E

Ear Preference

► Lateral Dominance

Early Onset Dementia

► Presenile Dementia

Early-Delayed Effects of Radiation

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Synonyms

Subacute radiotherapy effects

Definition

Therapeutic ionizing irradiation (XRT) affects the nervous system in different biological settings and with different mechanisms that manifest at different time points. The early-delayed phase of XRT, also termed subacute, is observed weeks to months after treatment is completed (measured 2–4 months post completion of XRT, but as early as 2 weeks), whether XRT is given alone or combined with chemotherapy. The cognitive effects are transient, and if reversal of symptoms does not occur within weeks to months after treatment is completed, a change in neoplastic process should be considered. In more severe cases, symptoms may be similar to those of the tumor, such as headache, lethargy, and worsening of lateralizing signs. Cognition is affected, but effects are thought to be mild and temporary (Rottenberg, 1991), and thus reverse again. L'hermitte's syndrome sometimes occurs following spinal XRT and is thought to be due to transitory demyelination of white matter (Dunbar & Loeffler, 1994). Changes may not be seen on MRI (Armstrong, Hunter, Hackney, Shabbout, Lustig, Goldstein, et al., 2005). In some cases, particularly with higher doses or whole brain doses, an increase in the size of the lesion and sometimes the occurrence of new contrast enhancement may be seen. Therefore, white matter changes that occur in the months after treatment may be difficult to differentiate from tumor growth.

Current Knowledge

Neural substrate and mechanisms: One explanation for the transient worsening of a patient's condition soon after completion of XRT is disruption of glial mitosis resulting in temporary demyelination. A loss of oligodendrocytes and abnormal and often multinucleated giant astrocytes have been found, and the latency in the onset and timing of the cellular resolution correspond to the cycle of myelin turnover (DeAngelis, Delattre, & Posner, 2001). The formation of necrotic tumor debris and other products of cell lysis that are not rapidly removed from the CNS may simulate surrounding edema causing capillary permeability defects or demyelination (Phillips, Delattre, Berger, & Rottenberg, 1987).

Changes in the hippocampal milieu have been observed in animals, including depression of neuroblast progenitors, and increase in inflammatory microglia (Monje, Mizumatsu, Fike, & Palmer, 2002; Monje & Palmer, 2003). In humans, changes in the hippocampus have been measured in a few studies. Children are more sensitive to XRT injury, and one study identified a decline in hippocampal volume over 2 years (Nagel, Palmer, Reddick, Glass, Helton, Wu, et al., 2004). In adults, a decline was found in hippocampal volume ipsilateral to tumor over 6 months following XRT; the contralateral hippocampus improved (Armstrong, Hampstead & Guglielmi, 2004). In children with leukemia who receive craniospinal XRT because of risk of CNS seeding of tumor cells, a somnolence syndrome may develop, characterized by headache, nausea, vomiting, or fever, but without focal neurological signs

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(DeAngelis et al., 2001). This is usually treated with steroids, and occurs much more frequently during the acute phase of treatment.

Cognitive effects: There is no general decline in cognitive function during the early-delayed period. From a comprehensive battery, a robust early-delayed group effect of XRT was limited to the decline and later rebound of a specific process of memory, that of retrieving semantic information from long-term storage (Armstrong, Corn, Ruffer, Pruitt, Mollman, & Phillips, 2000; Armstrong, Stern, Ruffer, & Corn, 2001; Llanes, Torres, Roeske, Mundt, Keedy, & Zakowski, 2004), possibly due to the diffuse effects of XRT and due to the sensitivity of hippocampal neural progenitor cells to irradiation. Other cognitive findings may occur but may be confounded by disease severity/grade, whole brain versus partial field XRT, and pre-existing white matter risk (Ahles, Silberfarb, Herndon, Maurer, Kornblith, Aisner, et al., 1998; Vigliani, Sichez, Poisson & Delattre, 1996). There is some variability in the severity of patients' responses, and individual host factors, such as the immune system, may mediate the cellular response of normal appearing brain tissue to XRT, thus accounting for the lack of association of cellular response and clinical tolerance (Corn, Yousem, Scott, Rotman, Asbell, Nelson, et al., 1994).

References and Readings

Ahles, T., Silberfarb, P., Herndon, J., Maurer, L., Kornblith, A., Aisner, J., et al. (1998). Psychological and neuropsychologic functioning of patients with limited small-cell lung cancer treated with chemotherapy and radiation therapy with or without warfarin: A study by the Cancer and Leukemia Group C. *Journal of Clinical Oncology, 16*, 1954–1960.

Armstrong, C., Corn, B., Ruffer, J., Pruitt, A., Mollman, J., & Phillips, P. (2000). Radiotherapeutic effects on brain function: Double dissociation of memory systems. *Neuropsychiatry Neuropsychol Behav Neurol, 13*, 101–111.

Armstrong, C., Stern, C., Ruffer, J., & Corn, B. (2001). Memory performance used to detect radiation effects on cognitive functioning. *Applied Neuropsychology, 8*(3), 129–139.

Armstrong, C. L., Hampstead, B., & Guglielmi, L. (2004). Hippocampal response to neuro-oncological stressors [abstract]. *Journal of the International Neuropsychological Society, 10*(S1), 65.

Armstrong, C. L., Hunter, J. V., Hackney, D., Shabbout, M., Lustig, R., Goldstein, B., et al. (2005). MRI changes during the early-delayed phase of radiotherapy effects. *International Journal of Radiation Oncology Biology and Physics, 63*(1), 56–63.

Corn, B., Yousem, D., Scott, C., Rotman, M., Asbell, S., Nelson, D., et al. (1994). White matter changes are correlated significantly with radiation dose. *Cancer, 74*, 2828–2835.

DeAngelis, L., Delattre, J.-Y., & Posner, J. (2001). Neurological complications of chemotherapy and radiation therapy. In M. Aminoff (Ed.), *Neurology and General Medicine* (3rd ed., pp. 437–458). Philadelphia: Churchill-Livingstone.

Dunbar, S., & Loeffler, J. (1994). Stereotactic radiation therapy. In P. Mauch & J. Leoffler (Eds.), *Radiation oncology: Technology and biology* (pp. 237–251). Philadelphia: WB Saunders.

