrom, E., Tambs, K., Aggen, S.H., . . . Knudsen, G.P. (2015). A longitudinal, population-based twin study of avoidant and obsessive-compulsive personality disorder traits from early to middle adulthood. *Psychological Medicine, 45*(16), 3539–3548. https://doi.org/10.1017/S0033291715001440

Gordon, O. M., Salkovskis, P. M., & Bream, V. (2016). The impact of obsessive compulsive personality disorder on cognitive behaviour therapy for obsessive compulsive disorder. *Behavioural and Cognitive Psychotherapy, 44*(4), 444–459. https://doi.org/10.1017/S1352465815000582.

Liggett, J., Carmichael, K. L. C., Smith, A., & Sellbom, M. (2017). Validation of self-report impairment measures for section III obsessive–compulsive and avoidant personality disorders. *Journal of Personality Assessment, 99*(1), 1–14. https://doi.org/10.1080/00223891.2016.1185613.

Park, J. M., Storch, E. A., Pinto, A., & Lewin, A. B. (2016). Obsessive–compulsive personality traits in youth with obsessive–compulsive disorder. *Child Psychiatry and Human Development, 47*(2), 281–290. https://doi.org/10.1007/s10578-015-0565-8.

Timpano, K. R., Raines, A. M., Shaw, A. M., Keough, M. E., & Schmidt, N. B. (2016). Effects of a brief anxiety sensitivity reduction intervention on obsessive compulsive spectrum symptoms in a young adult sample. *Journal of Psychiatric Research, 83*, 8–15. https://doi.org/10.1016/j.jpsychires.2016.07.022.

Obstructive Sleep Apnea Syndrome

Dona Locke
Psychiatry and Psychology, Mayo Clinic, Scottsdale, AZ, USA

Definition

Obstructive sleep apnea (OSA) syndrome is diagnosed in both adult and pediatric populations. OSA is characterized by repetitive episodes of upper airway obstruction (complete or partial)

during sleep, usually associated with a reduction in blood oxygen saturation and brief arousals from sleep. Diagnostic criteria by the ICSD-3 involve (A and B) or C:

A. One or more of the following:

1. The patient complains of sleepiness, nonrestorative sleep, fatigue, or insomnia.

2. The patient wakes with breath holding, gasping, or choking.

3. The bed partner reports habitual snoring, breath interruptions, or both.

4. The patient has been diagnosed with an associated medical disorder (e.g., hypertension).

B. Polysomnography (PSG) or out of center sleep testing (OCST) demonstrates five or more obstructive respiratory events per hour of sleep (PSG) or per hour of monitoring (OCST).

C. PSG or OCST demonstrates 15 or more predominantly obstructive respiratory events per hour of sleep (PSG) or per hour of monitoring (OCST).

Treatments can include positional therapy (used when apnea is limited to supine position only), weight loss (as there is a higher rate of sleep apnea in obese individuals), dental devices that either shift the lower jaw forward or prevent the tongue from falling back, continuous positive airway pressure (CPAP), bilevel positive airway pressure (BiPAP), or surgical procedures designed to open the airway.

Common symptoms of untreated sleep apnea include impairments of selective attention, sustained attention, and working memory. The Epworth Sleepiness Scale (ESS) is a useful, simple, self-report screening measure for excessive daytime sleepiness, a prominent symptom of sleep apnea.

Cross-References

► Sleep Apnea Syndrome

References and Readings

Aloia, M. S., Arnedt, T., Davis, J. D., Riggs, R. L., & Byrd, D. (2004). Neuropsychological sequelae of obstructive sleep apnea-hypopnea syndrome: A critical review. *Journal of the International Neuropsychological Society, 10*, 772–785.

American Academy of Sleep Medicine. (2014). *International classification of sleep disorders* (3rd ed.). Chicago: American Academy of Sleep Medicine.

Johns, M. W. (1991). A new method for measuring daytime sleepiness: The Epworth sleepiness scale. *Sleep, 14*, 540–545.

Verstraeten, E. (2007). Neurocognitive effects of obstructive sleep apnea syndrome. *Current Neurology and Neuroscience Reports, 7*, 161–166.

Occipital Lobe

William Guido
Anatomy and Neurobiology, Virginia Commonwealth University Medical Center, Richmond, VA, USA

Synonyms

Cerebral hemisphere; Occipital cortex

Definition

The lateral surface of each cerebral hemisphere is divided into four lobes (frontal, parietal, temporal, and the occipital), named for the bones that lie above them. The occipital lobe occupies the most posterior portion of the cerebral hemispheres. It is positioned behind the parietal and temporal lobes. The boundary between the parietal and occipital lobe is somewhat arbitrary, but typically, it is defined by a line that extends from the parieto-occipital sulcus to the preoccipital notch. The occipital lobe is essential for vision. There are two major gyri (cuneus and lingual) that are separated by the calcarine sulcus.

Current Knowledge

The occipital lobe contains the primary visual cortex and a number of visual association areas. The primary visual cortex resides on either side of the calcarine sulcus. The visual world is mapped onto to the primary visual cortex. The upper bank of the calcarine sulcus is the cuneus gyrus and provides a representation of the lower visual field, while the lower bank of the calcarine sulcus is the lingual gyrus and represents the upper visual field. Lesions or damages to the primary visual cortex

or underlying optic radiations result in highly stereotypic visual field defects known as scotomas. Damage to one hemisphere will result in a contralateral visual field loss. A contralateral homonymous hemianopsia arises when both the upper (cuneus gyrus) and lower banks (lingual gyrus) of the calcarine fissure of one hemisphere are damaged. A contralateral superior (upper visual field) homonymous quadrantanopsia occurs when the lingual gyrus of one hemisphere is damaged, and an inferior (lower visual field) homonymous quadrantanopsia occurs when the cuneus gyrus is damaged. Partial unilateral damage to primary visual cortex often results in macular sparing. This occurs because the representation of central visual space, which corresponds to the foveal or macular region of the retina, occupies a large region of the primary visual cortex. Adjacent to the primary visual cortex are a number of visual association areas that contribute to analyzing and perceiving color, form, or motion. These visual association areas extend into the parietal and temporal lobes. The dorsal occipital-parietal region is part of the "where" pathway and contributes to the analysis of motion and spatial relationships between objects. The inferior occipital-temporal region is part of the "what" pathway and contributes to the analysis of form. Specific areas within occipital temporal cortex are involved in the recognition or identification of color, faces, letters, numbers, or objects. Damage to visual association areas can lead to visual agnosia or the inability to recognize familiar objects, faces, or words. For example, bilateral damage to occipital-temporal cortex can result in prosopagnosia, the inability to recognize faces. Other defects can include color agnosia, a failure to discriminate between colors or names of colors; object agnosia, a defect related to naming objects; or simultanagnosia, an inability to recognize parts of an image as a whole.

Cross-References

- ▶ Agnosia
- ▶ Homonymous Hemianopsia
- ▶ Homonymous Quadrantanopsia
- ▶ Scotoma

Occult Head Injury

Matthew R. Powell[1] and Michael McCrea[2]
[1]Division of Neurocognitive Disorders, Department of Psychiatry and Psychology, Mayo Clinic, Rochester, MN, USA
[2]Departments of Neurosurgery and Neurology, Medical College of Wisconsin, Milwaukee, WI, USA

Definition

An occult head injury refers to a brain injury not readily apparent when a patient presents for physical examination. Symptoms may be subtle, particularly early on, and the mechanism of injury may not be reported by the patient or collateral, particularly if the injury was due to abuse or neglect (Christian et al. 2009; Narang and Clarke 2014; Paul and Adamo 2014). Mental status changes, nausea and vomiting, difficulty breathing, excessive crying, other bone fractures, and retinal hemorrhage are indicators of possible brain injury and suggest screening should occur (Paul and Adamo 2014). Infants, very young children, and children presenting from lower socioeconomic households have higher risk for occult head injury (Narang and Clarke 2014). Children with challenging developmental or medical histories are also at greater risk (Paul and Adamo 2014).

Cross-References

- ▶ Concussion
- ▶ Mild Traumatic Brain Injury
- ▶ Traumatic Brain Injury (TBI)

References and Readings

Christian, C. W., Block, R., Committee on Child, A, & Neglect, & American Academy of, P. (2009). Abusive head trauma in infants and children. *Pediatrics, 123* (5), 1409–1411. https://doi.org/10.1542/peds.2009-0408.

Narang, S., & Clarke, J. (2014). Abusive head trauma: Past, present, and future. *Journal of Child Neurology, 29*(12), 1747–1756. https://doi.org/10.1177/0883073814549995.

Paul, A. R., & Adamo, M. A. (2014). Non-accidental trauma in pediatric patients: A review of epidemiology, pathophysiology, diagnosis and treatment. *Translational Pediatrics, 3*(3), 195–207. https://doi.org/10.3978/j.issn.2224-4336.2014.06.01.

Occupational Therapy

Anthony Y. Stringer
Department of Rehabilitation Medicine, Emory University, Atlanta, GA, USA

Definition

Occupational therapy (OT) consists of the therapeutic use of activities to promote health, wellness, and rehabilitation for persons who have, or are at risk of, developing diseases, disorders, impairments, disabilities, or other conditions that limit participation in and performance of everyday life activities. Occupational therapy attempts to address sensory, physical, cognitive, and psychosocial aspects of everyday life activities. In physical rehabilitation therapeutic settings, occupational therapists often focus on upper extremity function, activities of daily living, and reintegration into home, work, and community environments. Occupational therapists and neuropsychologists assess and treat common domains (e.g., cognition and psychosocial functioning), although neuropsychological testing places greater emphasis on test standardization, quantification of results, and normative interpretation. In contrast, occupational therapists make extensive use of observational ratings of holistic, adaptive functioning across clinical and community settings. Both professions utilize similar cognitive rehabilitation techniques to address patient needs. The overlap in domains assessed and treated, as well as divergence in techniques, creates potential for fruitful collaboration between these two professional fields. OT emerged as a distinct profession in the 1920s and has grown to include more than 100,000 licensed professionals today. To practice in the USA, OTs must earn a master's degree and pass the National Board for Certification in Occupational Therapy's licensing exam.

Cross-References

▶ Cognitive Rehabilitation
▶ Physical Therapy
▶ Recreational Therapy
▶ Speech-Language Therapy

References and Reading

Roy, S. H., Wolf, S. L., & Scalzitti, D. A. (2013). *The rehabilitation specialist's handbook* (4th ed.). Philadelphia: FA Davis.

OCD

Amma A. Agyemang
Department of Physical Medicine and Rehabilitation, Virginia Commonwealth University Medical Center, Richmond, VA, USA

Definition

Obsessive-compulsive disorder (OCD) entails the presence of obsessions and/or compulsions. Obsessions are defined as unremitting thoughts that are at some point intrusive and unwanted and usually associated with significant anxiety or distress. With compulsions, the individual feels compelled to engage in stereotyped behaviors or mental acts in efforts to reduce distress or anxiety. The diagnosis requires that these obsessions and/or compulsions consume considerable time in an individual's day and/or cause clinically significant distress or impairment.

Categorization

OCD is classified with the Obsessive-Compulsive and Related Disorders in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*;

American Psychiatric Association 2013). Previously classified as an anxiety disorder, OCD has been reclassified since the DSM-5 with greater appreciation for other prominent features of the disorder, namely, the repetitive behaviors and thoughts that persist despite efforts to inhibit them. However, there is ongoing debate about the reclassification, as others point to neurobiological and treatment response differences between OCD and other disorders subsumed under the OCD-related umbrella.

Current Knowledge

Development and Course

The mean age of onset of OCD is 19.5 years, with onset after age 35 being less common. Onset is usually earlier in males than females, with males being more likely to be affected during childhood while females are more vulnerable as adults. Approximately 1.2% of the US population is diagnosed with OCD in a 12-month period, with risk factors including higher negative emotionality, childhood physical and sexual abuse, and family history of OCD.

Neurobiology and Associated Conditions

There is growing recognition of OCD as a disorder of emotional and cognitive dysfunction. A key feature in OCD is executive dysfunction, characterized by cognitive rigidity in processing irrelevant actions or thoughts. The neurocircuitry in individuals with OCD has been further specified and evidences pervasive under-activation in the frontal-parietal areas of the brain during mental shifting but does not show signs of under-activation during disinhibition. Instead, during mental shifting, OCD is associated with over-activation in the caudate and thalamus. These findings suggest distinct neural pathways for the different types of executive dysfunctions in OCD. Common comorbid conditions are anxiety disorders such as panic disorder, generalized anxiety disorder, and depressive or bipolar disorder.

Assessment and Treatment

Standardized measures for evaluating OCD are the Yale-Brown Obsessive-Compulsive Scale, which includes a Symptom Checklist and Severity Scale, and self-report measures including the Obsessive-Compulsive Inventory-Revised and the Yale-Brown Obsessive-Compulsive Scale-Self Report. Cognitive behavioral therapy is effective at treating OCD, though treatment effects appear to be smaller among adults and older individuals when compared to children. The most common pharmacological interventions are selective serotonin reuptake inhibitors such as citalopram, sertraline, and paroxetine, with antipsychotics and benzodiazepines often used as augmentation therapies.

See Also

- ▶ Body Dysmorphic Disorder
- ▶ Obsessive-Compulsive and Related Disorders
- ▶ Obsessive-Compulsive Personality Disorder

References and Readings

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. Washington, DC: American Psychiatric Association.

Costa, D. L., Diniz, J. B., & Miguel, E. C. (2016). How similar are the disorders included under the umbrella of obsessive-compulsive disorder and related disorders? *JAMA Psychiatry, 73*(8), 877–877.

Gonçalves, Ó. F., Carvalho, S., Leite, J., Fernandes-Gonçalves, A., Carracedo, A., & Sampaio, A. (2016). Cognitive and emotional impairments in obsessive – Compulsive disorder: Evidence from functional brain alterations. *Porto Biomedical Journal, 1*(3), 92–105.

Morein-Zamir, S., Voon, V., Dodds, C. M., Sule, A., van Niekerk, J., Sahakian, B. J., & Robbins, T. W. (2016). Divergent subcortical activity for distinct executive functions: Stopping and shifting in obsessive compulsive disorder. *Psychological Medicine, 46*(04), 829–840.

Olatunji, B. O., Davis, M. L., Powers, M. B., & Smits, J. A. (2013). Cognitive-behavioral therapy for obsessive-compulsive disorder: A meta-analysis of treatment outcome and moderators. *Journal of Psychiatric Research, 47*(1), 33–41.

Rapp, A. M., Bergman, R. L., Piacentini, J., & McGuire, J. F. (2016). Evidence-based assessment of obsessive–compulsive disorder. *Journal of Central Nervous System Disease, 8*, 13–29.

Van Ameringen, M., Simpson, W., Patterson, B., Dell'Osso, B., Fineberg, N., Hollander, E., ... & Marazziti, D. (2014). Pharmacological treatment strategies in obsessive compulsive disorder: A cross-sectional view in nine international OCD centers. *Journal of Psychopharmacology, 28*(6), 596–602.

Ocular Dominance

Mary-Ellen Meadows
Division of Cognitive and Behavioral Neurology, Brigham and Women's Hospital, Boston, MA, USA

Definition

Ocular dominance refers to the columnar organization of the visual cortex, which was demonstrated in the formative work of Hubel and Wiesel in the 1960s. Inputs to the primary visual cortex are segregated, depending on whether the information originates from the contralateral or ipsilateral eye. The inputs alternate from each eye and form columns in bands of cortex in layer 4C of the striate cortex. These columns can be visualized as stripes.

Current Knowledge

Most of the work in this area has been done using animal models. The concept of brain plasticity as a result of experience has been widely studied using ocular dominance columns as the outcome variable. During a critical period early in life, the ocular preference of single cells can be modified by visual deprivation. The ocular dominance columns can be visualized by various methods. Autoradiography is one method whereby [^{3}H] proline is injected in one eye, and the cortex is then sectioned and viewed under the microscope. Another method involves examining the level and distribution of cytochrome oxidase (CO) in the cortex. Monocular enucleation causes a reduction in CO activity, thus revealing the ocular dominance columns. Postmortem studies in humans have utilized the Glees silver stain. The CO technique has also been used in humans who became blind in one eye prior to death. Techniques have been developed that allow the cortex to be unfolded and flattened, and the sections can be digitized. Human ocular dominance column patterns have been mapped with functional magnetic resonance imaging (fMRI), though there are technical constraints since the cortex is so folded. There are also stimulus constraints in the scanner. There has yet to be an imaging study that has shown anatomical reduction in columns of an individual with amblyopia. Ocular dominance columns can also be visualized using experimental methods that measure intrinsic optical signals. These signals are recorded from primary visual cortex with a camera focused on the surface of the brain during presentation of visual stimuli. The activated regions in the cortex can be visualized depending on whether the stimulus was presented to the right or left eye.

The width of human ocular dominance columns is approximately 1 mm, which is twice the width of a macaque ocular column. The pattern consists of parallel stripes that are oriented perpendicular to the V1/V2 border. In the human, one cannot differentiate between thin and thick stripes, and they alternate between dark and pale stripes. In humans with dense amblyopia (decreased vision in one eye as a result of early visual deprivation), the columns of the normal eye expand and the columns of the amblyopic eye are reduced. This does not occur in individuals who develop strabismus later in life. Data suggest that column formation can be modified by deprivation and that there are critical periods when this occurs but that the basic column pattern is inherent. The innate signals that guide segregation and maturation of columns are unknown.

Ocular dominance columns are not present in all species. It is unclear why this is the case. However, all animals with ocular dominance columns are predators with frontally placed eyes and have largely overlapping visual fields. Visual experience is not required for column segregation. However, neuronal activity is essential. There is a great deal of interindividual variability in the size of the surface

area of striate cortex among species, as well as the width of the columns. It is not known whether or not this has any significance for functional vision.