Llanes, S., Torres, I. J., Roeske, A., Mundt, S., Keedy, S., & Zakowski, S. (2004). Temporal lobe radiation and memory in adult brain tumor patients. *Journal of the International Neuropsychological Society, 10*(S1), 185.

Monje, M. L., Mizumatsu, S., Fike, S., & Palmer, T. D. (2002). Irradiation induces neural precursor-cell dysfunction. *Nature Medicine, 8*(9), 955–962.

Monje, M. L., & Palmer, T. (2003). Radiation injury and neurogenesis. *Current Opinions in Neurology, 16*, 129–134.

Nagel, B. J., Palmer, S. L., Reddick, W. E., Glass, J. O., Helton, K. J., Wu, S. J., et al. (2004). Abnormal hippocampal development in children with medulloblastoma treated with risk-adapted irradiation. *American Journal of Neuroradiology, 25*, 1575–1582.

Phillips, P., Delattre, J., Berger, C., & Rottenberg, D. (1987). Early and progressive increase in regional brain capillary permeability following single and fractionated dose cranial irradiation in the rat (Abstr.). *Neurology, 37*(Suppl. 1), 301.

Rottenberg, D. (1991). Acute and chronic effects of radiation therapy on the nervous system. In D. Rottenberg (Ed.), *Neurological complications of cancer treatment* (pp. 3–17). Boston: Butterworths.

Vigliani, M., Sichez, N., Poisson, M., & Delattre, J. (1996). A prospective study of cognitive functions following conventional radiotherapy for supratentorial gliomas in young adults: 4-year results. *International Journal of Radiation Oncology, Biology Physics, 35*, 527–533.

Early-Onset Alzheimer's Disease

▶ Alzheimer's Dementia

Early-Onset HD

▶ Juvenile Huntington Disease

Early-Onset Huntington Disease

▶ Juvenile Huntington Disease

Early-Onset Parkinson's Disease

▶ Juvenile Parkinson's Disease

Echo

▶ Repetition

Echocardiogram

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Synonyms

Cardiac ultrasound

Definition

An *echocardiogram* is a medical test that uses ultrasound or sound waves to create a moving "real-time" image to examine the heart. It involves no radiation and no injection of foreign substances into the body, and therefore has little or no risk. It can be performed in a physician's office or in the hospital. A sonographer places a lubricant on the surface of the skin over the heart, and rubs a device that sends sound waves over the area, which are then bounced to varying degrees off the heart tissue. The amount, timing, pattern, and method of return of those sound waves back to the sensor reflects the structure, quality, and movement of the heart muscle and accompanying tissues. This allows "visualization" of the heart's chambers, walls, valves, and the major blood vessels that exit from the heart. These images can be studied as they are happening and they can be captured and recorded for a permanent record.

Current Knowledge

Measures are obtained of several structures and functions of the heart. The direction, velocity, and smoothness of blood flow are assessed. The pumping function is evaluated, usually measured as ejection fraction or EF, that is, the proportion of the blood in the left ventricle that is pumped out with each heart beat. Normal EF ranges from 55% to 65%, and heart failure or cardiomyopathy is present when EF is less than about 35%. The size, thickness, stiffness, and movement of the heart chambers and walls are measured, as are the structure, thickness, and movement of each heart valve, identifying leaking, tightening, or incompetence of some valves. The echocardiogram is used to find clots or infections in the heart.

Variations of the standard echocardiogram can be used to obtain additional information. During a stress echocardiogram, an echocardiogram is performed while the patient is exercising on a treadmill to measure the heart structure and function in response to exercise. A transesophageal echocardiogram is used to obtain visualization of difficult-to-see valves, vessels (especially the aorta), and other surrounding anatomic structures by having the ultrasound probe "swallowed" into the esophagus, allowing closer and clearer "pictures" of the heart.

Cross References

- ▶ Atherosclerosis
- ▶ Congestive Heart Failure
- ▶ Coronary Disease
- ▶ Myocardial Infarction
- ▶ Thrombosis

References and Readings

Hung, J., Lang, R., Flachskampf, F., Shernan, S. K., McCulloch, M. L., Adams, D. B., et al. (2007). 3D Echocardiography: A review of the current status and future directions. *Journal of American Society of Echocardiography, 20*, 213–233.

Echoic Memory

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Synonyms

Auditory sensory memory

Definition

The term auditory sensory (echoic) memory refers to the brain's ability to maintain short lived, but vivid recollections (echoes) of the acoustic qualities of simple auditory stimuli. Such recollections can persist for up to 30 s after the presentation of auditory stimulus, whether or not the listener attends to the stimulus.

Current Knowledge

The most common way to study echoic memory is to present two sounds (e.g., tones of the same or different

pitch) that are separated by a delay, called an inter stimulus interval (ISI). The task is to compare the sounds to make a subtle acoustic judgement (e.g., same or different). When there is silence between the sounds, performance is very good at short ISIs and declines exponentially at longer ISIs, reaching an asymptotically low level at ISIs longer than 10 s.

The time course of the decay of echoic memory is the same even if participants engage in silent mental activity during the ISI, regardless of the nature of the mental activity (e.g., counting vs. math equations). However, participants show much worse performance if the ISI contains an intervening sound. This suggests that echoic memory is an automatic process, unlike other forms of working memory. Consistent with this is the evidence that the normal time course of decay of echoic memory is unchanged whether or not the listener attends to the sound. The critical substrates of echoic memory seem to lie in areas of the auditory cortex, and not in the prefrontal cortex as do other components of working memory.

Individuals with schizophrenia show deficits in echoic memory, even at the shorter ISIs, but show no deficit when there is no delay between the sounds. However, if the difference in pitch between the tones is adjusted so that schizophrenic patients perform equivalent to typical adults at the shortest ISI, the subsequent performance between 1 and 20 s is equivalent between the groups. So individuals with schizophrenia display a normal time course of decay of echoic memory if the task is easier to begin with. This suggests that the deficit in echoic memory in schizophrenia is not related to their ability to maintain the auditory memory traces, but seems to result from impaired precision with which to encode the acoustic properties of sound.

Echoic memory is also impaired in other clinical populations. Patients with Alzheimer's disease (AD) do show a deficit in the time course of memory decay for echoic memory. Measured using an electroencephalogram (EEG), AD patients show lower amplitude of a particular electrical potential that reflects automatic detection of the direction of stimulus change. This amplitude decreases and a function of the ISI more in the Alzheimer's group than in the control group. This suggests that the memory trace decays faster in the AD patients than in age-matched healthy controls.

Cross References

► Alzheimer's Disease
► Auditory System
► Electroencephalography
► Evoked Potentials
► Working Memory

References and Readings

Alain, C., Woods, D. L., & Knight, R. T. (1998). A distributed cortical network for auditory sensory memory in humans. *Brain Research, 812*, 23–37.

Cowan, N. (1988). Evolving conceptions of memory storage, selective attention, and their mutual constraints within the human information-processing system. *Psychological Bulletin, 104*, 163–191.

Javitt, D. C., Strous, R. D., Grochowski, S., Ritter, W., & Cowan, N. (1997). Impaired precision, but normal retention, of auditory sensory ("echoic") memory information in schizophrenia. *Journal of Abnormal Psychology, 106*, 315–324.

Naatanen, R., Paavilainen, P., Alho, K., Reinkainen, K., & Sams, M. (1989). Do event-related potentials reveal the mechanism of the auditory sensory memory in the human brain? *Neuroscience Letters, 98*, 217–221.

Pekkonen, E., Jousmaki, V., Kononen, M., Reinikainen, K., & Partanen, J. (1994). Auditory sensory memory impairment in alzheimer's disease: an event-related potential study. *Neuroreport, 5*, 2537–2540.

Ecologic Illness

► Multiple Chemical Sensitivity

Ecological Validity

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Definition

In neuropsychological settings, ecological validity refers to the extent to which performance on a cognitive test predicts actual real-world behavior or future behavioral outcomes.

Current Knowledge

Ecological validity may be affected by complex environmental interactions that occur in the brain and behavior relationships. Egon Brunswik first used the term in 1955 to

describe the ability to generalize the findings obtained under controlled experimental conditions to behavior observed in a naturalistic environment (Tupper & Cicerone, 1990). Ecological validity can be conceptualized as having two aspects; verisimilitude, the extent to which the test procedures resemble the behavior to be predicted and veridicality, or the empirical demonstration of accurate predictions of the behavior of interest (Franzen & Wilhelm, 1996). Although these two aspects can be independent, the designed test procedures with a view toward increasing verisimilitude can increase the probability of the veridicality or capacity to accurately predict the target behavior. Ecological validity empirical investigations include examinations of performance on neuropsychological tests as predictors of activities of daily living, treatment response and adherence, recovery from injury, employability, and diagnostic outcome (Lezak, Howieson & Loring, 2004).

Ecological validity is often distinguished from a similar term, external validity. External validity is the ability to broadly generalize findings to a larger population once a given experiment has been completed. Compromises in external validity generally result from methodological limitations in study design, such as conducting a study in a sample of young college students with subsequent attempts to generalize findings to older adults. Ecological validity differs from external validity in that it primarily relates to the relationship between an individual's performance on a test and his/her ability to function in everyday settings (Tupper & Cicerone, 1990).

There is often a trade-off between the ecological validity of a neuropsychological test and its reliability (Cardwell & Flanagan, 2003). The artificial environment of the laboratory may produce high levels of reliable measurement, but poor ecological validity. On the other hand, studies conducted in naturalistic environments may produce high levels of ecological validity, but may have poor reliability of measurement. As an example, consider the ecological validity of memory assessment. In the highly standardized, artificial, optimal testing environment of the clinic, the neuropsychologist may be able to obtain reasonably reliable measures of memory performance. However, these laboratory measures may be a poor reflection of the memory demands required under real-life conditions of the home or occupational environment in which an individual may experience distractions, social pressures, visual cues, and multi-tasking challenges.

Another important contribution to ecological validity is the "decoupling" problem (Cardwell & Flanagan, 2003). Decoupling refers to the typical assessment of a particular aspect of cognition in which an attempt is made to isolate measurement of that ability from other cognitive abilities. Although the resulting assessment may be highly reliable, it may also have little direct application to the more complex interaction of cognitive systems that an individual engages for successful execution of activities of daily living.

Appreciation of issues related to ecological validity is an important challenge in neuropsychological practice and research. Sbordone (1996) proposed the following critical considerations that may impact ecological validity:

- Environmental conditions under which neuropsychological tests are administered may limit the generalizability of findings to a real-life setting.
- The psychometric properties of the neuropsychological tests themselves may limit the generalizability of findings to a real-life setting.
- An individual's performance on a neuropsychological test may be influenced by non-cognitive conditions (e.g., emotional issues, sensory impairment, medications) that may limit the generalizability of findings to a real-life setting.
- Over-reliance on neuropsychological test scores without consideration of additional environmental context may limit the generalizability of findings to a real-life setting.

Cross References

- ▶ Activities of Daily Living
- ▶ Generalizability Theory
- ▶ Test Reliability
- ▶ Test Validity

References and Readings

Cardwell, M., & Flanagan, C. (2003). *Psychology AS: The complete companion*. Cheltenham, UK: Nelson Thornes.

Franzen, M. D., & Wilhelm, K. (1996). Conceptual and theoretical considerations in ecological validity. In R. J. Sbordone & C. J. Lang (Eds.), *Ecological validity in neuropsychological testing* (pp. 91–112). Boca Raton, FL: CRC Press.

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

Sbordone, R. J. (1996). Ecological validity: Some critical issues for the neuropsychologist. In R. J. Sbordone & C. J. Lang (Eds.), *Ecological validity in neuropsychological testing* (pp. 15–42). Boca Raton, FL: CRC Press.

Tupper, D. E., & Cicerone, K. D. (1990). Introduction to the neuropsychology of everyday life: Assessment and basic competencies. In D. E. Tupper & K. D. Cicerone (Eds.), *The neuropsychology of everyday life* (pp. 3–18). New York: Springer.

Edema

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Definition

Edema is swelling caused by disruption of the fluid within the body's tissues. Edema is typically a localized inflammatory reaction following brain injury. Neural tissue is injured, which causes a release of interstitial fluids within a restricted space, the skull cavity.