References and Readings

Adams, D. L., & Horton, J. C. (2009). Ocular dominance columns: Enigmas and challenges. *The Neuroscientist, 15*, 62–77.

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

Hubel, D.H. (1997). *Eye, brain and vision*. The architecture of the visual cortex. Hubel.med.harvard.edu.

Hubel, D. H., & Wiesel, T. N. (1963). Receptive fields of cells in striate cortex of very young, visually inexperienced kittens. *Journal of Neurophysiology, 26*, 994–1002.

Oculomotor Nerve

Mary-Ellen Meadows
Division of Cognitive and Behavioral Neurology, Brigham and Women's Hospital, Boston, MA, USA

This is the third (III) cranial nerve (CN). The origin of the nerve is a complicated nucleus termed the oculomotor nucleus, which is ventral to the cerebral aqueduct. The accessory oculomotor nucleus, including the Edinger-Westphal nucleus, also transverses through the red nucleus and becomes part of the oculomotor nerve. These nuclei are located in the midbrain. Each oculomotor nerve traverses between the posterior cerebral and superior cerebellar arteries and lateral to the posterior communicating artery in the interpeduncular subarachnoid cistern. From there, it runs through the arachnoid and dura mater and enters the roof of the cavernous sinus. It runs above CN IV and then divides into the superior and inferior rami as it enters the orbit through the superior orbital fissure. The superior division has branches to the superior rectus muscle and to the main superficial lamina of the levator palpebrae superioris. The inferior division, which is larger than superior division, supplies the medial and inferior recti and the inferior oblique muscles.

Function

The oculomotor nerve has both somatic motor and parasympathetic functions. It controls eye muscles including the superior rectus muscle, levator palpebrae superior, medial and inferior recti, and oblique muscles. The exceptions are control of the superior oblique muscle, which is controlled by CN IV (trochlear), and the lateral rectus, which is controlled by CN VI (abducens). The oculomotor nerve also conveys parasympathetic fibers to intraocular structures as well, that is, the sphincter pupillae and ciliary muscles. It is responsible for constriction of pupil, participating in accommodation reflexes, and raising the upper eyelid.

Illness

Oculomotor nerve palsy is a frequent symptom of an unruptured aneurysm, usually secondary to an aneurysm of the internal carotid artery near the junction with the posterior communicating artery. Symptoms include ptosis (drooping of the upper eye lid), eye turning laterally and inferiorly and referred to as a "down-and-out" position, and a dilated pupil. Partial impairments of function can cause different combinations of symptoms that may vary in severity. Patients may report diplopia that is worse when looking at objects close by. Other causes of oculomotor nerve palsy include diabetic neuropathy, head trauma, infection, herniation, tumor, or venous thrombosis. Lesions in the midbrain affecting the oculomotor nucleus can also cause an oculomotor nerve palsy. In children, ophthalmoplegic migraine can cause a reversible oculomotor nerve palsy.

Cross-References

- ▶ Optic Nerve
- ▶ Trochlear Nerve

References and Readings

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

Netter, F. (1991). *The CIBA collection of medical illustrations: Volume I: Nervous system, Part I: Anatomy and physiology*. West Caldwell: CIBA-Geigy.

Ohio State University Traumatic Brain Injury Identification Method

John D. Corrigan and Jennifer Bogner
Department of Physical Medicine and Rehabilitation, Ohio State University, Columbus, OH, USA

Synonyms

Lifetime history of TBI; TBI history

Description

The Ohio State University Traumatic Brain Injury Identification Method (OSU TBI-ID) is a standardized procedure for eliciting a person's lifetime history of TBI via a 3–5 min structured interview. Retrospective self-report has weaknesses but remains the gold standard in research and clinical use for determining lifetime history of TBI. The OSU TBI-ID has proven reliable and valid, and studies have shown results are associated with brain structure and function detected from imaging. The instrument has demonstrated its utility in many settings, including medical, mental health, substance abuse, domestic violence, corrections, and aging. Health care and social service professionals need this tool to elicit a person's history of TBI.

Historical Background

Self-report of prior medical history is highly vulnerable to underreporting. Previous studies have observed that the words used to elicit self-report of TBI (e.g., "head injury," "traumatic brain injury," "concussion," "knocked out," "loss of consciousness") are interpreted differently by respondents, which can affect recall of an injury (National Center for Injury Prevention and Control 2003; Warner et al. 2005). To avoid biases created by differences in terminology, the OSU TBI-ID first elicits recall of all injuries to the head or neck. Previous studies of the validity of injury recall methods (Warner et al. 2000, 2005) were utilized to optimize personal recall of injuries experienced. The elicitation method subsequently concentrates on the occurrence of altered consciousness as an immediate consequence of external forces affecting brain function. Being dazed and confused is distinguished from loss of consciousness (LOC). Duration of LOC is determined to discern TBI severity. The age at which the injury occurred is also elicited. In a final step, the interviewer inquires further about periods in a person's life when they may have experienced multiple blows to the head.

Psychometric Data

The validity of the OSU TBI-ID is not based on elicitation of a veridical accounting of a person's lifetime history of TBI. Instead, the OSU TBI-ID provides data for calculating summary indices reflecting the likelihood that consequences have resulted from lifetime exposure to TBI. Initial validation research has supported the psychometric qualities of these summary indices. Reliability has been demonstrated by both inter-rater and test/retest reliability (Corrigan and Bogner 2007; Bogner and Corrigan 2009; Cuthbert et al. 2016). Criterion-related validity has been shown by the relationship between indices of lifetime history and measures of cognitive performance, affective status, interpersonal functioning, and aggression (Corrigan and Bogner 2007; Bogner and Corrigan 2009; Fortier et al. 2014; Albicini and McKinlay 2015). Lifetime

TBI detected using the OSU TBI-ID also has been associated with structural and functional brain changes observed in neuroimaging (Han et al. 2015, 2016; King et al. 2016; Epstein et al. 2016). Construct validity can be inferred from multiple studies that show differences between subpopulations with and without a history of TBI (Olson-Madden et al. 2010; Ferguson et al. 2012; Corrigan et al. 2012, 2013; Dams-O'Connor et al. 2013; Ray et al. 2014; Bogner et al. 2015; Brenner et al. 2013, 2015; Whiteneck et al. 2016a, b; Budd et al. 2017).

Clinical Uses

Research indicates that a person's lifetime history of TBI is useful for judging current cognitive and emotional states, particularly behavior associated with the executive functioning of the frontal parts of the brain (e.g., planning, impulsivity, addiction, interpersonal abilities). Due to how TBI damages the brain, more exposure (i.e., a worse history of lifetime TBI) increases the likelihood that an individual will struggle with current life stressors, whatever they are. A person who has compromised functioning in the frontal areas of the brain:

- adapts less well in new or stressful situations;
- has greater problems following through on recommendations from professionals;
- has more difficulty making lifestyle changes, particularly when rewards are in the future.

See Also

- ▶ Concussion
- ▶ Mild Traumatic Brain Injury
- ▶ Traumatic Brain Injury (TBI)

References and Readings

Albicini, M., & McKinlay, A. J. (2015). Internalizing disorders in adults with a history of childhood traumatic brain injury. *Clinical and Experimental Neurology, 37*(7), 776–784.

Bogner, J., & Corrigan, J. D. (2009). Reliability and predictive validity of the Ohio State University TBI identification method with prisoners. *Journal of Head Trauma Rehabilitation, 24*(4), 279–291.

Bogner, J., French, L. M., Lange, R. T., & Corrigan, J. D. (2015). Pilot study of traumatic brain injury and alcohol misuse among service members. *Brain Injury, 7*, 1–10.

Brenner, L. A., Homaifar, B. Y., Olson-Madden, J. H., Nagamoto, H. T., Huggins, J., & Schneider, A. L. (2013). Prevalence and screening of traumatic brain injury among veterans seeking mental health services. *Journal of Head Trauma Rehabilitation, 28*(1), 21–30.

Brenner, L. A., Bahraini, N., Homaifar, B. Y., Monteith, L. L., Nagamoto, H., & Dorsey-Holliman, B. (2015). Executive functioning and suicidal behavior among veterans with and without a history of traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 96*, 1411–1418.

Budd, M. A., Dixon, T. M., Barnett, S. D., Njoh, E., Goetz, L. L., & Ottomanelli, L. (2017). Examination of traumatic brain injury exposure among veterans with spinal cord injury. *Rehabilitation Psychology, 62*(3), 345–352.

Corrigan, J. D., & Bogner, J. (2007). Initial reliability and validity of the Ohio State University TBI Identification Method. *Journal of Head Trauma Rehabilitation, 22*(6), 318–329.

Corrigan, J. D., Bogner, J., & Holloman, C. (2012). Lifetime history of traumatic brain injury among persons with substance use disorders. *Brain Injury, 26*(2), 139–150.

Corrigan, J. D., Bogner, J., Mellick, D., Bushnik, T., Dams-O'Connor, K., & Hammond, F. M. (2013). Prior history of traumatic brain injury among persons in the Traumatic Brain Injury Model Systems National Database. *Archives of Physical Medicine and Rehabilitation, 94*(10), 1940–1950.

Cuthbert, J. P., Whiteneck, G. G., Corrigan, J. D., & Bogner, J. (2016). The reliability of a computer-assisted telephone interview version of the Ohio State University Traumatic Brain Injury Identification Method. *Journal of Head Trauma Rehabilitation, 31*(1), E36–E42.

Dams-O'Connor, K., Spielman, L., Singh, A., Gordon, W. A., Lingsma, H. F., Maas, A. I., & TRACK-TBI Investigators. (2013). The impact of previous traumatic brain injury on health and functioning: A TRACK–TBI study. *Journal of Neurotrauma, 30*(24), 2014–2020.

Epstein, D. J., Legarreta, M., Bueler, E., King, J., McGlade, E., & Yurgelun-Todd, D. (2016). Orbitofrontal cortical thinning and aggression in mild traumatic brain injury patients. *Brain and Behavior, 6*(12), e00581.

Ferguson, P. L., Pickelsimer, E. E., Corrigan, J. D., Bogner, J. A., & Wald, M. (2012). Prevalence of traumatic brain injury among prisoners in South Carolina. *Journal of Head Trauma Rehabilitation, 27*(3), E11–E20.

Fortier, C. B., Amick, M. M., & Grande, L. (2014). The Boston Assessment of Traumatic Brain Injury-Lifetime (BAT-L) semistructured interview: Evidence of research utility and validity. *Journal of Head Trauma Rehabilitation, 29*(1), 89–98.

Han, K., Chapman, S. B., & Krawczyk, D. C. (2015). Altered amygdala connectivity in individuals with chronic traumatic brain injury and comorbid depressive symptoms. *Frontiers in Neurology, 6*, 231.

Han, K., Chapman, S. B., & Krawczyk, D. C. (2016). Disrupted intrinsic connectivity among default, dorsal attention, and frontoparietal control networks in individuals with chronic traumatic brain injury. *Journal of the International Neuropsychological Society, 22*(2), 263–279.

King, J. B., Lopez-Larson, M. P., & Yurgelun-Todd, D. A. (2016). Mean cortical curvature reflects cytoarchitecture restructuring in mild traumatic brain injury. *NeuroImage: Clinical, 11*, 81–89.

National Center for Injury Prevention and Control (2003). Report to Congress on Mild Traumatic Brain Injury in the United States: Steps to Prevent a Serious Public Health Problem. Atlanta, GA, Centers for Disease Control and Prevention.

Olson-Madden, J. H., Brenner, L., Harwood, J. E., Emrick, C. D., Corrigan, J. D., & Thompson, C. (2010). Traumatic brain injury and psychiatric diagnoses in veterans seeking outpatient substance abuse treatment. *Journal of Head Trauma Rehabilitation, 25*(6), 470–479.

Ray, B., Sapp, D., & Kincaid, A. J. (2014). Traumatic brain injury among Indiana state prisoners. *Forensic Science, 59*(5), 1248–1253.

Warner, M., Barnes, P. M., Fingerhut, L. A. (2000). Injury and poisoning episodes and conditions: National Health Interview Survey, 1997. *Vital Health Stat*, *10*(202), 1–3.

Warner M, P. M. Schenker, N., Heinen, M. A., and Fingerhut L. A. (2005). The effects of recall on reporting injury and poisoning episodes in the National Health Interview Survey. *Injury Prevention*, *11*, 282–287.

Whiteneck, G. G., Cuthbert, J. P., Corrigan, J. D., & Bogner, J. A. (2016a). Risk of negative outcomes after traumatic brain injury: A statewide population-based survey. *Journal of Head Trauma Rehabilitation, 31*(1), E43–E54.

Whiteneck, G. G., Cuthbert, J. P., Corrigan, J. D., & Bogner, J. A. (2016b). Prevalence of self-reported lifetime history of traumatic brain injury and associated disability: A statewide population-based survey. *Journal of Head Trauma Rehabilitation, 31*(1), E55–E62.

Olanzapine

John C. Courtney[1] and Efrain Antonio Gonzalez[2,3]
[1]Socorro Mental Health, Presbyterian Medical Services, Socorro, NM, USA
[2]College of Psychology, Nova Southeastern University, Fort Lauderdale, FL, USA
[3]Utah State University, Logan, UT, USA

Generic Name

Olanzapine

Brand Name

Zyprexa, Zyprexa Relprevv, Zyprexa Zydis, Symbyax (in combination with fluoxetine)

Class

Antipsychotics, second generation, antimanic agents, and mood stabilizer

Proposed Mechanism(s) of Action

Dopamine 2 antagonist, 5HT2a antagonist (increasing presynaptic catecholamine release), theoretically antagonizes 5HT2C thus improving cognition and improving depression

Indication

Schizophrenia (acute and maintenance), acute agitation, and bipolar mood disorder (acute and maintenance)

Off-Label Use

Agitation associated with a myriad of psychiatric conditions and dementia, borderline personality disorder, disruptive behavioral disorders associated with childhood, chemotherapy-associated nausea, and vomiting

Side Effects

Serious

Hyperglycemia, rare neuroleptic malignant syndrome and seizures, and prolonged QT syndrome

Common

Significant weight gain, dizziness, constipation, tachycardia, orthostasis, rare tardive dyskinesia, and rash with overexposure to sunlight

References and Readings

Physicians' desk reference (71st ed.). (2017). Montvale: Thomson PDR.
Stahl, S. M. (2007). *Essential psychopharmacology: The prescriber's guide* (2nd ed.). New York: Cambridge University Press.

Additional Information

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

Older Americans' Resources and Services Multidimensional Functional Assessment Questionnaire

Tamara Bushnik
Inter-Hospital Research and Knowledge Translation, Rusk Rehabilitation, New York, NY, USA

Synonyms

OARS multidimensional functional assessment questionnaire

Description

It was developed in 1978 at Duke University to provide an assessment of individual functioning in elderly individuals (Multidimensional Functional Assessment 1978). It has been used for the clinical assessment, population surveys, program evaluation, personnel training, and service planning. It is a part of a larger instrument which contains three components: functional status (Older Americans' Resources and Services Multidimensional Functional Assessment Questionnaire [OARS MFAQ]), service use, and the link between the two. Information is collected about the individual's functional ability in five domains: social resources (quantity and quality of relationships with friends and family), economic resources (adequacy of income and other resources), mental health (extent of mental well-being and presence of organicity), physical health (presence of physical disorders and participation in physical activities), and activities of daily living (activities of daily living [ADLs]; capacity to perform instrumental and physical tasks). The interviewer makes an informed judgment about the functional status of the individual on a 6-point scale from 1 (excellent functioning) to 6 (totally impaired) (Kane and Kane 1981).

Since its development, the OARS MFAQ has undergone revision. A computerized summary rating protocol has been developed to improve reliability. Within each domain, multi-item scales have been developed to assess specific aspects of functioning. For social resources, three scales assess the frequency of social interaction, availability of emotional support, and perceived quality of social support. For economic resources, a perception of economic well-being scale was developed. Within mental health, there are four scales: life satisfaction, quality of sleep, psychiatric symptoms, and suspiciousness or paranoid ideation. In physical health, a measure of self-perceived health was developed. ADL scales assess instrumental self-care capacity and physical ADLS (George and Fillenbaum 1985).

Psychometric Data

Reliability: Test-retest reliability is 91% (Multidimensional Functional Assessment Questionnaire, 1978). Inter-rater reliability ranges from 0.67 to 0.87 across the five domains (Fillenbaum and Smyer 1981).

Validity: Scores on the five domains were compared with professional assessments appropriate to each domain; Kendall's tau values ranged between 0.60 and 0.89 (Fillenbaum and Smyer 1981).

Clinical Uses

The OARS MFAQ can be used clinically with older individuals; however, there are limitations. The interview is relatively long and interviewers need to be trained to administer and score (Arnold 1991). Disparities between the performance and capacity have been noted, potentially due to motivation, depression, and cognitive impairment (Feinsten et al. 1986).

Cross-References

► Activities of Daily Living (ADL)

References and Readings

Arnold, S. (1991). Measure of quality of life in the frail elderly. In J. Birren, J. Lubben, J. Rowe, & D. Deutchman (Eds.), *The concept and measurement of quality of life in the frail elderly* (pp. 50–74). New York: Academic.

Doble, S. E., & Fisher, A. G. (1998). The dimensionality and validity of the Older Americans Resources and Services (OARS) Activities of Daily Living (ADL) scale. *Journal of Outcome Measurement, 2*, 4–24.

Feinsten, A., Josephy, B., & Wells, C. (1986). Scientific and clinical problems in indexes of functional disability. *Annals of Internal Medicine, 150*, 413–420.

Fillenbaum, G. (2005). *Multidimensional functional assessment of older adults: The Duke Older Americans Resources and Services Procedures*. Hillsdale: Lawrence Erlbaum Associates. (Electronic version only; information at https://sites.duke.edu/centerforaging/services/older-americans-resources-and-services/).