Current Knowledge

In brain injury, there are two principle types of edema. Vasogenic edema occurs when there is a breakdown of the tight junction cells in capillaries that make up the blood–brain barrier. Cytoxic edema occurs when sodium and potassium pumps within the glial membrane break down, and as a result, sodium and water are retained within the cell membrane leading to swelling. In contrast to vasogenic edema, cytotoxic edema does not disturb the blood–brain barrier.

Focal brain edema often contributes to the effects of brain swelling to increase intracranial pressure following trauma. Clinical symptoms that should raise suspicion for the presence of edema or swelling include: alteration in the level of consciousness, bradycardia, rise in blood pressure, abnormal breathing pattern, extraocular eye movement abnormalities, pupil abnormalities, and abnormal reflexes on the same side as the suspected brain injury. Edema and swelling can be managed in a number of different ways. These treatment techniques include surgical evacuation of a lesion, decompressive craniectomy, stabilizing arterial blood pressure within the brain, administering corticosteroids to stabilize cellular membranes, and using drug protocols to actively reduce intracranial pressure. Emergent management of increased intracranial pressure is strongly associated with increased survival following severe brain injury. Decompressive craniectomy, in particular, has been shown to dramatically improve brain oxygenation, restore blood supply, and reduce impact of severe brain injury.

Cross References

► Brain Swelling
► Decompressive Craniectomy
► Intracranial Pressure

References and Readings

Ho, C. L., Wang, C. M., Lee, K. K, Ng, I., & Ang, B. T. (2008). Cerebral oxygenation, vascular reactivity, and neurochemistry following decompressive craniectomy for severe traumatic brain injury. *Journal of Neurosurgery, 108,* 943–949.

Marmarou, A., Signoretti, S., Fatouros, P. P., Portella, G., Aygok, G. A., & Bullock, M. R. (2006). Predominance of cellular edema in traumatic brain swelling in patients with severe head injuries. *Journal of Neurosurgery, 14,* 720–730.

Marmarou, A. (2007). A review of progress in understanding the pathophysiology and treatment of brain edema. *Neurosurgery Focus, 22,* E1.

Unterberg, A. W., Stover, J., Kress, B., & Kiening, K. L. (2004). Edema and brain trauma. *Neuroscience, 129,* 1021–1029.

Edge Detection

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Definition

Edge detection refers to the process of localizing discontinuities in an image or visual scene introduced by relatively sharp changes in luminance or other visual properties such as texture or color. This process is instrumental in image segmentation, the retrieval of object shapes, and the analysis of spatial relations in a visual scene.

Current Knowledge

An early visual process, edge detection is related to low-level image processing performed by the striate cortex. The operation of *simple cells* (Hubel & Wiesel, 1959, 1962) in the striate cortex can be described to some degree as edge or line detection. These cells respond vigorously to luminance edges or to bright/dark lines with specific orientations at specific retinal positions. The receptive fields of such cells have long elongated structures composed of excitation areas neighboring inhibition areas. Other cells in the striate cortex combine these properties with sensitivity to the motion or length of edges and lines while exhibiting more invariance for position and orientation. A different interpretation of the operation of simple cells favors their description in terms of local spatial frequency analysis. This interpretation finds support in the finding that the receptive fields of many simple cells

exhibit smaller areas of excitation/inhibition next to their primary ones (De Valois & De Valois, 1988). This structure, resembling the shape of a *Gabor function*, makes them suitable for piecewise local frequency analysis accomplished by multi-scale multi-orientation *filters*. Consistent with this idea, simple cells are organized continuously along two dimensions, frequency and orientation.

Another line of evidence is provided by human psychophysics. When presented with sinusoidal gratings of variable frequency, orientation, and contrast, observers *adapt* to a given frequency and orientation after sustained exposure to them (Blakemore & Campbell, 1969). Adaptation is evidenced by higher contrast values necessary to detect a grating when compared with those required before prolonged exposure. Critically, this drop in sensitivity is only noticed around the frequency and orientation values of gratings to which observers have previously been exposed. The explanation of this result is couched in terms of frequency-dedicated psychophysical channels. Channels responsible for a given frequency *fatigue* after prolonged exposure and require a higher contrast level to prompt grating detection. While these channels are psychophysical constructs postulated on the basis of behavioral results, their existence and functioning are in line with the operation of simple cells described above.

The operation of simple cells has been differently modeled according to the two views above. A symbolic description of images in terms of edges and lines as visual primitives was earlier proposed (Marr, 1982), and methods for automatic edge detection that approximate reasonably well the performance of an idealized simple cell edge detector were designed (Canny, 1986). The alternative approach, on the other hand, explores the advantage of using sets of filters at multiple scales and orientations as visual primitives (Adelson & Bergen, 1985; Malik & Perona, 1990). Explanations of the receptive fields of simple cells have also been provided by invoking optimal tuning to the statistics of natural images (Olshausen & Field, 1996). In the broader context of visual object recognition, edge detection is modeled as the first step in recovering object shape as performed by the visual cortex (Riesenhuber & Poggio, 1999).

Developmental studies found that contrast sensitivity is significantly reduced in infants when compared with adults and, critically, sensitivity is restricted to low-level frequencies (Atkinson, Braddick, & Moar, 1977). The immediate implication of this finding is that infants are not able to perceive sharp edges as adults do. Cortical cells appear to exhibit critical periods depending on their level in the visual system: the critical development of lower-level cells occurs before that of higher-level cells. Consistent with this is the fact that visual deprivation in early life impacts selectivity for edge orientation and direction of movement as proven by animal studies (Hubel & Wiesel, 1963). In humans, early removal of cataracts present at birth is critical in allowing normal visual development including normal edge detection and spatial frequency processing.

Cross References

- ▶ Filtering
- ▶ Spatial Frequency Analysis
- ▶ Striate Cortex

References and Readings

Adelson, E. H., & Bergen, J. R. (1985). Spatiotemporal energy models for the perception of motion. *Journal of the Optical Society of America. A, Optics and Image Science, 2*(2), 284–299.

Atkinson, J., Braddick, O., & Moar, K. (1977). Development of contrast sensitivity over the first 3 months of life in the human infant. *Vision Research, 17*(9), 1037–1044.

Blakemore, C., & Campbell, F. W. (1969). On the existence of neurones in the human visual system selectively sensitive to the orientation and size of retinal images. *The Journal of Physiology, 203*(1), 237–260.

Canny (1986). A computational approach to edge detection. *IEEE Transactions on Pattern Analysis and Machine Intelligence, 8*, 769–798.

Hubel, D. H., & Wiesel, T. N. (1959). Receptive fields of single neurones in the ca's striate cortex. *The Journal of Physiology, 148*, 574–591.

Hubel, D. H., & Wiesel, T. N. (1962). Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. *The Journal of Physiology, 160*, 106–154.

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.

Malik, J., & Perona, P. (1990). Preattentive texture discrimination with early vision mechanisms. *Journal of the Optical Society of America. A, Optics and Image Science, 7*(5), 923–932.

Marr, D. (1982). *Vision*. San Francisco: W. H. Freeman.

Olshausen, B. A., & Field, D. J. (1996). Emergence of simple-cell receptive field properties by learning a sparse code for natural images. *Nature, 381*(6583), 607–609.

Riesenhuber, M., & Poggio, T. (1999). Hierarchical models of object recognition in cortex. *Nature Neuroscience, 2*(11), 1019–1025.

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

EDH

- ▶ Epidural Hematoma

Edinburgh Handedness Inventory

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Description

A ten-item questionnaire designed to assess handedness by self-report of the preferred hand for carrying out common activities such as writing and drawing, throwing, and using utensils such as a toothbrush, knife, and spoon. Subjects place 1 or 2 check marks under "left" or "right," indicating strength of preference for each activity; 2 checks are to be used if the individual "would never try to use the other hand unless absolutely forced to" for the given function. As some activities require the use of both hands, the directions specify which component reflects hand preference (e.g., in striking a match, the hand that holds the match). A laterality quotient ($LQ = R - L/R + L \times 100$) can be calculated where a score of 100 reflects complete dextrality, and a score of -100 is obtained by complete sinistrals.

Some methodological problems with the Edinburgh were pointed out by Williams (1991). First, the procedure for calculating the LQ makes it possible to achieve a particular score in more than one way. For example, a score of 100 (complete right-hand preference) can be earned by placing between 10 and 20 check marks under "right" as long as no marks are placed under "left." Hence, varying degrees of strong dextrality are not distinguished. Williams also questioned the justification for weighting a "strong preference" as twice the strength of a lesser preference. He also doubted whether equal weighting of all items was justified in calculating the LQ.

Historical Background

The Edinburgh Handedness Inventory was created by R. C. Oldfield (1971) using a sample of 1,100 subjects. From 20 items initially considered, the list was reduced to 10 in an effort to limit the measure to items that would be unbiased with respect to factors such as gender, socioeconomic status, or geographical location. In this normative dataset, 26% of males and 16.6% of females reported some use of the left hand for certain activities.

Psychometric Data

McMeekan and Lishman (1975) reported test–retest reliability separately for two handedness groups totalling 73 subjects. Those initially classified as right-handers (i.e., positive LQ) had a coefficient of 0.75, while those first classified as left-handers (negative LQ) had a coefficient of 0.86. Williams (1991) reported internal consistency (coefficient alpha) of 0.93. He also noted that the least consistent items were those concerning opening boxes and using brooms.

Clinical Uses

The Edinburgh offers a rapid method for the assessment of expressed hand preference. As indicated by Oldfield (1971), the measure should not be considered sufficient for assessing individual hand preference but is useful for screening and in situations where large numbers of individuals must be evaluated. It must be kept in mind, however, that hand *preference* and *performance* are not isomorphic (Fennell, 1986), and clinical situations certainly arise where, for example, a self-described strong left-hander shows roughly comparable unimanual performances with both hands.

Cross References

▶ Handedness

References and Readings

Fennell, E. B. (1986). Handedness in neuropsychological research, In H. J. Hannay (Ed.), *Experimental techniques in human neuropsychology* (pp. 15–44). New York: Oxford University Press.

McMeekan, E. R., & Lishman, W. A. (1975). Retest reliabilities and interrelationships of the Annett Hand Preference Questionnaire and Edinburgh Handedness Inventory. *British journal of Psychology, 66*, 53–59.

Oldfield, R. C. (1971). The assessment of handedness: The Edinburgh inventory. *Neuropsychologia, 9*, 97–113.

Williams, S. M. (1991). Handedness inventories: Edinburgh versus Annett. *Neuropsychology, 5*, 43–48.

EDSS

▶ Expanded Disability Status Scale

EDTA Therapy

► Chelation

Education

► Patient-Family Education

Educational Productivity

► Academic Competency

Educational Testing

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Synonyms

Academic skills

Definition

Education can be defined as learning knowledge and skills after receiving specialized training, such as teaching, tutoring, or advising. Testing can be viewed as a process or tool used to evaluate a person's skills or performance in a specialized area. Educational testing is the evaluation of students' academic abilities to determine current levels of achievement, such as reading, vocabulary, or basic skills. In our current age of accountability, educational testing is often used as an *inappropriate* indicator of quality instruction. Such a score is not useful because learning is multifaceted and cannot be limited to a single number. However, educational testing should be a critical component of the learning process within the education enterprise.

Cross References

► Academic Competency
► Basic Achievement
► Reading
► Vocabulary

References and Readings

D'Amato, R. C., Crepeau-Hobson, F. C., Huang, L. V., & Geil, M. (2005). Ecological neuropsychology: An alternative to the deficit model for conceptualizing and serving students with learning disabilities. *Neuropsychology Review, 15*(2), 97–103.

Witsken, D., Stoeckel, A., & D'Amato, R. C. (2008). Leading educational change using a neuropsychological response-to-intervention approach: Linking our past, present, and future. *Psychology in the Schools, 45*(9), 781–798.

Efferent

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Definition

Efferent is an anatomical term which indicates functional directionality. In nervous tissue, efferent is often used synonymously with motor when it refers to nerves carrying impulses out of the central nervous system to initiate muscle contraction. Efferent can also be used in general to refer to any connection emanating from a structure within the nervous system. The opposite direction of conduction is afferent.