Fillenbaum, G. G., & Smyer, M. A. (1981). The development, validity, and reliability of the OARS multidimensional functional assessment questionnaire. *Journal of Gerontology, 36*, 428–434.

George, L., & Fillenbaum, G. (1985). OARS methodology: A decade of experience in geriatric assessment. *Journal of the American Geriatrics Society, 33*, 607–615.

Kane, R., & Kane, R. (1981). *Assessing the elderly*. Lexington: Lexington Books.

Multidimensional functional assessment: The OARS methodology, a manual (2nd ed.). (1978). Durham: Center for the Study of Aging and Human Development, Duke University.

Van Hook, M. P., Berkman, B., & Dunkle, R. (1996). Assessment tools for general health care setting: PRIME-MD, OARS, and SF-36. *Health and Social Work, 21*, 230–234.

Olfaction

Nathan D. Zasler
Concussion Care Centre of Virginia, Ltd., Richmond, VA, USA

Synonyms

Smell

Definition

Olfaction refers to the sense of smell. Olfaction is mediated by specialized sensory cells in the nasal cavity in humans. Olfaction is a form of chemoreception.

Historical Background

The first published report of post-traumatic anosmia appeared in the London Hospital Report in 1864. Early studies of post-traumatic olfactory loss suggest a 4–7% incidence of anosmia following head injury. More recent studies have shown a higher incidence of anosmia, reaching 60% in severe head injury. This might be due to (1) increased awareness of olfactory dysfunction in head trauma, (2) the use of standardized tests for the measurement of olfactory function, or (3) a selection bias introduced when studying patients who present to chemosensory centers for evaluation and treatment of their dysfunction. Several studies have shown that the likelihood of anosmia is associated with the severity of the injury. Parosmia or a distorted sense of olfaction, often resulting in phantom, nonexistent, and mostly unpleasant smells, has also been reported in 25–33% patients with head trauma.

Current Knowledge

CN I mediates sensation. It is responsible for determining flavors. CN V mediates somatosensory

sensations, including burning, cooling, irritation, and tickling. The olfactory neuroepithelium is a pseudostratified columnar epithelium. The olfactory epithelium is situated in the superior aspect of each nostril, including cribriform plate, superior turbinate, superior septum, and sections of the middle turbinate. It harbors sensory receptors of the main olfactory system and some CN V free nerve endings. The olfactory epithelium loses its general homogeneity postnatally, and as early as the first few weeks of life, metaplastic islands of respiratory-like epithelium appear. The metaplasia increases in extent throughout life. It is presumed that this process is the result of insults from the environment, such as viruses, bacteria, and toxins. There are six distinct cell types in the olfactory neuroepithelium. The odorant receptors are located on the cilia of the receptor cells. Each receptor cell expresses a single odorant receptor gene. There are approximately 1000 classes of receptors at present. The olfactory receptors are linked to the stimulatory guanine nucleotide-binding protein Golf. When stimulated, it can activate adenylate cyclase to produce the second messenger cAMP, and subsequent events lead to depolarization of the cell membrane and signal propagation. Although each receptor cell only expresses one type of receptor, each cell is electrophysiologically responsive to a wide but circumscribed range of stimuli. This implies that a single receptor accepts a range of molecular entities.

Olfactory sensory neurons project axons to the brain within the olfactory nerve (cranial nerve I). These axons pass to the olfactory bulb through the cribriform plate, which in turn projects olfactory information to the olfactory cortex and other areas. The axons from the olfactory receptors converge in the olfactory bulb within small (~50 μm in diameter) structures called glomeruli. Mitral cells in the olfactory bulb form synapses with the axons within glomeruli and send the information about the odor to multiple other parts of the olfactory system in the brain where multiple signals may be processed to form a synthesized olfactory perception. The mitral cells leave the olfactory bulb in the lateral olfactory tract, which synapses on five major regions of the olfactory cortex: the anterior olfactory nucleus, the olfactory tubercle, the orbitofrontal cortex, the pyriform cortex, and the entorhinal cortex. The anterior olfactory nucleus projects, via the anterior commissure, to the contralateral olfactory bulb, inhibiting it. The olfactory tubercle projects to the medial dorsal nucleus of the thalamus, which then projects to the orbitofrontal cortex. The orbitofrontal cortex mediates conscious perception of the odor. The three-layered pyriform cortex projects to a number of thalamic and hypothalamic nuclei, the hippocampus and amygdala, and the orbitofrontal cortex, but its function is largely unknown. The entorhinal cortex projects to the amygdala and is involved in emotional and autonomic responses to odor. It also projects to the hippocampus and is involved in motivation and memory. Odor information is easily stored in long-term memory and has strong connections to emotional memory. This is possibly due to the olfactory system's close anatomical ties to the limbic system and hippocampus, areas of the brain that have long been known to be involved in emotion and place memory, respectively.

Since any one receptor is responsive to various odorants, and there is a great deal of convergence at the level of the olfactory bulb. Half the neurons in the orbitofrontal cortex are responsive only to one odor and the rest to only a few. Each individual odor gives a particular specific spatial map of excitation in the olfactory bulb. Many animals, including most mammals, have two distinct and segregated olfactory systems: a main olfactory system, which detects volatile stimuli, and an accessory olfactory system, which detects fluid-phase stimuli. Behavioral evidence suggests that these fluid-phase stimuli often function as pheromones, although pheromones can also be detected by the main olfactory system. In women, the sense of olfaction is strongest around the time of ovulation, significantly stronger than during other phases of the menstrual cycle and also stronger than the sense in males.

Olfaction, taste, and trigeminal receptors together contribute to flavor. The human tongue can only distinguish among seven to eight distinct types of taste, while the nose can distinguish among hundreds of substances, even in minute quantities. Olfaction amplifies the sense of taste. Olfactory disorders can be classified as follows:

1. Anosmia: inability to detect qualitative olfactory sensations (i.e., absence of smell function)
2. Partial anosmia: ability to perceive some, but not all, odorants
3. Hyposmia or microsmia: decreased sensitivity to odorants
4. Hyperosmia: abnormally acute smell function
5. Dysosmia (cacosmia or parosmia): distorted or perverted smell perception or odorant stimulation
6. Phantosmia: dysosmic sensation perceived in the absence of an odor stimulus (aka olfactory hallucination)
7. Olfactory agnosia: inability to recognize an odor sensation

It is also useful to classify olfactory dysfunction into three general classes:

1. Conductive or transport impairments from obstruction of nasal passages (e.g., chronic nasal inflammation, polyposis, etc.)
2. Sensorineural impairments from damage to neuroepithelium (e.g., viral infection, airborne toxins, etc.)
3. Central olfactory neural impairment from central nervous system damage (e.g., tumors, masses impacting on olfactory tract, neurodegenerative disorders, etc.)

These categories are not mutually exclusive. For example, viruses can cause damage to the olfactory neuroepithelium, and they may also be transported into the central nervous system via the olfactory nerve causing damage to the central elements of the olfactory system.

Olfaction is intimately involved with taste, hedonic pleasures of food, eating, aroma appreciation, and sexual intimacy, among other phenomena. It is critical in certain situations where smell of noxious odors would be relevant to avoiding harm. Certain vocations rely heavily on smell such as a cook, perfumist, or chemist. Homemakers with impaired smell may mistakenly cook with spoiled food or not smell dirty diapers. Smell impairment is more frequently noted in smokers, older individuals, traumatic brain injury, toxic exposures, autoimmune disorders, and rhinosinusitis, among other conditions.

Post-traumatic olfactory dysfunction may be caused by several mechanisms: (1) sinonasal tract alteration, (2) shearing injury of olfactory nerve filament, or (3) brain contusion and hemorrhage within the olfactory-related brain regions. Although olfactory dysfunction caused by sinonasal tract alteration is not common, it is important to recognize it. Following head trauma, patients may have mucosal hematoma, edema, or avulsion within the olfactory cleft with direct injury to the olfactory neuroepithelium. Also scarring due to nasal adhesions, nasal skeletal fracture or post-traumatic rhinosinusitis may subsequently alter the airflow that prevents odorants from reaching the olfactory epithelium. This conductive olfactory dysfunction is potentially treatable. Often, the reduction of mucosal edema, the repair of nasal fractures with relief of airway obstruction, or the treatment of sinusitis can improve olfaction.

The axons of olfactory receptor cells are delicate and pass through small foramina of the cribriform plate at the base of the skull and synapse directly in the olfactory bulb. Any tearing or shearing of the axons during the trauma can result in olfactory dysfunction. It may occur with fractures of the naso-orbito-ethmoid region, involving the cribriform plate, or with rapid translational shifts in the brain secondary to coup or contrecoup forces generated by blunt head trauma. Cranial trauma often results in traumatic brain injury in the form of cortical contusion or intraparenchymal hemorrhage. Contusion of the olfactory bulbs or cortical lesions at the olfactory-related brain regions (e.g., amygdale, temporal lobe region, frontal lobe region) can lead to post-traumatic anosmia. Based on the functional anatomy noted, anosmia may be a clue to underlying frontotemporal insult and should always be assessed for in the context of both medical and neuropsychological evaluations.

In evaluation of the patient with cranial as well as brain injury and olfactory dysfunction, the patient should be asked about pre-traumatic olfactory function, present olfactory function, and the severity and nature of the loss. The

time course of the olfactory disturbance is important. Most post-traumatic olfactory losses are due to shear injury to the olfactory nerve fibers. Such injuries typically result in immediate smell loss. The gradual, progressive losses are more commonly associated with an inflammatory process. The patient should also be asked about clear rhinorrhea or "salty-tasting" post-nasal drip to rule out cerebrospinal fluid (CSF) leak. On physical exam, nasal endoscopy is recommended to inspect the olfactory cleft for mucosal edema, hematoma, ecchymosis, laceration, or CSF leak. Radiographic imaging is valuable in the diagnosis of post-traumatic smell loss. High-resolution CT with axial and coronal cut can show cribriform plate fractures. Intracranial soft tissue injury is best evaluated by MRI.

Many post-traumatic olfactory disturbances are irreversible with most improvements occurring within the first 6–12 months post injury based on the available literature. Some patients may actually regress relative to chemosensory functioning with time, and very few with neurogenic olfactory dysfunction regain normal smell. Appropriate medical evaluation is critical to assess for treatable causes, and when no treatable cause can be identified, it is important to provide appropriate chemosensory compensatory strategies and safety education.

Future Directions

Further research on olfactory system neuroplasticity and neurorecovery may potentially lead to better treatment methodologies. Additionally, research examining screening techniques may allow for more sensitive and specific markers of post-traumatic olfactory dysfunction which may also correlate with and/or be a marker for frontotemporal neuropsychological impairments.

Cross-References

► Cranial Nerves
► Taste

References and Readings

http://www.anosmiafoundation.com/treatments.shtml (n.d.). Accessed 5/23/16.
Coelho, D. H., & Costanzo, R. M. (2016). Posttraumatic olfactory dysfunction. *Auris, Nasus, Larynx, 43*(2), 137–143.
Doty, R. (2015). *Handbook of olfaction and gustation* (3rd ed.). Hoboken: Wiley-Blackwell.
Gaines, A. D. (2010). Anosmia and hyposmia. *Allergy and Asthma Proceedings, 31*(3), 185–189.
Hawkes, C. H., & Doty, R. L. (2009). *The neurology of olfaction*. Cambridge, UK: Cambridge University Press.
Menini, A. (Ed.). (2010). *The neurobiology of olfaction*, *Frontiers in Neuroscience*. Boca Raton: CRC Press/ Taylor & Francis.

Olfactory Bulb

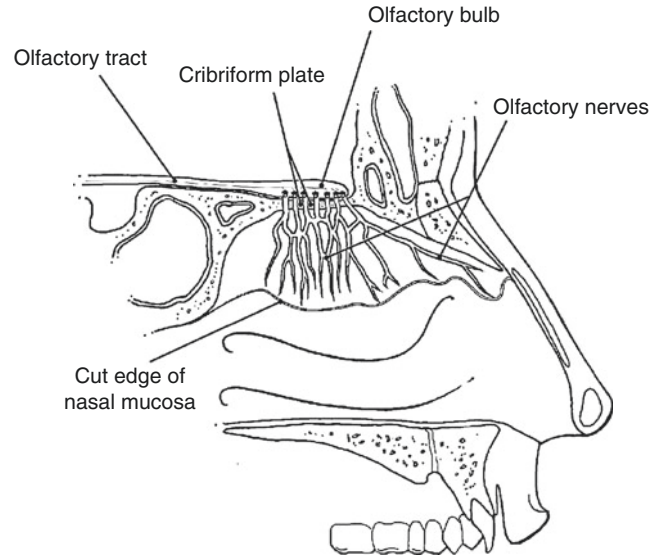
John E. Mendoza
Department of Psychiatry and Neuroscience, Tulane Medical School and SE Louisiana Veterans Healthcare System, New Orleans, LA, USA

Definition

Enlargement at the anterior end of the olfactory tract. The bulbs lie in the olfactory sulci between the gyrus rectus and the orbital gyri on the ventral surface of the frontal lobes. They receive olfactory input via axons of the epithelial cells of the nasal mucosa which pass through the cribriform plate of the ethmoid bone at the base of the skull (Fig. 1). Nerve cells within the bulbs then give rise to second-order axonal fibers which then make up the olfactory tract. The axons of the epithelial cells can easily be sheared when the brain is subjected to rapid deceleration as might occur in traumatic brain injury and this can result in loss of smell (anosmia).

Cross-References

► Olfaction
► Olfactory Tract



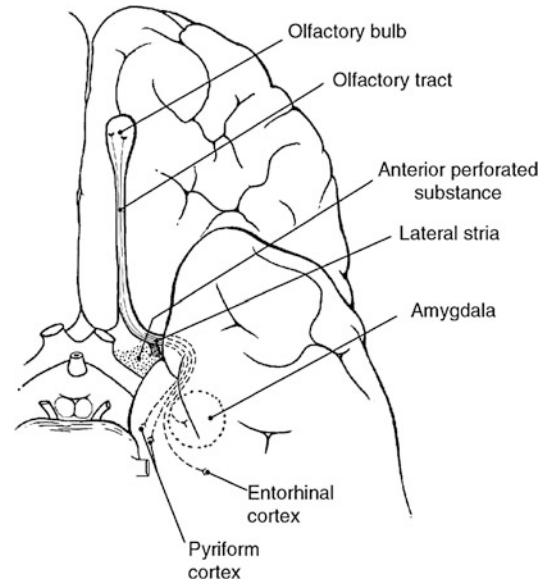
Olfactory Bulb, Fig. 1 Schematic drawing showing relationship of olfactory nerves in nasal mucosa to olfactory bulb lying above cribriform plate at the base of the skull

Olfactory Tract

John E. Mendoza
Department of Psychiatry and Neuroscience, Tulane Medical School and SE Louisiana Veterans Healthcare System, New Orleans, LA, USA

Definition

Prominent fiber pathways on the ventral surface of the frontal lobes. They lie in the olfactory sulci between the gyrus rectus (medially) and the more lateral frontal orbital gyri. These two parallel tracts contain second-order olfactory fibers from the olfactory bulbs and turn into the lateral olfactory stria at the level of the optic chiasm and terminate in the olfactory cortex in the anteromedial portion of the temporal lobes (Fig. 1). Compression of an olfactory tract, as might occur from a tumor or aneurysm can result in loss of smell in the ipsilateral nostril.



Olfactory Tract, Fig. 1 Schematic drawing showing olfactory bulb and tract on the ventral surface of the frontal lobes

Cross-References

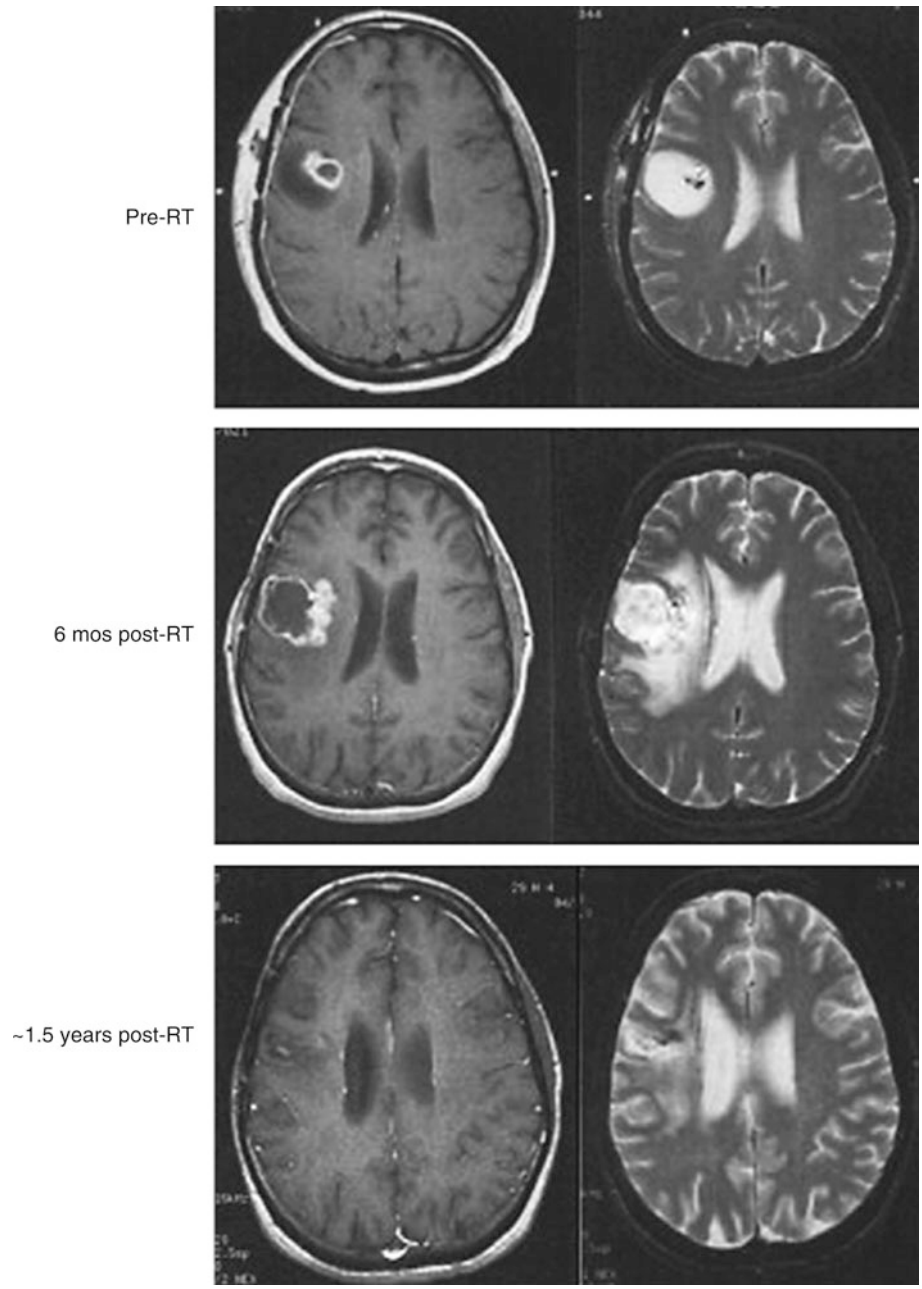
▶ Olfactory Bulb

Oligoastrocytoma

Jacqueline L. Cunningham
Department of Psychology, Children's Hospital of Philadelphia, Philadelphia, PA, USA

Definition

Oligoastrocytomas are variants of oligodendrogliomas, with their differential diagnosis resting on the detection of two different but definitely neoplastic glial components, one



Oligoastrocytoma, Fig. 1 Courtesy Hui-Kuo Shu, MD, and Carol Armstrong

oligodendroglial and the other astrocytic. Similarly to oligodendroglioma, the tumor is moderately cellular, may contain microcalcifications or cystic degeneration, and demonstrates little or no mitotic activity, necrosis, nor endothelial proliferation (Kleihues, Burger, Scheithauer). The median age for discovering the tumor is 35–45 years. A small percentage arises in children. Because the tumor involves cortical gray matter as well as white matter, it is usually associated with seizure activity. It usually occurs in the frontal or temporal lobe and less frequently in the parietal or occipital lobe. The tumor is considered a grade II lesion. Surgical resection is the main form of therapy, with chemotherapy advocated as the preferred therapy when recurrent disease is detected. Radiation therapy is reserved for disease, which does not respond to chemotherapy. Even with the achievement of gross total resection, practically every patient ultimately develops recurrence of their disease, usually at the surgical site and several years following resection. With recurrence, a malignant transformation to an anaplastic form of the oligoastrocytoma or to glioblastoma multiforme is possible (Kleihues and Cavenee).

Molecular genetic subtypes of oligoastrocytomas have been identified that correlate with the tumor location and survival outcomes. Tumor protein p53 (TP53), deletions involving chromosomes 1p and 19q, a promoter methylation protein MGMT, abnormalities in the PTEN tumor suppressor gene and the BRAF oncogene, and isocitrate dehydrogenase mutations have emerged, and 1P and 19q and MGMT are effective enough to be used as stratification factors in phase III clinical trials (Fig. 1).

Cross-References

- ▶ Astrocytoma
- ▶ Brain Tumor
- ▶ Glioblastoma Multiforme
- ▶ Neoplasms
- ▶ Oligodendroglioma
- ▶ Tumor Grade

References

Bourne, T. D., & Schiff, D. (2010). Update on molecular findings, management and outcome in low-grade gliomas. *Nature Reviews Neurology, 6*, 695–701.

Kleihues, P., & Cavenee, W. K. (2000). *Pathology and genetics of tumours of the nervous system*. Lyon: IARC Press.

Kleihues, P., Burger, P. D., & Scheithauer, B. W. (1993). *Histological typing of tumours of the central nervous system* (2nd ed.). New York: Springer-Verlag.

Oligodendroglioma

Mi-Yeoung Jo
Sherman Oaks, CA, USA

Definition

A type of slow-growing glial tumor that arises from oligodendrocytes or their precursor cells. Oligodendrogliomas are the third most common type of adult primary intracranial tumors and comprise only a small percentage of pediatric brain tumors. It occurs mostly in middle-aged adults and is more common in men than women (about 2:1). Oligodendrogliomas are typically classified as grade II (low grade) or III (anaplastic). They are most often found in the frontal and temporal lobes. Seizures, either focal or generalized, are often the first symptoms. Patients can also present with symptoms of increased intracranial pressures and, less frequently, focal cerebral signs such as hemiparesis. Frontal lobe tumors may cause mood and personality changes, and temporal lobe tumors may result in speech and memory problems. Surgery is one of the principal treatment options, and degree of resection may affect prognosis. Chemotherapy and radiotherapy are also frequently used as adjuvant treatments. However, in slow-growing, indolent tumors, observation may be the treatment of choice. Prognosis depends on various factors including age, tumor grade, and possibly genetic markers (Jaeckle et al. 2006).

Cross-References

- ▶ Brain Tumor
- ▶ Glioma
- ▶ Neoplasms
- ▶ Radiotherapy
- ▶ Tumor Grade

References and Readings

Engelhard, H. H., Stelea, A., & Mundt, A. (2003). Oligodendroglioma and anaplastic oligodendroglioma: Clinical features, treatment and prognosis. *Surgical Neurology, 60*, 443–456.

Jaeckle, K. A., Ballman, K. V., Rao, R. D., Jenkins, R. B., & Buckner, J. C. (2006). Current strategies in treatment of oligodendroglioma: Evolution of molecular signatures of response. *Journal of Clinical Oncology, 24*(8), 1246–1252.

Oppositional Defiant Disorder

Daniel W. Klyce
Virginia Commonwealth University – School of Medicine, Richmond, VA, USA

Synonyms

Externalizing behavior

Short Description or Definition

Oppositional defiant disorder (ODD) is defined in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; American Psychiatric Association 2013) by a pattern of (a) angry/irritable mood, (b) argumentative/defiant behavior, or (c) vindictiveness. The pattern of behavior must have been present for at least 6 months and be distressing to the individual or to others. The frequency and severity of symptoms should represent a pattern of function that is distinct from normative variation in expectations for behavior based on an individual's stage of development.

Categorization

The disorder is classified with the disruptive, impulse-control, and conduct disorders in *DSM-5*.

Current Knowledge

Development and Course

Symptoms of ODD tend to develop in childhood, though the pattern of mood and behavior characterized by this disorder may be present throughout the life span. A recent study by Nock et al. (2007) estimated a lifetime prevalence rate of 10.2%, with a median age of onset of 12.0 years (interquartile range 7.0–13.0). An earlier age of onset (i.e., younger than 8 years old) is typically associated with a more difficult course, including a pattern of function that is less responsive to treatment. Symptoms tend to abate as individuals age, with 70% of individuals experiencing symptom resolution by age 18. Throughout the life span, the duration of symptoms tends to be longer for males than for females (Nock et al. 2007).

Associated Disorders and Current Research

Oppositional defiant disorder is strongly associated with the development of other mental health disorders later in life, including mood disorders, anxiety disorders, substance use disorders, and other impulse control disorders. The disorder is often observed to be a precursor to more severe behavioral disturbances associated with disorders such as conduct disorder. As such, ODD and conduct disorder are commonly studied together. Recent investigations of possible subtypes of ODD have revealed evidence supporting an "irritable" and a "headstrong" subtype. Rowe et al. (2010) found that these subtypes held predictive value for the development of later disorders, with the irritable

subtype predicting later anxiety disorders and the headstrong subtype predicting the development of substance use disorders. Given high rates of comorbidity between ODD and attention-deficit/hyperactivity disorder (ADHD), ODD is often associated with neurocognitive deficits in attention and executive function; however, further research is needed to clarify the pattern of neuropsychological deficits that may be specific to ODD when controlling for ADHD (e.g., Mayes and Calhoun 2007).

Assessment and Treatment

Given that symptoms of ODD may be situationally bound, comprehensive assessment typically involves observation in various settings and information collected from multiple informants. Assessment of ODD should also involve effort to distinguish the observed behavior from what may be normative variation in the developmental, cultural, or environmental context (American Academy of Child and Adolescent Psychiatry 2006). Treatment for ODD typically involves parent training (e.g., behavioral interventions, contingency management) with focus on reinforcement of positive or prosocial behaviors, minimizing reinforcement of negative behaviors, and consequences for disruptive behaviors. Pharmacological interventions may also be considered as adjunctive treatments.

See Also

- ▶ Attention Deficit Hyperactivity Disorder
- ▶ Conduct Disorder
- ▶ Disruptive, Impulse-Control, and Conduct Disorders

References

American Academy of Child and Adolescent Psychiatry. (2006). Practice parameter for the assessment and treatment of children and adolescents with oppositional defiant disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 46*(1), 126–141.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association Publishing.

Mayes, S. D., & Calhoun, S. L. (2007). Learning, attention, writing, and processing speed in typical children and children with ADHD, autism, anxiety, depression, and oppositional-defiant disorder. *Child Neuropsychology, 13*(6), 469–493.

Nock, M. K., Kazdin, A. E., Hiripi, E., & Kessler, R. C. (2007). Lifetime prevalence, correlates, and persistence of oppositional defiant disorder: Results from the national comorbidity survey replication. *Journal of Child Psychology and Psychiatry, 48*(7), 703–713.

Rowe, R., Costello, J. E., Angold, A., Copeland, W. E., & Maughan, B. (2010). Developmental pathways in oppositional defiant disorder and conduct disorder. *Journal of Abnormal Psychology, 119*(4), 726–738.

Optic Ataxia

Joan Swearer[1] and Margaret Tuttle[2]
[1]Department of Neurology, University of Massachusetts Medical School, Worcester, MA, USA
[2]Department of Psychiatry, Massachusetts General Hosptial, Boston, MA, USA

Synonyms

Defective visual localization; Ocular dysmetria; Visuomotor ataxia

Short Description or Definition

Optic ataxia is a deficit of visually guided hand movements toward a normally perceived object in the peripheral visual field(s).

Epidemiology

Unknown, rare.

History

Deficits in reaching movements have been described since the late nineteenth century (Pisella

et al. 2008). In 1909, Rezsö Bálint (1909) used the term "optische ataxie" to describe the difficulties one of his patients had in making visually guided hand movements with his right hand. Reaching with his left hand was accurate. Reaching for parts of his body with his right hand with his eyes closed was accurate. Thus, the problem was neither visual nor motor, but rather visuomotor. The term "optic ataxia" was apt. The patient presented with additional symptoms of a significant restriction of visual attention and unilateral neglect. At autopsy, the patient was found to have bilateral occipitoparietal lesions.

Holmes (1918) described a related syndrome of "visual disorientation" in soldiers with bilateral parietal lesions. These patients had significant difficulty in maintaining visual fixation, and their gaze wandered when searching for objects in peripheral vision.

Optic ataxia is often encountered in the context of Bálint's syndrome, which is now generally considered to consist of a triad of symptoms including (1) a deficit of visually guided hand movements (optic ataxia), (2) difficulties in visual fixation and scanning ("optic apraxia"), and (3) an extreme restriction of visual attention (similar to "simultanagnosia"). Optic ataxia can be a persistent and residual deficit in patients recovering from Bálint's syndrome. It can also occur in isolation, but it was not until 1967 when optic ataxia was identified as a specific entity separate from Bálint's syndrome (Garcin et al. 1967).

Clinical Presentation

Bálint's patient described his right hand as clumsy and reported often lighting a cigarette in its middle instead of its end. A patient with optic ataxia might reach for food on someone else's plate rather than his own. On neurological exam, the patient may appear to have cerebellar ataxia, with overshooting and undershooting the target. The difference is that with optic ataxia, patients can reach accurately if they have proprioceptive cues. They also lack intention tremor and dysdiadochokinesia.

There may also be other impairments in optic ataxia, such as difficulty shaping the hand correctly for grasping, inability to correct ongoing movements, and failure to recognize obstacles in the path to the object. It is also possible to see impaired ability to touch an object with a toe under visual guidance alone.

Neuropsychology of Optic Ataxia

Patients with optic ataxia have a deficit in reaching out to objects in their peripheral visual field, despite normal recognition and naming of the object and in the absence of primary visual or motor deficits. Reaching is normal when performed with central fixation of the object. With bilateral lesions, as in Bálint's syndrome, optic ataxia is apparent in both visual fields, and there may or may not be an inferior quadrant visual field defect. Isolated optic ataxia following a unilateral lesion results in contralateral symptoms ("field effect"), with greater relative errors performed by the contralesional hand ("hand effect"). Proprioception is largely spared as patients can, with eyes closed, point to their body parts touched by the examiner and point to named body parts (e.g., their nose) (Pisella et al. 2008). Isolated optic ataxia is associated with lesions located around the intraparietal sulcus and superior parietal lobe or from disconnections from the visual and visuomotor areas to the frontal lobe (Devinsky and D'Esposito 2004). Bálint's syndrome is a result of bilateral damage to the occipitoparietal region. This is usually due to infarctions in the border zone (watershed) between the territories of the anterior and posterior cerebral arteries from sudden and severe hypotension (Damasio et al. 2000).

The neuropsychology of optic ataxia is incompletely understood. Andersen et al. (2014) provide a detailed systematic review of ongoing research. Optic ataxia is now understood to be a high-order deficit, at an integrative sensorimotor level, that can occur with lesions in the posterior parietal cortex (PPC) associated with a variety of conditions, ranging from neurodegenerative diseases such as posterior cortical atrophy to traumatic

brain injury, brain tumors, and infections. Pure optic ataxia is more rare than Bálint's syndrome because of the more localized and discrete lesions that produce optic ataxia alone. It has been found that optic ataxia in isolation can be caused by damage to a specific region within the PPC called the parietal reach region.

As described by Andersen et al. (2014), there are currently three frameworks proposed for optic ataxia: disruption of (1) visuomotor processing, (2) visual orientation, or (3) online visuomotor control. It is difficult to distinguish between these models with human case reports alone, so research is ongoing with nonhuman primate and animal subjects. Each framework is conceptualized as consisting of multiple functional modules. Bálint's syndrome and optic ataxia are believed to be caused by disruption of multiple functional modules within the PPC. Research into the framework for optic ataxia has the added benefit of elucidating the functional organization of the PPC for action planning.

Outcomes and Treatment

Functional outcome in cases of isolated optic ataxia is usually good because the lesion is not extensive and reaching behavior under conditions of central fixation is normal and visuospatial functions are spared. Patients spontaneously adapt to the deficit by moving central fixation to the target in their peripheral visual field. Functional prognosis is poor in full Bálint's syndrome due to the extent of the bilateral lesions and the presence of the other symptoms of the syndrome (Pisella et al. 2008).

Cross-References

► Balint's Syndrome

References and Readings

Andersen, R. A., Andersen, K. N., Hwang, E. J., & Hauschild, M. (2014). Optic ataxia: From Bálint's syndrome to the parietal reach region. *Neuron, 81*(5), 967–983.

Bálint, R. (1909). Seelenlähmung des "Schauens," optische Ataxie, räumliche Störung der Aufmerksamkeit. *Monatsschrift für Psychiatrie und Neurologie, 25*, 51–81.

Damasio, A. R., Tranel, D., & Rizzo, M. (2000). Disorders of complex visual processing. In M.-M. Mesulam (Ed.), *Principles of behavioural and cognitive neurology* (2nd ed., pp. 332–372). New York: Oxford University Press.

Devinsky, O., & D'Esposito, M. (2004). *Neurology of cognitive and behavioural disorders*. New York: Oxford University Press.

Garcin, R., Rondot, P., & Recondo, J. (1967). Optic ataxia localized in 2 left homonymous visual hemifields (clinical study with film presentation). *Revue Neurologique (Paris), 116*, 707–714.

Holmes, G. (1918). Disturbances of visual orientation. *British Journal of Ophthalmology, 2*, 449–468.

McIntosh, R.D. (2016). Optic ataxia. In *Encyclopædia Britannica*. Retrieved from http://www.britannica.com/science/optic-ataxia.

Pisella, L., Ota, H., Vighetto, A., & Rossetti, Y. (2008). Optic ataxia and Bálint's syndrome: Neuropsychological and neurophysiological prospects. In G. Goldenberg & B. L. Miller (Eds.), *Handbook of clinical neurology, Vol. 88 (3rd series) neuropsychology and behavioural neurology* (pp. 393–415). Amsterdam: Elsevier.