Effort

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Definition

Effort is derived from the French word meaning "to force." Today it is widely used to refer to the expenditure of energy (i.e., work) to achieve a particular goal. Within psychology, effort refers to controlled attention or intentional processing that is required to complete demanding tasks that require intense attentional focus or sustained performance.

Historical Background

Effort was one of the subjective experiences that psychologists of the early twentieth century tried to account for as they contemplated the nature of consciousness, attention, intention and "will." James (1890) in his Principles of Psychology and lectures to teachers on attention distinguished between spontaneous passive attention and voluntary attention, which is "deliberate and effortful." He stated that deliberate attention could not be sustained indefinitely, thus linking attentional effort to the idea that people have limited capacity for sustained attention.

Kahneman (1973) formalized the construct of effort in relationship to capacity limitations that influence selective attention. He proposed that attentional capacity is limited by available cognitive resources and also arousal present at a given point in time, and the amount of effort that can be allocated for attention is constrained by this capacity. This theory placed strong emphasis on the role of drive, disposition, and biological state in determining available capacity, an important advance from earlier information processing based theories of attention. Variations in focused attention can be observed on measures of psychophysiological response (Hitchcock et al., 2003; Kahneman, Beatty, & Pollack, 1967).

Shiffrin and Schneider (1977) and Schneider and Shiffrin (1977) proposed that a distinction exists between automatic and controlled attention. They conducted experiments, which showed that consistent memory demands tend to enable greater automaticity on target detection tasks, resulting in faster response times. When stimulus and task parameters were varied in a more random manner, and memory load increased, controlled processing was required, which tended to be experienced as effortful. Hasher and Zacks (1979) extended these ideas, making a distinction between automatic and effortful processing. They showed that differences in effortful processing occur as a function of differences in attentional capacity within and between people, as well the fact that attentional demands depend on the requirement for memory encoding, subsequently showing that demands for effortful processing occur as people reach advanced age, under distracting conditions, and as a result of environmental stress.

Pribram and McGuinness (1975) proposed one of the first neurophysiological models to link effort as a critical process in the control of attention. Effort was conceived as the byproduct of the arousal and activation associated with the control of anticipation and the sensory and response selection demands that govern attention. This model had considerable influence on neuropsychological theories of attention.

Current Knowledge

All cognitive tasks differ with respect to their effortful demands, i.e., the extent to which controlled attentional focus is required for successful performance. Generally sensory selective attention can be performed more automatically than tasks requiring response intention and executive control. However, it is possible to create selective attention tasks with conjoint stimulus features, high levels of interference, stimulus ambiguity, or other features that will increase the requirement for controlled effortful processing. Most tasks can also be made more effortful by increasing their duration so as to require greater sustained attention (Grier et al., 2003). People typically report exerting high levels of attentional effort when a task requires working memory and intense attentional focus, as is the case for tests like the Paced Serial Addition Test (PASAT). The assessment of impairments related to attention effort is best accomplished through the use of tests with increasing levels of demand. For example, by administering a working memory task, such as the n-back paradigm, it is possible to determine whether a dramatic decrease in performance occurs when the patient is required to perform the 3-back version of the task compared to the more standard 2-back version. Memory load tends to cause decrements in sustained attention (Parasuraman, 1979).

Problems with attentional effort are characteristic of a number of clinical disorders. Patients with psychiatric disorders frequently have great difficulty as demands for attentional effort increase. This is most evident in patients with major affective disorders (Byrne, 1977; Cohen, Lohr, Paul, & Boland, 2001). Patients with both major depression and mania have difficulty with attentional effort, though for different reasons. While depression is associated with apathy and a tendency to exert insufficient effort as demands increase, manic patients have difficulty in performing effortful tasks as well because of a tendency to respond impulsively and break from response set. Schizophrenia also affects focused attention and performance in effortful tasks (Nuechterlein, Garmezy, Devine, Schulz, & Tamminga, 1989).

Damage to frontal and subcortical brain systems tends to affect effortful processing (Cohen, 1993). Following cingulotomy, patients usually show preserved intellectual functioning, yet have reduced capacity for behavioral initiation and spontaneity, seeming more apathetic particularly when the situation is challenging. Similarly, patients with frontal lobe disturbances usually have considerable difficulty as effortful demands increase. This is also the case for patients having disturbances of subcortical systems that govern arousal and drive, and for diseases that affect

subcortical white matter systems, such as multiple sclerosis, HIV, and cerebral microvascular disease. In disorders such as multiple sclerosis, problems with attentional effort may correspond with the subjective experience of fatigue, a very common symptom in this neurological disease.

Disturbed attentional effort resulting from psychiatric or neurological brain disorders is not volitional, as the patient is not trying to purposely reduce effort for some gain. However, clinical neuropsychologists are often confronted with questions regarding the degree to which certain patients are exerting adequate effort. The concern is that a particular patient may not be motivated to perform well on a task because of some incentive associated with having a cognitive impairment (e.g., disability compensation). Usually the underlying question is whether the patient is malingering. It is important to distinguish failure to exert effort because of situational incentive from attention impairments on tasks with effortful demands. Often patients who are malingering and not exerting sufficient effort actually fail on tasks with little effortful demand, particularly when the task is assessing a cognitive function that they believe should be impaired.

Future Directions

There is now considerable experimental data on factors that influence attentional effort and also disturbances of effortful processing associated with various clinical disorders. However, to date neuropsychological methods have not fully integrated methods for assessing performance in the context of effortful attentional demands systematically in most clinical test batteries. Test development directed at this are needed. There is also a need for further research on the relationship between the subjective experience of effort and fatigue and objective performance measures of controlled attentional processing. There is thought to be an implicit relationship between controlled processing and these subjective experiences, though they do not necessarily correspond in an exact way for all task or clinical disorders. The characteristics of reduced effort associated with malingering and intentionality has received considerable forensic neuropsychology consideration, but has not been adequately examined relative to the broader existing literature on attention and effort. The issue of dissociating conscious intention and volitional reductions in effort from those related to psychiatric and neurological status remains a major challenge. Functional brain imaging methods will undoubtedly continue to shed light on how the brain is responding under different conditions of effortful attentional demands and motivation (Garavan, Ross, Murphy, Roche, & Stein, 2002; Hester, D'Esposito, Cole, & Garavan, 2007; Kubler, Murphy, Kaufman, Stein, & Garavan, 2003).