Optic Chiasm

John E. Mendoza
Department of Psychiatry and Neuroscience, Tulane Medical School and SE Louisiana Veterans Healthcare System, New Orleans, LA, USA

Definition

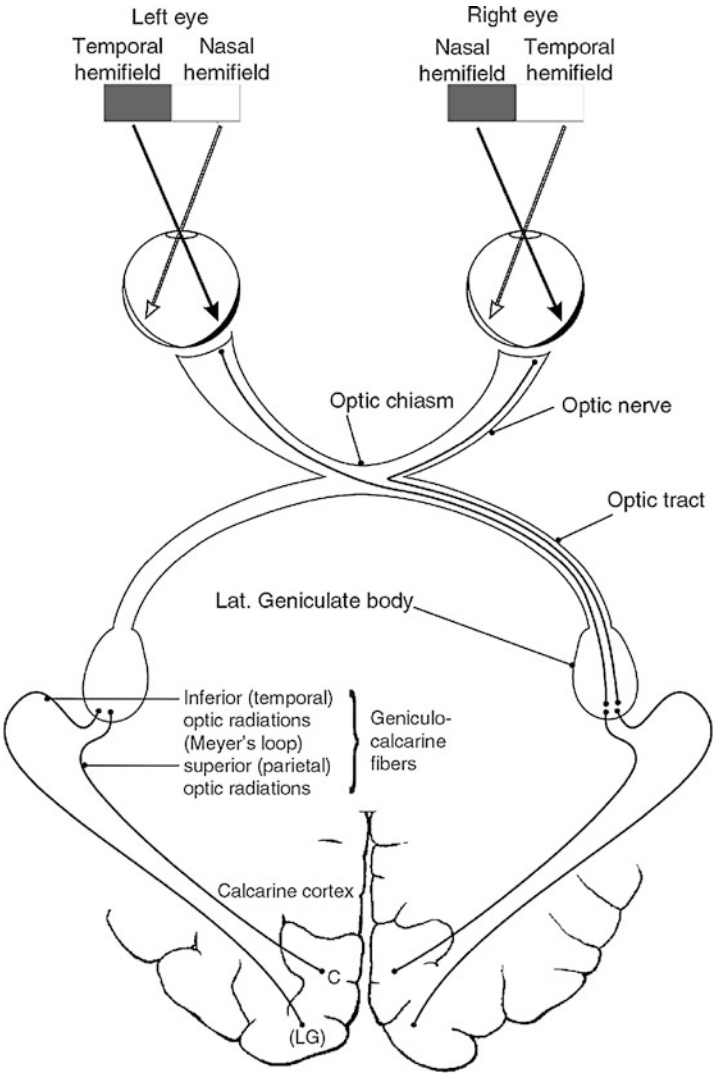
"X"-shaped fiber pathway that lies on the posterior ventral portion of the frontal lobes just anterior to the pituitary stalk.

Current Knowledge

The chiasm represents an intersectional transition between the *optic nerves* (anteriorly) and the *optic tracts* (posteriorly), providing an anatomical

mechanism for the partial crossing of the optic nerves. While the optic nerves that originate in the ganglion cells in the temporal halves of the retina remain ipsilateral as they proceed posteriorly (as the optic tracts) to the lateral geniculates, those that come from the nasal halves of the retina in each eye cross in the central portion of the optic chiasm before continuing to the lateral geniculates (Fig. 1). It is this partial crossing that allows visual cortex to use both eyes to see images in the contralateral visual field. Light rays travel in straight lines as they pass through the pupillary opening. Thus, light rays coming from the left will stimulate the nasal retina of the left eye and the temporal retina of the right eye. Due to this partial crossing of the nasal fibers, for example, information derived from light rays from the left visual field that strike each eye will be channelled to the right hemisphere.

Because of the arrangement of the temporal and nasal fibers in the chiasm, a tumor such as a pituitary adenoma, which can exert local pressure in the central part of the chiasm (where the nasal retinal fibers cross), often results in a bi-temporal visual field defect. By contrast, a tumor which may surround the chiasm exerting pressure on



Optic Chiasm,
Fig. 1 Axial MRI image at level of the midbrain showing optic chiasm and optic nerves

both sides (thus affecting the uncrossed fibers from the temporal portions of both retinas) can produce a bi-nasal field cut.

Cross-References

► Visual System

Optic Glioma, Optic Pathway Glioma

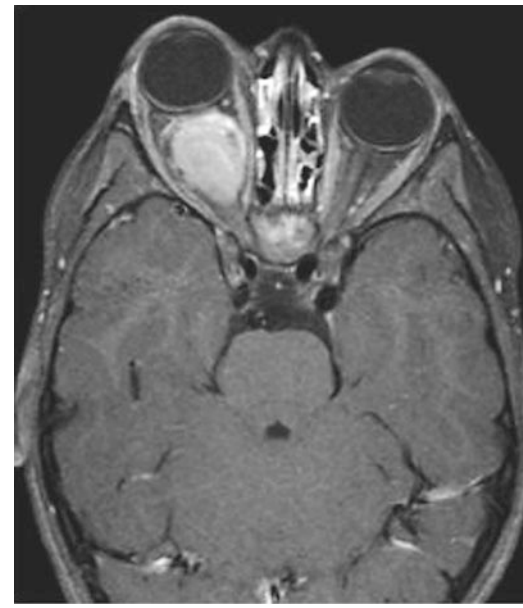
Robert Rider
Department of Psychology, Drexel University, Philadelphia, PA, USA

Synonyms

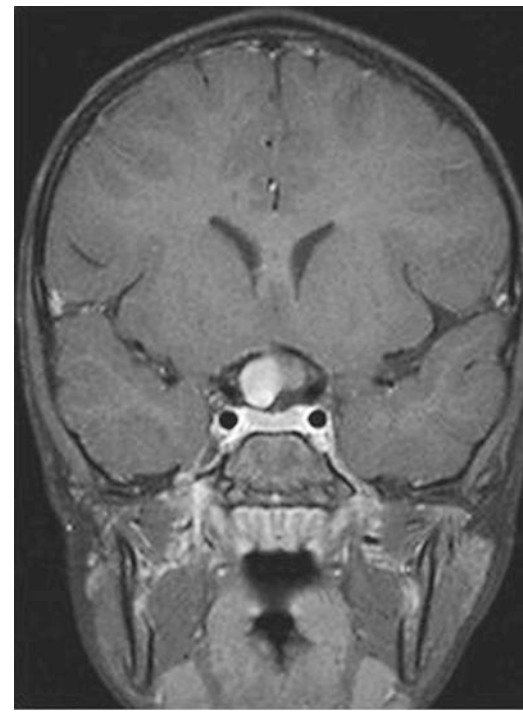
Optic nerve glioma

Definition

Optic gliomas and optic pathway gliomas are most frequently diagnosed in children and adolescents and twice as frequently in girls. Tumors are generally benign but can sometimes become malignant. These types of tumors may occur in any or all of the following areas: optic nerve, optic chiasm, optic tract, hypothalamus, or within the orbit. Cases in which tumor growth is restricted to the optic nerve occur almost exclusively in the context of Neurofibromatosis type I. Symptoms and signs of optic gliomas and optic pathway gliomas manifest as changes in visual functioning, such as visual loss, strabismus, nystagmus, papilledema, homonymous, or heteronymous hemianopsia, or symptoms of hypothalamic dysfunction depending on the regions involved (Figs. 1 and 2).



Optic Glioma, Optic Pathway Glioma, Fig. 1 Courtesy Jean Belasco, Michael Fisher, Beverly Lange, Jane Minturn, and Peter Phillips



Optic Glioma, Optic Pathway Glioma, Fig. 2 Courtesy Jean Belasco, Michael Fisher, Beverly Lange, Jane Minturn, and Peter Phillips

Optic Nerve

John E. Mendoza
Department of Psychiatry and Neuroscience, Tulane Medical School and SE Louisiana Veterans Healthcare System, New Orleans, LA, USA

Definition

That portion of the visual pathway that extends from the eye to the optic chiasm. The optic nerve consists of the axons of the ganglion cells of the retina. With a few exceptions, most then travel uninterrupted to the lateral geniculates. However, only that portion which lies between the optic disc and the chiasm is referred to as the *optic nerve*. The post-chiasmatic fibers are known as the *optic tracts*. Because of the partial crossing that takes place in the chiasm, the optic nerve contains fibers from one eye, whereas the optic tract contains fibers from both eyes. Consequently a lesion involving an optic nerve can result in monocular visual loss.

Cross-References

- ▶ Optic Chiasm
- ▶ Visual System

Optic Neuropathy

Kathleen L. Fuchs
Department of Neurology, University of Virginia Health System, Charlottesville, VA, USA

Definition

A change in or loss of vision as a result of damage to the optic nerve. For example, demyelination of the optic nerve (optic neuritis) is a common early symptom of multiple sclerosis (MS) that can result in temporary monocular blindness. Other causes of optic neuropathy include compression lesions (i.e., tumors), ischemia, Leber's hereditary optic neuropathy, and chronic relapsing inflammatory optic neuropathy (CRION), which is characterized by recurring episodes of optic neuritis marked by severe visual loss and orbital pain. In this condition, a contrast-enhanced MRI may show swelling of the optic nerve but no evidence of the demyelinating lesions in the brain characteristic of MS.

Cross-References

- ▶ Multiple Sclerosis
- ▶ Optic Nerve
- ▶ Visual Evoked Potentials

References and Reading

Miller, D., & Compston, A. (2006). The differential diagnosis of multiple sclerosis. In A. Compston et al. (Eds.), *McAlpine's multiple sclerosis* (4th ed., pp. 389–437). Philadelphia, PA: Elsevier.

Optic Radiations

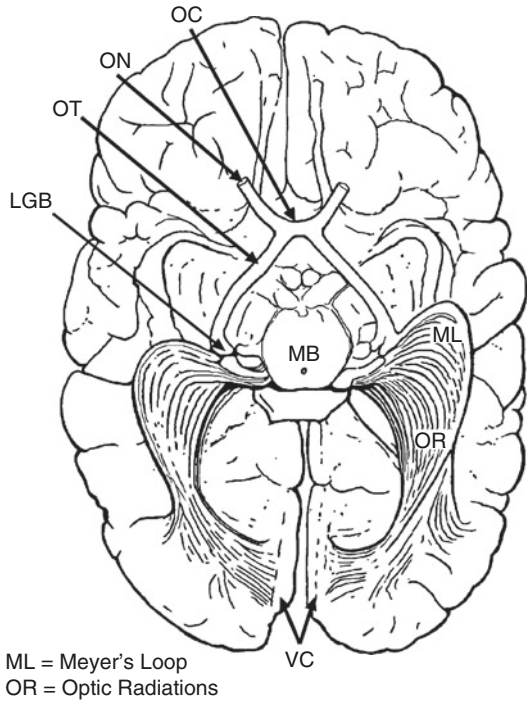
John E. Mendoza
Department of Psychiatry and Neuroscience, Tulane Medical School and SE Louisiana Veterans Healthcare System, New Orleans, LA, USA

Synonyms

Geniculocalcarine fibers (pathway)

Definition

Posterior portion of the visual pathways extending from the lateral geniculates to the primary visual cortex (Fig. 1). Unlike the optic nerves and optic tracts which form compact, easily identified



Optic Radiations, Fig. 1 Schematic drawing of axial section of brain at the level of the midbrain showing the optic radiations and their temporal extensions (Meyer's loop) from the lateral geniculate bodies to the visual cortex. Legends: *LGB* lateral geniculate body, *MB* midbrain, *OC* optic chiasm, *ON* optic nerve, *OT* optic tract, *VC* visual cortex

structures, the axons comprising the optic radiations fan out and form a wide band of fibers that make up part of the subcortical white matter pathways underlying the parietal and temporal cortices. That portion of this band of fibers that represents input from the ganglion cells in the upper half of the retina, or inferior visual field, remains in a more superior position, underlying the parietal lobe. Conversely, the fibers representing visual input to the lower half of the retina (superior visual field) maintain a more ventral, temporal route (▶ Meyer's loop). The fibers making up the optic radiations as a whole in each hemisphere represent visual stimuli striking the temporal portions of the retina in the ipsilateral eye and the nasal retina of the contralateral eye (contralateral visual field). Because the optic radiations are so spread out, lesions rarely affect the entire pathway. As a result, partial contralateral visual field deficits (e.g., quadrantanopia) rather than complete hemianopia typically result from such lesions.

Cross-References

- ▶ Meyer's Loop
- ▶ Quadrantanopia
- ▶ Visual System

References and Readings

Mendoza, J. E., & Foundas, A. L. (2008). The cranial nerves. In J. E. Mendoza & A. L. Foundas (Eds.), *Clinical neuroanatomy – a neurobehavioral approach* (pp. 107–152). New York: Springer.

Optic Tract

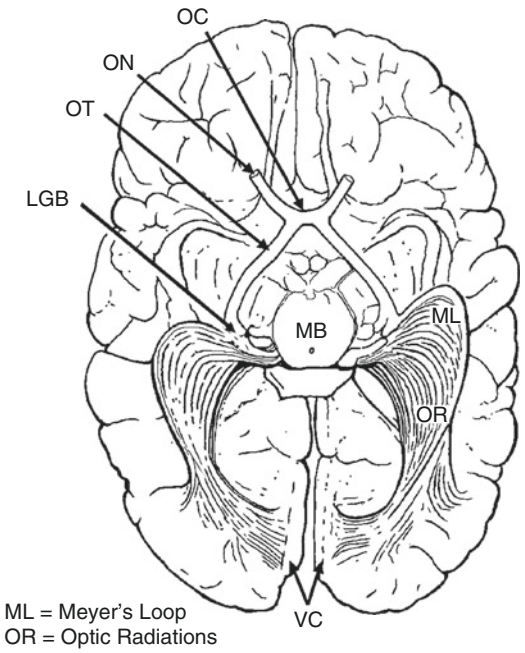
John E. Mendoza
Department of Psychiatry and Neuroscience, Tulane Medical School and SE Louisiana Veterans Healthcare System, New Orleans, LA, USA

Definition

That part of the visual pathway that extends from the optic chiasm (OC) to the lateral geniculate (LGB) nuclei of the thalamus (Fig. 1). Consisting of both crossed (nasal) and uncrossed (temporal) fibers, the optic tracts (OT) are visible on the ventral surface of the brain from the chiasm to the level of the mammillary bodies where they continue posteriorly between the cerebral peduncles and the medial aspect of the temporal lobe until reaching the lateral geniculate nucleus. Because they are a rather compact pathway, unlike the optic radiations (OR) and contain both temporal and nasal retinal fibers, lesions affecting the tract will commonly result in a contralateral homonymous hemianopsia.

Cross-References

- ▶ Homonymous Hemianopsia
- ▶ Visual System



Optic Tract, Fig. 1 Visual pathways (in order to avoid having to put in a large legend for the abbreviations, we can incorporate the relevant ones into the text – see below). If you want to add the others in as a separate legend, they are *ON* optic nerve, *MB* midbrain, *ML* Meyer's loop, *VC* visual cortex

References and Readings

Mendoza, J. E., & Foundas, A. L. (2008). The cranial nerves. In J. E. Mendoza & A. L. Foundas (Eds.), *Clinical neuroanatomy – A neurobehavioral approach* (pp. 107–152). New York: Springer.

Optokinetic Reflex

Ronald A. Cohen
Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA
Center for Cognitive Aging and Memory, McKnight Brain Institute, University of Florida, Gainesville, FL, USA

Definition

The optokinetic reflex causes eye movement in response to objects moving in the periphery while the head is stationary.

Current Knowledge

The optokinetic reflex is extremely important for stabilizing the image of visual input on the retina and provides for primary control of this capacity when the head is stationary. It interacts with the vestibular optic reflex when the head is rotating in stationary visual environment. Negative feedback systems associated with the optokinetic reflex enable the retina to achieve and maintain stabilization over time. It first appears in infants at approximately 6 months of age and remains throughout life. This reflex plays an important role in everyday experience for most people in the context of driving, as objects tend to move rapidly past the driver in the periphery, which requires a rapid ocular response, while maintaining primary fixation on the road. In this sense, it likely had a value for primate survival enabling the detection of predators and other potential dangers outside of the central field of vision.

From a neuropsychological perspective, the optokinetic reflex is of interest and has clinical relevance for a number of reasons. Nystagmus often occurs when this reflex is disturbed due to drug, traumatic, or extreme conditions involving body or environmental motion. Nystagmus is a condition that involves halting smooth pursuit movements in one direction, alternating with saccadic movements in the opposite direction, most often horizontally. This results in a sawtooth pattern of movement, which creates instability of the visual field and interacts with the vestibular system to affect balance and positional sense. Optokinetic reflex is often impaired among substance abusers. Patients with Wernicke-Korsakoff syndrome secondary to chronic alcoholism and malnutrition typically exhibit nystagmus and reduced gain (i.e., sensitivity) of this reflex (Probst 1983). Impairments of the optokinetic reflex occur secondary to a variety of brain disorders. Patients with multiple sclerosis often exhibit disturbances of this reflex (Todd et al. 2001). Other brain diseases in which impaired optokinetic reflex is found include brain tumor, stroke, ataxia, and head trauma. Because this ocular response is reflexive, requiring no training, shows minimal tendency to fatigue or adapt, and produces a reliable and quantifiable signal when eye movements are required, some

investigators have argued that it has the potential to be a rapid neurobehavioral screening test of brain dysfunction (Cahill and Nathans 2008). Currently, it is not widely evaluated as part of a standard neuropsychological assessment, though clinical observation of eye movements should be part of a clinical work-up, whether done through use of eye movement recording or behavioral observation.

Cross-References

- ▶ Dorsal Visual Pathway
- ▶ Oculomotor Nerve
- ▶ Saccadic Eye Movements
- ▶ Trochlear Nerve

References and Readings

Cahill, H., & Nathans, J. (2008). The optokinetic reflex as a tool for quantitative analyses of nervous system function in mice: Application to genetic and drug-induced variation. *PLoS ONE, 3*(4), e2055.

Probst, R. (1983). Neuro-otologic findings in the Wernicke-Korsakoff syndrome. *HNO, 31*(4), 123–127.

Todd, L., King, J., Darlington, C. L., & Smith, P. F. (2001). Optokinetic reflex dysfunction in multiple sclerosis. *Neuroreport, 12*(7), 1399–1402.

Orbitofrontal System

Denise Krch
Kessler Foundation, East Hanover, NJ, USA

Definition

The orbitofrontal system comprises two parallel subsystems (i.e., the lateral and medial), which share substantial overlap in anatomy and behavioral functions. The orbitofrontal subsystems originate in Brodmann's areas 10 and 11; the lateral orbitofrontal system projects to the ventromedial caudate, and the medial orbitofrontal system projects to the ventral striatum. Both subsystems then project to the medial portion of the globus pallidus interna (GPi) and the rostromedial substantia nigra pars reticulata (SNr). Projections are sent from the GPi and SNr to the medial magnocellular division of the ventral anterior thalamus, as well as the inferomedial sector of the magnocellular division of the mediodorsal thalamus. These subsystems are then completed by connecting back to the orbitofrontal cortex. The orbitofrontal system also has extensive connections with the amygdala, hippocampus, and olfactory system. The orbital frontal branches of the anterior cerebral artery (ACA) and the middle cerebral artery supply the lateral orbital cortex. The proximal anterior cerebral, posterior cerebral, carotid, posterior communicating, and basilar arteries supply other aspects of the orbitofrontal system.