Cross References

- ▶ Attention
- ▶ Automaticity
- ▶ Capacity
- ▶ Fatigue
- ▶ Focused Attention
- ▶ Malingering
- ▶ Sustained Attention

References and Readings

Byrne, D. G. (1977). Affect and vigilance performance in depressive illness. *Journal of Psychiatric Research, 13*, 185–191.

Cohen, R., Lohr, I., Paul, R., & Boland, R. (2001). Impairments of attention and effort among patients with major affective disorders. *The Journal of Neuropsychiatry and Clinical Neurosciences, 13*, 385–395.

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

Grier, R. A., Warm, J. S., Dember, W. N., Matthews, G., Galinsky, T. L., Szalma, J. L., et al. (2003). The vigilance decrement reflects limitations in effortful attention, not mindlessness. *Human Factors, 45*, 349–359.

Hagen, J. W. (1967). The effect of distraction on selective attention. *Child Development, 38*, 685–694.

Hasher, L., & Zacks, R. (1979). Automatic and effortful processes in memory. *Journal of Experimental Psychology: General, 108*(3), 356–88.

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

Hitchcock, E. M., Warm, J. S., Matthews, G., Dember, W. N., Shear, P. K., Tripp, L. D., et al. (2003). Automation cueing modulates cerebral blood flow and vigilance in a simulated air traffic control task. *Theoretical Issues in Ergonomics Science, 4*, 89–112.

James, W. (1890). *The principles of psychology.* New York: H. Holt and Company.

Kahneman, D. (1973). *Attention and effort.* Englewood Cliffs, NJ: Prentice-Hall.

Kahneman, D., Beatty, J., & Pollack, I. (1967). Perceptual deficit during a mental task. *Science, 157*, 218–219.

Koelega, H. S., Brinkman, J. A., Hendriks, L., & Verbaten, M. N. (1989). Processing demands, effort, and individual differences in four different vigilance tasks. *Human Factors, 31*, 45–62.

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

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

Nuechterlein, K. H., Garmezy, N., Devine, V. T., Schulz, S. C., & Tamminga, C. A. (1989). *Sustained, focused attention under high processing loads: Relevance to vulnerability to schizophrenia. Schizophrenia: Scientific Progress* (pp. 95–102). New York: Oxford University Press.

Parasuraman, R. (1979). Memory load and event rate control sensitivity decrements in sustained attention. *Science, 205*, 924–927.

Pribram, K. H., & McGuinness, D. (1975). Arousal, activation, and effort in the control of attention. *Psychological Review, 82*, 116–149.

Schneider, W. & Shiffrin, R. M. (1977). Controlled and automatic human information processing: I. Detection, search, and attention. *Psychological Review, 84*(1), 1–66.

Shiffrin, R. M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending and a general theory. *Psychological Review, 84*(2), 127–190.

Effort Testing

► Symptom Validity Assessment
► Test of Memory Malingering

Effortful Control

► Self-Regulation

Eighth Cranial Nerve

► Vestibulocochlear Nerve

Electrical Brain Injury

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Synonyms

Electrical injury; Electrocution; Lightning injury

Definition

For the purposes of this entry, "electrical brain injury" (EBI) refers to cerebral damage, system dysfunction, or death sustained from direct or indirect exposure to an electric current or source. Following the conventions generally present in the literature, the term "electrical injury" (EI) is used to distinguish injuries due to man-made sources of electricity from natural sources, referred to as "lightning injury" (LI).

Historical Background

Records of electricity-related injuries related to natural phenomena date back to the beginning of recorded history in cultures across the globe. Injury sustained from man-made electrical power is understandably a more recent occurrence, as generated electricity is a relatively modern development.

An appreciation of the safety risks of electrical power grew as the technology to harness and apply electricity for commercial and residential use became more common in the middle of the nineteenth century. In 1879, the first fatality attributed to exposure to electrical current was recorded. In 1881, after observing an accidental electrocution, Alfred Southwick developed the electric chair and championed its use as a method of capital punishment. The first state-sponsored death by electrocution occurred on August 6, 1890. Widespread attention was called to the potential lethality of electricity when Thomas Edison publicly vilified the use of the electric chair in an unsuccessful attempt to sway public opinion away from the use of alternating current in households and businesses and toward his system of direct current.

Current Knowledge

Epidemiology

The available statistics indicate that EI accounts for over 500 deaths per annum in the United States. The majority of these injuries occur in the workplace, with an average of 320 deaths and 4,000 injuries due to EI recorded in the past decade. The vast majority of EI injuries, perhaps as high as 93%, are sustained by males. From 1992 to 1998, EI resulted in 2,268 deaths and 32,309 nonlethal injuries across all occupations. EI accounts for approximately 5–6% of all work-related fatalities; some occupations, such as construction, are more prone to EI. The direct and

indirect costs to employers are substantial, estimated to be over $15 million per injury. In private residences, over 200 deaths per year due to EI are recorded, typically due to faulty or mishandled electrical appliances. In 2003, an estimated 160 electrocutions related to consumer products were reported. In the United States, lightning injury is reported to cause approximately 50–300 deaths annually, with perhaps up to four times that number incurring non-fatal injuries. Lightning injuries have been reported to result in up to ten times the morbidity than other types of electricity-related injuries.

These statistics are generally considered to be underestimates of the true nature and extent of EBI, as many cases of "mild" EBI may not report for treatment to occupational health centers, emergency departments, and primary care physician offices.