Current Knowledge

The orbitofrontal system is the neocortical representation of the limbic system and mediates empathetic and socially appropriate responses. It is involved in the determination of the appropriate time, place, and strategy for environmentally elicited behavioral responses, impulse control, maintenance of set, and maintenance of ongoing behaviors. Damage to any part of this system may result in an orbitofrontal syndrome. A lateral orbitofrontal syndrome may result in personality change (i.e., emotional incontinence, tactlessness, impulsivity, irritability, aggressive outbursts, disinhibited sexual and hunger drive, undue familiarity), environmental dependency (i.e., utilization behavior, imitation behavior), mood disorders (i.e., depression, lability, mania), and obsessive-compulsive spectrum symptoms. A medial orbitofrontal syndrome may result in personality change (i.e., emotional incontinence, impulsivity, antisocial behavior), abnormal automatic responses to socially meaningful stimuli, and poor stimulus-response learning. Patients with orbitofrontal system dysfunction often perform poorly on tasks that require changing sets (e.g., Trail Making Test B) or response inhibition (e.g., go/no-go tasks).

Etiologies most commonly associated with orbitofrontal syndrome include Huntington's disease (i.e., caudate dysfunction) and manganese intoxication (i.e., globus pallidus lesions). Other etiologies in which orbitofrontal syndrome may present include carbon monoxide toxicity, Creutzfeldt-Jakob disease, frontotemporal dementia, Tourette's syndrome, severe closed brain injuries (e.g., Phineas Gage), herpes simplex viral encephalitis, multiple sclerosis, neuroacanthocytosis, obsessive-compulsive disorder, postencephalitic Parkinson's disease, ruptured ACA aneurysm, stroke, and tumor.

Cross-References

- ▶ Frontal Lobe Syndrome
- ▶ Gage, Phineas (1823–1860)
- ▶ Go/No-go Testing
- ▶ Huntington's Disease
- ▶ Trail Making Test
- ▶ Utilization Behavior

References and Reading

Alexander, G. E., DeLong, M. R., & Strick, P. L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience, 9*, 357–381.

Miller, B. L., & Cummings, J. L. (Eds.). (1999). *The human frontal lobes: Functions and disorders*. New York: Guilford Press.

Organic Brain Syndrome

Bruce J. Diamond and Krista Dettle
Department of Psychology, William Paterson University, Wayne, NJ, USA

Synonyms

Chronic organic brain syndrome; Organic brain disease; Organic mental disorder (OMD); Organic mental syndrome (DSM-III-R)

Short Description or Definition

Organic brain syndrome is a general diagnostic term earlier used to group symptoms related to brain dysfunction of organic etiology (Dorland 2011). Since the publication of DSM-IV (1994), new developments in technology have led to a greater understanding of the brain, including neural processes, pathology, etiology, and prognosis (Foley and Heck 2014). Breakthroughs in genetics, neuroimaging, and neurotoxicology gave way to a revision of theory and terminology that formerly divided functional and organic syndromes (Goldstein 2014).

Within this context, organic brain syndrome, later described as delirium, dementia, amnestic, and other cognitive disorders, is now referred to as neurocognitive disorders (Goldstein 2014). Given the heterogeneity of brain dysfunction, the former term was found to be too imprecise in addressing clinically relevant complexities, including implications for treatment, management, and discharge planning (Goldstein 2014). Contemporary psychiatry discourages the use of the term organic brain syndrome, as it implies that other mental disorders are not organic in nature (Dorland 2011). These developments have been accompanied by improvements in differential diagnosis and the establishment of diagnostic criteria for specific disease processes, including methods to assess cognition, function, and presence of decline in the DSM-5 (Foley and Heck 2014).

Categorization

Neurocognitive Disorders comprise a variety of diseases, conditions, and disorders including those affecting the central nervous system (CNS), vascular disorders, trauma induced injury, metabolic, endocrine or nutritional disease, epilepsy, infectious diseases, drug- and alcohol-related complications, toxins, and a variety of other diseases (e.g., kidney, liver, cancer) (Berg et al. 1994).

Diagnostic procedure now encompasses a differentiation between mild or major neurocognitive disorder, followed by assignment of an etiological

classification (e.g., Alzheimer's NCD) (Blazer 2013), and is structured around distinct cognitive domains (e.g., executive function) (Reichenberg 2013). Major neurocognitive disorder subsumes the term dementia as a symptom (Reichenberg 2013). Delirium remains a separate classification with new criteria (Foley and Heck 2014).

DSM-5 (American Psychiatric Association 2013) specifications include neurocognitive disorder due to:

- Alzheimer's disease
- Frontotemporal lobar degeneration
- Lewy body disease
- Vascular disease
- Traumatic brain injury
- Substance/medication use
- HIV infection
- Prion disease
- Parkinson's disease
- Huntington's disease
- Other medical condition
- Multiple etiologies
- Unspecified (Table 1)

Epidemiological Factors

Delirium is one of the most common mental disorders, and it has been estimated that 10–15% of medical-surgical patients and up to 50% of geriatric patients satisfy the criteria for delirium during hospitalization (Khandelwal 1996). Approximately 1.7 million people sustain a TBI, about 52,000 die, and 275,000 are hospitalized. Approximately 511,000 TBIs occur among children ages 0–14 years. Falls are the leading cause of TBI, and rates are highest for children ages 0–4 years and for adults 65 years or older with adults ages 75 years or older having the highest rates of TBI-related hospitalization and death. In every age group, TBI rates are higher for males than for females (Faul et al. 2010).

Natural History and Outcomes

The course of neurocognitive disorder may be insidious or sudden, and the outcome can range from reversible (i.e., 10–30% of dementia cases) (Clarfield 1988) to irreversible. Symptoms will vary as a function of the specific disease, disorder, or condition.

Neuropsychological and Psychological Outcomes

Sequelae that figure prominently include dysnomia and impairments in memory, executive function, processing speed, visuospatial processing, fluency, learning, working memory, and changes in intelligence. Psychological symptoms include possible mood swings, changes in psychosocial functioning, personality, and affective regulation. Neurological symptoms may include tremors, bradykinesia, dyskinesia, and impairments in motor, perceptual-motor, and sensory systems.

Assessment and Treatment

Both battery assessments and more specialized assessments will have to take into account etiological categories and associated symptom clusters. Treatments are diverse and include pharmacological interventions, cognitive rehabilitation, behavioral health interventions, and psychosocial training.

Overall, the current clinical conceptualization of neurocognitive disorders, formerly referenced as organic brain syndrome, promotes enhanced diagnostic sensitivity and treatment planning and is supported by recent research distinguishing specific syndromes (Foley and Heck 2014), reflecting continuing growth in the field (Blazer 2013).

Cross-References

- ▶ Alzheimer's Disease
- ▶ Encephalitis (Viral)
- ▶ Herpes Simplex Encephalitis
- ▶ Kuru
- ▶ Meningitis
- ▶ Multi-infarct Dementia
- ▶ Multiple Sclerosis

Organic Brain Syndrome, Table 1 Neurocognitive disorders. Specific etiologies, course, symptoms, and treatments

Disorders	Course	Symptoms	Treatment
Degenerative Parkinson's	Insidious, progressive worsening	Bradykinesia, resting tremor, rigidity, memory disturbance, orientation, expressionless face. Ninety percent of patients exhibit neuropsychological impairments (Cumming 1985)	Levodopa, dopamine agonists
Huntington's	Basal ganglia disorder with patient demise usually occurring within 15 years	Impairments in memory, executive function visual-spatial functions affect, motor impairments dyskinesia, processing speed (Folstein 1989; Brandt and Butters 1986; Brown et al. 2001)	Antidepressants and treatment for involuntary movements (e.g., tetrabenazine) and social support techniques (Folstein 1991)
Multiple sclerosis	Chronic, unpredictable course	Disturbances in working memory, processing speed, mood, and motor/sensory systems (Diamond et al. 2008)	Glatiramer acetate (Copaxone®)
			Interferon beta 1a – intramuscular (Avonex®)
			Interferon beta 1a – subcutaneous (Rebif®)
			Interferon beta 1b (Betaseron®)
			Mitoxantrone (Novantrone®)
			Natalizumab (Tysabri®)
Alzheimer's disease	Insidious onset with early onset associated with more severe and rapid course (Lezak 1983)	Memory impairments, executive dysfunction, lower performance IQ than verbal, reduced verbal fluency, dysnomia, visuospatial deficits	Donepezil (Aricept)
			Galantamine (Razadyne)
			Memantine (Namenda)
			Rivastigimine (Exelon)
			Tacrine (Cognex)
Vascular disorders: multi-infarct dementia	Rapid onset, step-wise deterioration	Pseudobulbar state (exaggerated laughter, crying). Memory impairments and mood swings	Behavioral and drug treatment for high blood pressure, high cholesterol, and diabetes
Subarachnoid hemorrhage of the ACo artery	Acute onset	Disturbances in memory, executive function, information processing, and personality	Cognitive rehabilitation, i.e., executive orthotics for processing visual-spatial information (Diamond et al. 1997)
Trauma induced injury: concussions, contusions, open and closed head	Sudden onset	Disturbances in cognition, affective, psychosocial functions	Neuropsychological rehabilitation and psychosocial training
Infectious diseases meningitis	Inflammation resulting in increased cerebral pressure can rapidly progress to drowsiness, stupor, and possibly coma	May be accompanied by motor, perceptual-motor, and cognitive-neurologic difficulties	Antibiotics and following vaccine schedule can help prevent bacterial meningitis (Zeuter and Zaiter 2015)
Drugs alcohol/ nutritional	Reversible, Wernicke-Korsakoff syndrome	Memory, motor, personality	Thiamine
Toxins: e.g., lead	Insidious	Children: learning impairments, encephalopathy, decreased intelligence and growth	Chelation therapy, environmental changes

References and Readings

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Berg, R. A., Franzen, M. D., & Wedding, D. (1994). *Screening for brain impairment: A manual for mental health practice*. New York: Springer Publishers.

Blazer, D. (2013). Neurocognitive disorders in DSM-5. *The American Journal of Psychiatry, 170*(6), 585–587. https://doi.org/10.1176/appi.ajp.2013.13020179.

Brandt, J., & Butters, N. (1986). The neuropsychology of huntington's disease. *Trends in Neurosciences, 9*, 118–120.

Brown, R. G., Redondo-Verge, L., Chacon, J. R., Lucas, M. L., & Channon, S. (2001). Dissociation between intentional and incidental sequence learning in Huntington's disease. *Brain: A Journal of Neurology, 124*, 2188–2202.

Clarfield, A. M. (1988). The reversible dementias: Do they reverse? *Annals of Internal Medicine, 109*, 476–486.

Cummings, J. L. (1985). Organic delusions: Phenomenology, anatomical correlations, and review. *British Journal of Psychiatry, 146*, 184–197.

Diamond, B. J., DeLuca, J., & Kelley, S. M. (1997). Memory and executive functions in amnesic and non-amnesic patients with aneurysms of the anterior communicating artery. *Brain: A Journal of Neurology, 120*, 1015–1025.

Diamond, B. J., Johnson, S. K., & Kaufman, M. (2008). Relationships between information processing, depression, fatigue, and cognition in multiple sclerosis. *Archives of Clinical Neuropsychology, 32*, 189–199.

Organic Mental Disorder. (2011). In W. Dorland, *Dorland's illustrated medical dictionary*. Philadelphia: Elsevier Health Sciences. Retrieved from https://ezproxy.wpunj.edu/login?url=http://ezproxy.wpunj.edu:4276/content/entry/ehsdorland/organic_mental_disorder/0

Faul, M., Xu, L., Wald, M. M., & Coronado, V. (2010). *Traumatic brain injury in the United States: Emergency department visits, hospitalizations and deaths, 2002–2006*. Atlanta: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.

Foley, J. M., & Heck, A. L. (2014). Neurocognitive disorders in aging: A primer on DSM-5 changes and framework for application to practice. *Clinical Gerontologist: The Journal of Aging and Mental Health, 37*(4), 317–346. https://doi.org/10.1080/07317115.2014.907595.

Folstein, S. (1989). *Huntington's disease: A disorder of families*. Baltimore: Johns Hopkins University Press.

Folstein, S. (1991). The psychopathology of Huntington's disease. In P. R. McHugh & V. A. McKusick (Eds.), *Genes, brain, and behavior* (pp. 181–191). New York: Raven Press.

Goldstein, G. (2014). Neurocognitive disorders. In D. C. Beidel, B. C. Frueh, M. Hersen, D. C. Beidel, B. C. Frueh, & M. Hersen (Eds.), *Adult psychopathology and diagnosis* (7th ed., pp. 705–738). Hoboken: Wiley.

Khandelwal, S. K. (1996). Organic brain syndrome. In D. Tantam, L. Appleby, & A. Duncan (Eds.), *Psychiatry for the developing world* (pp. 132–159). Washington, DC: American Psychiatric Publishers.

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

National Institute of Health. (2006). Organic brain syndrome. In *Medline plus medical encyclopedia*. Retrieved from http://www.nlm.nih.gov/medlineplus/ency/article/001401.htm.

Reichenberg, L. W. (2013). *DSM-5 essentials the savvy clinician's guide to the changes in criteria*. Somerset: Wiley. Retrieved from http://www.ebrary.com

Zeuter, A. M., & Zaiter, A. (2015). Infectious meningitis. *Clinical Microbiology Newsletter, 37*, 43–51. https://doi.org/10.1016/j.clinmicnews.2015.02.004.

Orientation

Candace Hughes and Jacqueline Estrada
School of Psychology, The Chicago School of Professional Psychology, Chicago, IL, USA

Updated Definition

Orientation relates to the relative position of oneself and/or that of objects in the environment. The concept of orientation is a complex one, for it not only relates to personal navigation replete with social and psychological manifestations but also involves mental representations unique to brain anatomy and physiology, and the sophisticated human nervous system. To move effectively and produce models of the world, the motor-sensory system is engaged with the seven senses, especially the visual sense and the two "hidden" vestibular and kinesthetic/proprioceptive senses. Orientation therefore relates to embodied conceptual knowledge, its memory foundations, and organizing principles and processes. Thus, orientation is synonymous with concepts that range from navigational relations like direction, path, route, track, course, travel, wayfaring, bearing, and point of reference to the more tangible synonym of compass reading (Baird et al. 2006).

Processing of spatial, temporal, and social relations relies on mental cognitive maps, on which the behaving self is oriented relative to different places, events, and people. High-resolution

functional MRI scanning reveal that mental orientation in space, time, and person produces a sequential posterior–anterior pattern of adjacent and partially overlapping activity with the brain's default-mode network, a system involved in self-referential processing (Peera et al. (2015).

Cross-References

► Orientation (Left-Right)

References and Readings

Baird, A., Dewar, B. K., Critchley, H., Dolan, R., Shallice, T., & Ciplotti, L. (2006). The role of the anterior cingulate cortex in social and emotional functions. *Cognitive Neuropsychiatry, 11*(4), 369–388.

Peera, M., Salomonc, R., Goldbergb, I., Blankec, O., & Arzya, S. (2015). Brain system for mental orientation in space, time, and person. *Psychology and Cognitive Sciences, 112*(35), 11072–11077. https://doi.org/10.1073/pnas.1504242112.

Orientation (Left-Right)

Eleazar Eusebio[1] and Doris S. Mok[2]
[1]School Psychology, The Chicago School of Professional Psychology, Chicago, IL, USA
[2]Department of Psychology, Faculty of Social Sciences and Humanities University of Macau, Taipa, Macau SAR, China

Definition

Orientation is also known as directional (right-left) orientation (Lezak et al. 2012). Examination of right-left orientation involves directions in neuropsychological assessment and is usually accomplished through simple verbal commands involving the pointing of body parts (e.g., "place your right hand on your left knee" or "touch your left cheek with your left thumb"). Regular functioning adults should make no mistakes on right-left discrimination on their own and others' body parts; errors usually indicate impaired right-left orientation, standardized format such as the Right-Left Orientation Test (RLOT) (Benton et al. 1994). http://www4.parinc.com/Products/Product.aspx?ProductID=RIGHTLEFT), and similar tests are often included in neuropsychological test batteries to detect suspected problems. Patterns of errors are usually noted to identify specific deficits.

Cross-References

► Orientation

References and Readings

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

Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (2012). *Neuropsychological assessment* (5th ed.). New York: Oxford University Press.

Orientation Log

Alicia Nuñez and Daniel N. Allen
Department of Psychology, University of Nevada, Las Vegas, NV, USA

Synonyms

O-Log

Description

The Orientation Log (O-Log) was designed to assess orientation to time, place, and situation in persons who have posttraumatic amnesia as a result of traumatic brain injury. Posttraumatic amnesia is a state of disorientation and confusion that commonly occurs following traumatic brain injury and is related to a number of important short- and long-term outcomes. The O-Log consists of ten equally weighted items that can receive full or partial credit, with total scores ranging from 0 to 30. The O-Log

was developed for the daily monitoring of cognitive abilities and was designed to be particularly useful for inpatients who have sustained severe injuries. For example, while spontaneous correct responses receive full credit (three points), partial credit may be obtained if a correct response is given after the examiner provides a logical cue (two points) or if the correct answer is recognized when presented in a multiple choice format (one point). Thus, the response format of the O-Log minimizes the need for intact verbal and motor abilities, making it appropriate for patients who have language or motor disturbances. A modified-orientation log (MO-Log) was designed by Salam et al. (2012) to assess hepatic encephalopathy. The MO-Log is comprised of eight items: five on time (year, month, day, date, and clock time) and three on place (city, type of place, and name of hospital). It follows the same administration procedures and scoring rules as the O-Log (see above), with total scores ranging from 0 to 24.

Current Knowledge

The O-Log has excellent reliability, with high interrater reliability and internal consistency. With regard to validity, O-Log scores exhibit high correlations with other measures of orientation, such as the Mini-Mental State Examination and the Galveston Orientation and Amnesia Test (GOAT), and the O-Log is equally effective as the GOAT in determining the duration of posttraumatic amnesia. The O-Log may also provide better prediction of rehabilitation outcomes when compared with similar measures such as the GOAT. Finally, O-Log scores are significantly correlated with neuropsychological measures of verbal learning and memory, attention, psychomotor speed, mental flexibility, and intelligence.

Likewise, the MO-Log has good concurrent validity, discriminant validity, and inter-rater reliability in patients hospitalized with hepatic encephalopathy. The MO-Log has been found to be a valid tool for differentiating cirrhotic patient with and without hepatic encephalopathy and for predicting patient outcomes (mortality and time to reach normal mentation) in hospital settings.

Cross-References

- ▶ Galveston Orientation and Amnesia Test
- ▶ Modified Mini-Mental State Examination
- ▶ Post-Traumatic Confusional State
- ▶ Traumatic Brain Injury (TBI)

References and Readings

Frey, K. L., Rojas, D. C., Anderson, C. A., & Arciniegas, D. B. (2007). Comparison of the O-Log and GOAT as measures of posttraumatic amnesia. *Brain Injury, 21*, 513–520.

Jackson, W. T., Novack, T. A., & Dowler, R. N. (1998). Effective serial measurement of cognitive orientation in rehabilitation: The Orientation Log. *Archives of Physical Medicine and Rehabilitation, 79*, 718–720.

Novack, T. A., Dowler, R. N., Bush, B. A., Glen, T., & Schneider, J. J. (2000). Validity of the Orientation Log, relative to the Galveston Orientation and Amnesia Test. *Journal of Head Trauma Rehabilitation, 15*, 957–961.

Penna, S., & Novack, T. A. (2007). Further validation of the Orientation and Cognitive Logs: Their relationship to the Mini-Mental State Examination. *Archives of Physical Medicine and Rehabilitation, 88*, 1360–1361.

Salam, M., Matherly, S., Farooq, I. S., Stravitz, R. T., Sterling, R. K., Sanyal, A. J., ... & Fuchs, M. (2012). Modified-orientation log to assess hepatic encephalopathy. *Alimentary Pharmacology & Therapeutics*, *35*(8), 913–920.

Orienting Response

Anna MacKay-Brandt
Nathan S. Kline Institute for Psychiatric Research, Orangeburg, NY, USA
Taub Institute for Research on Alzheimer's Disease and the Aging Brain, Columbia University, New York, NY, USA

Synonyms

Orienting reflex; OR

Definition

The reflexive response to an environmental stimulus such that there is a transient orientation of the individual to the stimulus.

Historical Background

This phenomenon was first described by Sechenov in the 1850s and then developed by Pavlov as a critical element in conditioning theory. Pavlov referred to this response as the "what is it" reflex and identified it as necessary to the development of a conditioned response. The key criteria were that the stimulus was not so strong as to elicit an unconditioned response without conditioning but also that the stimulus was strong enough to result in observable sensory awareness. A stimulus that fits into this circumscribed middle ground was considered to elicit an orienting response. Further, orienting responses were observed to decline in strength over time with repeated exposure to the stimuli, without reinforcement. This change in response over time is called habituation (Sokolov 1960).

Current Knowledge

The adaptive response of orienting to a stimulus that in some way differs from the background is present from birth and allows for a quick reaction to a changing environment. This response is generally accepted to be a basic element of attention that has allowed for the study of the fundamental ability to capture and release attention and serve as a gateway for information to enter the focus of attention (e.g., Gati and Ben-Shakhar 1990). Features that distinguish a stimulus change from background must be attended to some degree, but the range of distinctions can range from perceptual to semantic.

Several neural mechanisms to explain the orienting response and habituation of the response have been proposed. Sokolov (1976; 1990) highlights the role of the hippocampus in novelty detection and thus serving to inhibit the role of the reticular activating system. While Groves and Thompson (1970) proposed a neural model of competition between habituation and sensitization that is attractive for its simplicity and ability to be applied to neural systems of differing degrees of complexity (e.g., habituation in the *Aplysia*, see Kandel 1976), models aimed to account for OR and habituation in humans have elaborated on the dual process theory of Groves and Thompson to include multiple neural interactions in the limbic-hypothalamic-frontal system (Waters and Wright 1979). Cognitive models of this phenomenon have implicated short-term memory and comparisons of information held in short-term and long-term memory to explain the phenomena of OR and habituation. Due to the complex neural systems involved in these cognitive models, they are difficult to specify (Wagner 1981; Ohman 1983).

The orienting network is one of the three distinct attentional networks proposed by Posner and colleagues (Fan et al. 2002) with the goal of advancing psychological science by providing a foundational framework with dissociable behaviors and neural circuitry. These purportedly independent networks carry out stimulus recognition and response initiation by maintaining a state of alertness (*alerting*), orienting to specific-sensory stimuli (*orienting*), and resolving conflict (*executive attention*). A task was introduced by the authors to tap these aspects of attention (see The Attention Network Task, Fan et al. 2002). This task was made widely available and has been used across many studies of attention in healthy and clinical populations (see Posner and Rothbart 2007, for a review). MacLeod et al. (2010) provide an analysis of the psychometric properties of this task and some useful guidance for future research.

References and Readings

Fan, J., McCandliss, B. D., Sommer, T., Raz, A., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience, 14*, 340–347.

Gati, I., & Ben-Shakhar, G. (1990). Novelty and relevance in orientation and habituation: A feature-matching

approach. *Journal of Experimental Psychology: General, 119*, 251–263.
Groves, P. M., & Thompson, R. F. (1970). Habituation: A dual-process theory. *Psychological Review, 77*(5), 419–450.
Kandel, E. R. (1976). *Cellular basis of behaviour*. San Francisco: Freeman.
MacLeod, J. W., McConnell, M. M., Lawrence, M. A., Eskes, G. A., Klein, R. M., & Shore, D. I. (2010). Appraising the ANT: Psychometric and theoretical considerations of the Attention Network Test. *Neuropsychology, 24*(5), 637–651.
Ohman, A. (1983). The orienting response during Pavlovian conditioning. In D. Siddle (Ed.), *Orienting and habituation: Perspectives in human research* (pp. 315–370). New York: Wiley.
Pavlov, I. P. (1935). *Physiology and pathology of the higher nervous functions*. Leningrad: Academy of Sciences.
Posner, M. I., & Rothbart, M. K. (2007). Research on attention networks as a model for the integration of psychological science. *Annual Review of Psychology, 58*, 1–23.
Sokolov, E. N. (1960). Neuronal models and the orienting reflex. In M. A. B. Brazier (Ed.), *The central nervous system and behaviour, 3rd conference* (pp. 187–286). New York: Josiah Macy, Jr. Foundation.
Sokolov, E. N. (1990). The orienting response and future directions of its development. *Integrative Psychological and Behavioral Science, 25*(3), 142–150.
Wagner, A. R. (1981). SDP: A model of automatic memory processing in animal behaviour. In N. E. Spear & R. R. Miller (Eds.), *Information processing in animals: Memory mechanisms*. New York: Psychology Press.
Waters, W. F., & Wright, D. C. (1979). Maintenance and habituation of the phasic orienting response to competing stimuli in selective attention. In H. D. Kimmel, E. H. Van Olst, & J. F. Orlebeke (Eds.), *The orienting in humans* (pp. 101–121). Hillsdale: Erlbaum.

Orthostatic Tremor

Anna DePold Hohler[1] and
Marcus Ponce de Leon[2]
[1]Boston University Medical Center, Boston, MA, USA
[2]Madigan Army Medical Center, Tacoma, WA, USA

Synonyms

Standing tremor

Definition

Orthostatic tremor is considered to be a variant of essential tremor. This type of tremor occurs in the legs immediately on standing and is relieved by sitting down. It is usually of high frequency, and no other clinical signs and symptoms are present.

Cross-References

▶ Tremor

References and Readings

Jankovic, J. (2007). Tremor and Myorhythmia. In S. Fahn & J. Jankovic (Eds.), *Principles and practice of movement disorders* (pp. 451–479). Philadelphia: Churchill Livingstone Elsevier.

Other Health Impairment

Erica McConnell
Jefferson County Public Schools, School Psychologist, Golden, CO, USA

Definition

According to the Federal Office of Special Education and Rehabilitative Services, other health impairment (Sec. 300.8(c)(9)) is a term used to describe acute or chronic health conditions, including but not limited to heart conditions, tuberculosis, rheumatic fever, nephritis, asthma, sickle-cell anemia, hemophilia, epilepsy, lead poisoning, leukemia, diabetes, attention deficit hyperactivity disorder, heightened alertness to environmental stimuli, and Tourette syndrome, which adversely affects a child's educational performance (http://idea.ed.gov/explore/search; http://nichcy.org/Disabilities/Categories). The Individuals with Disabilities Education Act (IDEA) is the primary federal program that authorizes state and local aid for special education and

related services for children with disabilities (http://idea.ed.gov/). In order to qualify for services under the other health impairment category within IDEA, the child must be diagnosed with an acute or chronic health condition, and that condition must cause limited strength, vitality, or alertness or heightened alertness to the educational environment. Furthermore, the limited alertness must affect the child's educational performance and create a need for special education services (Grice 2002).

Categorization

The categories of disabilities under IDEA law include Autism, Deaf-Blindness, Deafness, Developmental Delay, Emotional Disturbance, Hearing Impairment, Intellectual Disability, Multiple Disabilities, Orthopedic Impairment, Other Health Impairment, Specific Learning Disability, Speech or Language Impairment, Traumatic Brain Injury, and Visual Impairment Including Blindness. Special education services for any of the conditions may vary depending on the needs of the child. However, to receive special education services under the Other Health Impairment category, it must be demonstrated that a child's educational performance is adversely affected by the condition and that the child's condition does not fit into any of the other categories listed above (McIntosh and Decker 2005).

Epidemiology

The prevalence of chronic health conditions in childhood ranges based on the conditions and has been estimated as high as 44% of children nationwide meeting some criteria of having a chronic health condition (van der Lee et al. 2007). For example, the prevalence for ADHD among children aged 4–17 is estimated to between 5.6% for girls and 13.3% for boys (Pastor et al. 2015), whereas the prevalence for asthma is estimated between 5.6% for ages 0–4 and 17.1% for ages 12–17 (Bloom and Freeman 2015). A National Survey of Children's Health in the United States was completed in 2009–2010 and estimated that approximately 2.6 million out of 74 million children were identified with a special health care need, chronic condition, and/or functional limitations (www.childhealthdata.org).

Neuropsychology and Psychology of Other Health Impairments

The neuropsychological and psychological implications vary based on the individual and impairment. For example, a child identified with attention deficit hyperactivity disorder may be referred for executive dysfunction, such as short attention span, lack of planning, organization, inhibition, or set-shifting (ADHD or executive functioning for more information). The neuropsychological implications associated with diabetes may include significant cognitive dysfunction, impaired sensory input, eye-hand coordination difficulties, attention, and visual-spatial impairment (Donnelly 2005). It is important that physicians and school psychologists work closely to identify the potential neuropsychological and psychological impact of the acute or chronic condition so that appropriate interventions can be provided and the child's educational needs are met.

Evaluation

A thorough evaluation of any condition involves reviewing the student's file to obtain a developmental, medical, and educational history; knowing about and understanding the medical or medication regimen of the health impairment identified; consulting with the child's physician treating the condition; conducting formal and informal observations of the child in various environments such as in the classroom, on the playground, or in the child's home when possible; obtaining social, emotional, cognitive, achievement, and adaptive functioning data; and administering a battery of neuropsychological tests that covers all aspects of brain-behavior relationships pertinent to the referral question and hypotheses, including but not limited to tests that address

visual-spatial processing, sensorimotor functioning, memory and learning, and attention (Fletcher-Janzen 2005).

Treatment

Treatment of the other health impairment will vary depending on the condition and may include but is not limited to medications as prescribed by their treating physician, physical and educational accommodations and modifications, and other specialized psychoeducational programs needed to meet the child's physical, emotional, and educational needs.

Cross-References

- ▶ Attention Deficit Hyperactivity Disorder
- ▶ Diabetes Mellitus
- ▶ Epilepsy
- ▶ Executive Functioning
- ▶ Lead Exposure
- ▶ Seizure
- ▶ Sickle-Cell Disease
- ▶ Tourette Syndrome

References and Readings

Bloom, B., & Freeman, G. (2015). Tables of summary health statistics for U.S. children: 2014. *National Health Interview Survey*. Retrieved June 20, 2016, from http://www.cdc.gov/nchs/nhis/SHS/tables.htm

Categories of disability under IDEA Law. (n.d.). *The national dissemination center for children with disabilities*. Retrieved July 5, 2009, from http://nichcy.org/Disabilities/Categories

Donnelly, J. P. (2005). Providing neuropsychological services to learners with chronic illnesses. In R. C. D'Amato, E. Fletcher-Janzen, & C. R. Reynolds (Eds.), *Handbook of school neuropsychology* (pp. 511–532). Hoboken: Wiley.

Fletcher-Janzen, E. (2005). The school neuropsychological examination. In R. C. D'Amato, E. Fletcher-Janzen, & C. R. Reynolds (Eds.), *Handbook of school neuropsychology* (pp. 172–212). Hoboken: Wiley.

Grice, K. (2002). Eligibility under IDEA for other health impaired children. *School Law Bulletin, 33*, 7–12.

McIntosh, D. E., & Decker, S. L. (2005). Understanding and evaluating special education, IDEA, ADA, NCLB, and section 504 in school neuropsychology. In R. C. D'Amato, E. Fletcher-Janzen, & C. R. Reynolds (Eds.), *Handbook of school neuropsychology* (pp. 365–382). Hoboken: Wiley.

National Survey of Children with Special Health Care Needs. NS-CSHCN. (2009/10). *Data query from the child and adolescent health measurement initiative, data resource center for children and adolescent health website*. Retrieved June 20, 2016, from www.childhealthdata.org

Other health impairment (Sec. 300.8(c)(9)). (n.d.). *Building the legacy: IDEA 2004*. Retrieved June 20, 2016, from http://idea.ed.gov

Pastor, P. N., Reuben, C. A., Duran, C. R., & Hawkins, L. D. (2015). *Association between diagnosed ADHD and selected characteristics among children aged 4–17 years: United States, 2011–2013*, *NCHS data brief, no 201*. Hyattsville: National Center for Health Statistics.

van der Lee, J. H., Mokkink, L. B., Grootenhuis, M. A., Heymans, H. S., & Offringa, M. (2007). Definitions and measurement of chronic health conditions in childhood. *JAMA, 297*(24), 2741–2751.

Otis-Lennon School Ability Test

Yuan Yuan Wang[1] and Kathryn K. Reva[2]
[1]Faculty of Health Sciences, University of Macau, Macao, China
[2]University of Northern Colorado, New York, NY, USA

Synonyms

OLSAT 7

Definition

The Otis-Lennon School Ability Test Seven (OLSAT 7) is designed around the belief that students must have specific skills and abilities prior to learning new information. These prerequisite skills include accurate perceptions, the ability to recognize and recall what has been perceived, logically thinking, understanding relationships, the ability to abstract from a set of particulars, and knowing how to apply

generalizations to new and different contexts. These skills and abilities serve as the basis of the OLSAT 7 and are measured through student performance on numerous tasks.

In order to address the broad range of ability levels found in students in kindergarten through 12th grade students, the OLSAT 7 contains seven levels as designated by alphabetical letters. For example, Level A is designed for students in kindergarten and Level B is for first grade students. It also happens that Levels A and B are the only dictated versions of the OLSAT to address the limited reading abilities of most students in these grades. Level C is designated for students in second grade whereas Level D is for third grade students. Level C is partially self-administered and partially dictated or paced by the teacher. Level D through G are entirely self-administered with Level E serving fourth and fifth grade students, Level F designated for students in sixth, seventh, and eighth grade, and Level G for students in grades 9–12. The basic structure of the OLSAT 7 is designed to provide an accurate and efficient measure of student's ability. Regardless of the level being administered, the OLSAT 7 can be administered in a group or individually. Approximately 1 h should be set aside for administration with less time needed for the lower grade levels.

Each of the seven OLSAT 7 levels contains both a verbal cluster and a nonverbal cluster as determined by whether or not knowledge of language is required for students to be successful in answering the items correctly. If this is true, essentially students who do not know English could still be successful in answering the nonverbal items so long as they understand the directions given. Upon completion of the OLSAT 7, students receive a total score, a verbal score, and a nonverbal score. Since student's ability to achieve success in the school setting is dependent upon adequate nonverbal and verbal abilities, the total score is endorsed as the best indicator of school learning ability. The verbal and nonverbal scores can give important information on the respective domains and possible discrepancies in a student's skill set.

Although the verbal and nonverbal domains are the basic structure of all seven levels of the OLSAT 7, the types of questions and subtests will vary according to developmental abilities of the students at different grades. The verbal cluster contains questions focusing on two different skill sets: verbal comprehension and verbal reasoning. Verbal comprehension, a measure of the OLSAT 7, examines the student's ability to perceive the relational aspects of words and combinations, identify word meanings, find differences in similar words or phrases, and manipulate words to create meaning and are focused on identifying the student's understanding of the nature of language. The multitude of verbal comprehension skills is examined with questions focusing on following directions, antonyms, sentence completion, and sentence arrangement. Antonym questions, found only on Level A-C, measure the student's ability to recognize opposite meaning of words. Sentence completion items examine the student's ability to understand relationships among words and sentence structure to supply a missing word(s) needed to create a meaningful sentence. The student's ability to group seemingly random words to create a meaningful sentence is assessed in the sentence arrangement section.

The second overarching group of questions found on the verbal domain of the OLSAT 7 is titled verbal reasoning. The skills measured here include the student's ability to infer relationships between words, apply inferences to novel situations, evaluate conditions requiring necessary versus optional, and perceive similarities and differences. These items are centered on assessing the complex though processes of the student require higher level cognitive thinking. The types of questions used to assess verbal reasoning abilities include aural reasoning, arithmetic reasoning, logical selection, word/letter matrix, verbal analogies, verbal classification, and inference. Aural reasoning questions, found only on the orally administered levels (Level A-C), assess the student's ability to apply reasoning to information presented orally. Arithmetic reasoning questions are found on all seven levels of the OLSAT 7 and examine the ability to solve verbal problems that require numerical reasoning. The logical selection items focus on the student's ability to apply simple logic to everyday situations or

events. The capacity to provide a missing element in a word or letter matrix, which requires inferences among relationships of words and letters, is measured with word/letter matrix items. Verbal analogy items focus on the ability of the student to infer the relationship between two words and apply that relationship to another group of words or letters, whereas verbal classification items require the student to identify a principle operating within a set of words and determine which word does not belong. Lastly, inference items focus on assessing evaluation of various conditions to determine necessary versus optional in a given set of circumstances.

Similar to the verbal domain structure, the nonverbal types of questions and subtest vary according to the developmental abilities of students at different grade levels. For example, OLSAT Levels A through C contain nonverbal questions with pictures focusing on reasoning, classification, analogies, and series. By presenting the information in pictorial format, the emphasis on number and numeric abilities is eliminated. Figural reasoning, an overarching category of questions found on both the early levels of the OLSAT and the higher levels, assesses inferring relationships with geometric figures, perceiving patterns or progressions, and identifying how to continue the pattern, making generalizations among figures and making spatial manipulations. A student's ability to reasoning with geometric figures provides important information regarding his or her nonverbal ability. Figural analogies are used to assess the ability to infer relationships between geometric shapes whereas the ability to identify missing elements in a matrix composed of geometric elements is assessed with pattern matrix items. Lastly, figural series items examine the ability to identify a rule to a pattern and predict which figure would come next based on the established rule. The second category of nonverbal questions is found in quantitative reasoning, which focuses on the student's ability to use numbers to infer relationships, identify computational rules, and make predictions. These skills are considered to be an important nonverbal contributor to school achievement. Number series questions focus on the identification of numbers or letters that should occur in a specific place in a series and numeric inference items examine the student's ability to determine how a set of numbers is related and identify associated numbers. Lastly, number matrix items require the student to supply missing numbers in a number matrix.

Historical Background

The Otis-Lennon School Ability Test is currently in its seventh edition and dates back to 1918 when the origin test series was created. The focus of this measure is on abstract thinking and reasoning ability of students from kindergarten through Grade 12. Appropriate utilization of this measure include (1) identifying an individual's ability to cope with specific learning tasks specific to the school setting, (2) suggesting a child's specific placement for school learning functions, and (3) evaluating an individual's level of achievement relative to his or her specific talents.

Tests

The Otis test series can be tracked back to 1918 (Grotelueschen 1969). Otis was one of the leading figures in intelligent testing, who first created Otis Group Intelligence Scale after his name. After that, Otis's IQ tests developed into various versions and the names changed over the years, becoming the Otis-Lennon Mental Ability Test (Otis and Lennon 1967), and the Otis-Lennon School Ability Tests was the most recent iteration (Sacks 2007).

The Otis-Lennon Mental Ability Test is the fourth major edition of Otis test series (Grotelueschen 1969). The Otis-Lennon Mental Ability Test was the precursor of the Otis-Lennon Mental Ability Test. The Otis-Lennon Mental Ability Test measured mental ability in six levels: the Otis-Lennon Primary I, Primary II, Elementary I, Elementary II, Intermediate level, and the Advanced level. The six different levels of Otis-Lennon Mental Ability Test were designed to

replace previous Alpha, Beta, and Gamma Tests of the Quick-Scoring series (Groteluesche n 1969).

The Otis-Lennon School Ability Test has been updated since the release of the first version (i.e., Otis and Lennon 1977, 1979, 1982, 1988, 1996, 2003). The Otis-Lennon School Ability Test is currently in its eighth edition (OLSAT 8; Otis and Lennon 2003) and the ninth edition will be released soon. OLSAT 8 can be administrated online, by paper and pencil, and by group administration. The completion time of OLSAT 8 varies by level, and the maximum time is 75 min. The scoring can be calculated by machine scoring online (e.g., via Pearson Scoring and Reporting Services at www.perasonassessments.com), or hand scoring.

Cross-References

- ▶ Academic Skills
- ▶ Educational Testing
- ▶ Reading

References and Readings

Groteluesche n, A. (1969). Review work: Otis-Lennon mental ability test by Arthur S. Otis, Roger T. Lennon. *Journal of Educational Measurement, 6*(2), 111–113.

Otis, A. S., & Lennon, R. T. (1967). *Otis-Lennon mental ability test*. New York: Harcourt Brace Jovanovich Incorporated.

Otis, A. S., & Lennon, R. T. (1977). *Otis-Lennon School Ability Test (intermediate level 1, form R)*. New York: Harcourt Brace Jovanovich.

Otis, A. S., & Lennon, R. T. (1979). *Otis-Lennon School Ability Test manual for administering and interpreting*. New York: The Psychological Corporation.

Otis, A. S., & Lennon, R. T. (1982). *Otis-Lennon School Ability Test*. New York: Harcourt Brace Jovanovich.

Otis, A. S., & Lennon, R. T. (1988). *Otis-Lennon School Ability Test* (6th ed.). San Antonio: Harcourt Brace.

Otis, A. S., & Lennon, R. T. (1996). *Otis-Lennon School Ability Test* (7th ed.). San Antonio: Harcourt Brace. OLSAT 7).

Otis, A. S., & Lennon, R. T. (2003). *Otis-Lennon School Ability Test* (8th ed.). San Antonio: Harcourt Brace Educational Measurement.

Sacks, P. (2007). *Tearing down the gates: Confronting the class divide in American education*. Berkeley: University of California Press.

Outcome, Outcome Measurement

James F. Malec
Department of Physical Medicine and Rehabilitation, Indiana University School of Medicine and the Rehabilitation Hospital of Indiana, Indianapolis, IN, USA

Synonyms

Participation; Program evaluation

Definition

Outcome measurement describes the tools and processes used to evaluate and record key elements representing natural recovery or the effects of an intervention on an illness or injury.

Historical Background

Outcome measurement to evaluate the effectiveness of medical, surgical, and rehabilitative practices for traumatic injuries and other disabling conditions has been an integral part of development of these practices. The most widely used outcome scales have focused on the evaluation of abilities and activities that are relevant to reintegration in personal and community life.

Current Knowledge

The Functional Independence Measure (FIM™) instrument has become the standard for the evaluation of inpatient rehabilitation following traumatic injury, stroke, and other disabling conditions. Using FIM, the staff who provide inpatient rehabilitation services rate the patient's functional status in a number of key areas that include physical (e.g., locomotion), cognitive (e.g., memory), and functional (e.g., dressing) abilities and activities.

The psychometric properties of the FIM have been well established through years of research. Database and reporting services (eRehabData, https://web2.erehabdata.com/erehabdata/index.jsp; Uniform Data System for Medical Rehabilitation, http://www.udsmr.org/) are available through which providers who use the FIM can compare outcomes of their inpatient rehabilitation programs with those of the other providers.

A metrically sound standard for measuring the outcomes of postacute brain injury rehabilitation services is not currently established. The FIM shows unacceptable ceiling effects when patients with brain injury are measured at 1 year or more post-injury. The Glasgow Outcome Scale (GOS) and the Glasgow Outcome Scale-Extended(GOS-E) have been extensively used in outcomes research. However, these scales also show unacceptable ceiling effects in the postacute period. Although the GOS and GOS-E have considerable face validity, the metric structure of the scale is limited. The Glasgow scales are essentially one-item scales in which the evaluator selects a single level that best characterizes the outcome. This one-item rating process obviously limits the precision of the measurement tool.

The Disability Rating Scale (DRS) has also been widely used in brain injury outcome studies. The DRS, however, also shows unacceptable ceiling effects when used postacutely and is also metrically flawed. In rating patients postacutely after BI, most of the variance in the DRS is accounted for by two items reflecting levels of independent living and employment. The rating on the employment item is subjective, that is, the rater is asked to assess at what level the patient is capable of functioning rather than at what level they actually function, introducing the potential for measurement error. Recent study within the TBI Model Systems has developed an interview form for the DRS that has stronger psychometric properties.

The Craig Handicap Assessment and Report Technique (CHART) is a measure of community participation, or reintegration, that was originally developed for use with individuals with spinal cord injury. The CHART has been applied to other populations including those with brain injury.

Modern measurement theory and techniques have led to the development of more sophisticated measurement approaches. Item response theory (IRT) and the Rasch model provide methods for developing items that represent key unidimensional aspects of the full range of outcome as well as associated rating scales that support precision in measurement. Identifying key items that are measured with precision provides a scale with a metric structure which often requires only a small set of items. Short scales enhance usability by busy clinicians. Through analytic methods based on IRT, a set of items is identified that reliably represents the underlying construct of interest. In an aggregate, these items form the basis for measurement. Specific items also represent specific levels of the construct. Identification of the ordinal relationship among items of a scale forms the basis for the development of item administration algorithms that allow for a precise measurement of outcome with very few items. That is, because the ordinal relationship among items is known, a given individual's level on the scale can be quickly identified by administering a few items in a manner that bracket the individual's level. Application of such item administration algorithms has been termed as computer-adaptive testing (CAT). The development of the FIM exemplifies scale construction using IRT. The family of SF-36 measures of quality of life signifies seminal work in the use of IRT and CAT.

Using modern metric theory and techniques, the Mayo-Portland Adaptability Inventory, now in its fourth revision (MPAI-4), was developed as a measure for the evaluation of patients and outcomes after brain injury. The MPAI-4 includes three subscales that reflect ability, adjustment, and participation. The eight-item participation subscale of the MPAI-4 provides a brief measure for the assessment of long-term outcome that has minimal floor and ceiling effects. The MPAI-4 can be completed by medical and rehabilitation staff, patients, and their significant others to capture the perspectives of each group.

Goal achievement is another traditional measure of the effectiveness of outpatient rehabilitation interventions. Goal attainment scaling

provides a method for quantifying goal achievement for clinical or research purposes.

Future Directions

In 2009, the National Institutes of Health (NIH) in collaboration with the Veterans Administration and the National Institute for Disability and Rehabilitation Research (NIDRR) convened a consensus conference to recommend measures for use in traumatic brain injury research. While the most appropriate measures to use in a given study continues to be debated, the goal of greater consistency in the use of outcome measures across studies to facilitate comparison of results is widely endorsed by clinical researchers and funding agencies.

Another important initiative is the Patient-Reported Outcomes Measurement Information Systems (PROMIS) which has resulted in the development of self-report measures in a number of health and quality of life domains using IRT. Measures within PROMIS domains have also been further refined for specific populations, such as those with neurological disorders (Neuro-QoL), spinal cord injury (SCI-QoL), and traumatic brain injury (TBI-QoL). PROMIS and associated measures can be administered using CAT.

GAS uses a different approach than standardized measures. Krasny-Pacini and colleagues have proposed criteria for constructing and evaluating the quality of GAS measures.

Websites have also been developed that detail outcome measures including their psychometric properties:

- The website for the Center for Outcome Measurement in Brain Injury (COMBI; http://www.tbims.org/combi) offers an array of more specific outcome measures that may be of value to providers and researchers in assessing specific interventions and aspects of outcome.
- The NIDRR-funded Rehabilitation Research and Training Center (RRTC) on measuring rehabilitation outcomes and effectiveness based at the Rehabilitation Institute of Chicago (http://www.ric.org/research/centers/cror/projects/rrtc/index.aspx). The purpose of this project is to develop comprehensive measures of participation following brain injury and other disabling conditions. Most available outcome measures focus on aspects of outcome that are of cultural value, e.g., independence in living and work. However, individuals with brain injury may attach varying value and satisfaction to these activities. For instance, participation in community-wide social activities can be an indicator of a successful outcome after a brain injury. However, such activities may be of limited interest and value to a highly introverted individual who may derive more satisfaction from activities such as reading or dyadic interpersonal interactions. The work of this RRTC led by Dr. A. W. Heinemann has a goal of developing measures that capture both objective indicators of outcome (e.g., return to work, independent living) and outcomes valued by the individual with a disability. This focus on subjective as well as more objective assessment of outcome was introduced by Drs. Margaret Brown and Marcel Dijkers at Mt. Sinai, New York, in their development of the Participation Objective-Participation Subjective (POPS) scale.

Cross-References

- ▶ Craig Handicap Assessment and Reporting Technique
- ▶ Functional Independence Measure
- ▶ Functional Independence Measure for Children
- ▶ Glasgow Outcome Scale
- ▶ Glasgow Outcome Scale: Extended
- ▶ Mayo-Portland Adaptability Inventory
- ▶ Participation Objective, Participation Subjective

References and Readings

Bond, T. G., & Fox, C. M. (2007). *Applying the Rasch model. Fundamental measurement in the human sciences* (2nd ed.). Mahwah: Lawrence Erlbaum.

Heinemann, A. (Ed.). (2007). State-of-the-science on post-acute rehabilitation: Setting a research agenda and developing an evidence base for practice and public policy. *Archives of Physical Medicine and Rehabilitation, 88*, 1478–1481.

Krasny-Pacini, A., Evans, J., Sohlberg, M. M., & Chevingnard, M. (2016). Purposed criteria for appraising goal attainment scales used as outcome measures in rehabilitation research. *Archives of Physical Medicine and Rehabilitation, 97*, 157–170.

Tate, R. L. (2010). *A compendium of tests, scales, and questionnaires: The practitioner's guide to measuring outcomes for acquired brain impairment*. New York: Psychology Press.

Wilde, E. A., Whiteneck, G. G., Bogner, J., Bushnik, T., Cifu, D. X., Dikmen, S., French, L., Giacino, J. T., Hart, T., Malec, J. F., Millis, S. R., Novack, T. A., Sherer, M., Tulsky, D. S., Vanderploeg, R. D., & von Steinbuechel, N. (2010). Recommendations for the use of common outcome measures in traumatic brain injury research. *Archives of Physical Medicine and Rehabilitation, 91*, 1650–1660.

Web Sites

Center for Outcome Measurement in Brain Injury (COMBI). www.tbims.org/combi

Patient Reported Outcome Measurement Information System (PROMIS). http://www.nihpromis.org/

eRehabData. https://web2.erehabdata.com/erehabdata/index.jsp

Rehabilitation Research and Training Center (RRTC) on Measuring Rehabilitation Outcomes and Effectiveness. http://www.ric.org/research/centers/cror/projects/rrtc/index.aspx

SF tools, Computerized Adaptive Testing. www.SF36.org

Uniform Data System for Medical Rehabilitation. http://www.udsmr.org/

Oxazepam

John C. Courtney[1], Cristy Akins[2] and Efrain Antonio Gonzalez[3,4]

[1]Socorro Mental Health, Presbyterian Medical Services, Socorro, NM, USA

[2]Mercy Family Center, Metarie, LA, USA

[3]College of Psychology, Nova Southeastern University, Fort Lauderdale, FL, USA

[4]Utah State University, Logan, UT, USA

Generic Name

Oxazepam

Brand Name

Serax

Class

Anxiolytics, benzodiazepines, and antianxiety agents

Proposed Mechanism(s) of Action

Increases inhibitory effects of GABA by binding to benzodiazepine receptors on GABA-A ligand-gated chloride channel complexes

Indication

Anxiety, anxiety with depression, and alcohol withdrawal

Off-Label Use

No common off-label use

Side Effects

Serious

Respiratory depression, liver dysfunction, kidney dysfunction, and blood dyscrasias

Common

Fatigue, depression, ataxia, slurred speech, dizziness, confusion, nervousness, hallucinations, hypotension, and dry mouth

References and Readings

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

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

Additional Information

Drug Interaction Effects. http://www.drugs.com/drug_interactions.html

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

Oxcarbazepine

John C. Courtney[1] and Efrain Antonio Gonzalez[2,3]
[1]Socorro Mental Health, Presbyterian Medical Services, Socorro, NM, USA
[2]College of Psychology, Nova Southeastern University, Fort Lauderdale, FL, USA
[3]Utah State University, Logan, UT, USA

Generic Name

Oxcarbazepine

Brand Name

Trileptal and Oxtellar XR

Class

Anticonvulsants

Proposed Mechanism(s) of Action

Use-dependent antagonist of voltage-sensitive sodium channels. It also appears to inhibit the release of glutamate.

Indication

Partial seizures in adults and children

Off-Label Use

Bipolar mood disorder, acute mania, diabetic neuropathy, and neuralgia/neuropathy

Side Effects

Serious

Hyponatremia (a metabolic condition in which there is not enough sodium in the body fluids outside the cells) and (rare) activation of suicidal ideation

Common

Sedation, dizziness, headache, ataxia, nystagmus, nausea, abdominal pain, vomiting, diplopia, vertigo, rash, abnormal gait, and fatigue

References and Readings

Physicians' desk reference (71st ed.) (2017). Montvale: Thomson PDR.
Stahl, S. M. (2007). *Essential psychopharmacology: The prescriber's guide* (2nd ed.). New York: Cambridge University Press.

Additional Information

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