Neuropsychological Effects of EBI

Physical, affective, and cognitive symptoms are routinely noted after EBI. However, the pathophysiology of neurological injury in EBI is not clear in many cases, and a high degree of variability in the resultant symptom constellation is present. The nature and degree of sequelae are determined by multiple factors, including:

1. Properties of the electrical current (as described in Duff and McCaffrey, 2001):
   (a) Voltage (the force behind electrons as they travel through a conducting substance). This does not appear to represent a dose–response relationship, as exposure to lower voltages may or may not lead to a greater sparing of functioning when compared with higher voltages.
   (b) Amperage (intensity of the current). Higher current intensity is generally more lethal and more likely to lead to injury to interior bodily tissues.
   (c) Resistance (the degree of force electrons must overcome as they travel through a conductor). Within the human body, the level of resistance varies dramatically among different tissue types. Nerves and blood vessels have the least amount of resistance, skin an intermediate amount, and bones the highest. Tissues with higher levels of resistance will be exposed to significant heat, which is very likely to lead to severe burns or destruction of the tissue. Tissues with lower resistance, such as nerves, are most likely to conduct the electrical current; this likely accounts for the high level of neurocognitive and affective complaints in EBI.
   (d) Type of current. Alternating current (AC) consists of a cyclical, back-and-forth flow of electrons. Direct current (DC) entails a unidirectional pattern of electron flow. Lightning injury is a brief, but massive, exposure to direct current. At high voltages, AC and DC have similar injurious effects. At lower voltages, DC is more likely to cause a single contraction of muscles and propel the individual back from the source of the electricity. AC tends to cause muscular contractions in the victim that can extend the amount of exposure to the current; because of this tendency, AC is considered to present a threefold risk to the body over that of DC.
2. Direct versus indirect cerebral exposure to the current.
   (a) In cases where the electrical current is suspected to have passed directly through the brain (such as in many lightning strikes), brainstem, and respiratory control centers may be immediately affected, causing respiratory cessation. Acute deficits in cranial nerve functions, intracranial hematomas, seizures, cerebral edema, and other factors that may represent the effects of direct CNS injury have also been reported. Some investigators have speculated a more diffuse CNS injury in cases that appear to reflect a more peripheral path of electricity through the body.
   (b) Indirect compromise of cerebral functioning may result from ischemic and/or anoxic insult secondary to EBI-induced cardiac dysrythmias or tissue destruction. Metabolic instability may arise from surface or (more commonly) deep tissue burns, renal failure (kidneys are rarely directly affected themselves, but succumb easily to ischemia and anoxia), and other pathologies.
3. The route of the current through the body. Electrons flow toward a grounding source through substances offering the least resistance. It is thought that neurological and cognitive complications are more extensive when the entry and exit points imply a direct, vertical course through the body (e.g., head to foot), as this pathway would be expected to entail the brain and many vital organs. However, an increasing amount of data suggest that more "peripheral" injuries (e.g., the current entering one hand, crossing the body, and exiting the opposite hand) may substantially involve the CNS as well. This is thought to involve a diffuse electrical insult to the CNS. This phenomenon is speculative, as little is understood about the exact behavior of electricity after it penetrates the body.

4. The length of time the individual is exposed to the current.
5. Individual and contextual factors. Injuries are usually more substantial when sustained by infants and young children, who have thinner skin and lower external resistance when compared with an adult. Moist or wet conditions can reduce the resistance of the skin. The fact that many children place electrical cords or appliances in their mouths adds lowered resistance due to moisture to their greater general susceptibility to EBI.
6. The presence of other injuries, such as traumatic brain injuries sustained as a result of the individual being propelled backward or off of high places by the force of the electrical event.

Functional Impairments

Subjective, self-reported complaints after EBI tend to represent a broad range of cognitive and affective symptoms. Pliskin, Ammar, Fink, Hill, Malina, & Ramati (1998) compared 53 males and 10 females with peripheral EBI versus matched, non-EBI electrical worker controls. A significantly greater proportion of the EBI patients (nearly 50%) complained of paresthesias, headaches, word finding problems, and affective complaints (anxiety, stress, and feelings of sadness) over the non-EBI controls. A significantly greater number of EBI subjects (over 40%) versus controls reported other physical symptoms such as muscle twitching/spasms, pain, weakness, and loss of sensation, slowed thinking, lowered memory, and distractibility. Those who had incurred an EBI in the past 3 months reported fewer neuropsychological and affective symptoms than persons who sustained an EBI further in the past. It is important to note that these subjects sustained no known direct cerebral injuries. Also, different motivational factors (e.g., litigation) may limit the generalizability of these results.

Objective test results also tend to show high frequency of deficits in a variety of areas, but no clear symptom constellation emerges. In general, neurological complications in electrical injuries have been estimated to be quite high, perhaps occurring in nearly 70% of all patients. Brain structure often appears normal on neuroimaging, but studies of brain functioning, such as electroencephalography and regional cerebral blood flow, often show compromise, as is often the case in other diffuse cerebral pathologies. Neuropsychological testing remains the most sensitive objective functional measure.

On neuropsychological examination, memory and attention appear to be the most frequent cognitive domains affected. In their extensive review of the literature, Duff and McCaffrey (2001) reported that the most common neurocognitive deficits in EBI involved memory (37% of all impairments), attention (14.8%), sensory-motor (12.8%), and visual-motor symptoms (12.5%). Language impairments (9.6%) and certain executive deficits (5.1%) were observed less frequently. Pliskin et al. (2006) found significantly greater deficits in aspects of attention, processing speed, and motor functions in EBI patients versus controls; these deficits could not be attributed to other, non-EBI factors. Affective distress, including depressive, anxious (e.g., PTSD, specific phobias), somatoform (e.g., conversion), and adjustment symptoms are routinely found on objective assessment.

Chronic Presentation

Typically, symptom recovery tends to be most dramatic up to 12 months post EBI. Further improvement in the patient's symptom picture is likely for an additional 2 years.

However, in some persons, symptom emergence, prolongation, or exacerbation in cognitive, affective, and physical domains has been noted after the acute phase of their injury has passed.

Some chronic EBI patients have been likened to the subset of patients with "persistent post-concussion syndrome (PPCS)," who experience lingering symptoms after purported mild traumatic brain injuries. Like PPCS, there is often a dearth of "objective" indicators that the person has indeed sustained an EBI. Some have postulated that the exacerbation could be secondary to a slow, progressive neurological deterioration set into motion by the initial EBI. The apparent exacerbation may also reflect that the injured person does not realize the extent of resultant impairments until he or she returns to the demands of pre-EBI work and daily life. Lowered effort (e.g., due to affective distress, secondary gain, malingering, or other factors) may maintain symptoms in some cases. It has been estimated that the base rate of malingering in EBI is approximately 25%. The neurological insult suffered by individuals who meet criteria for malingering often is more ambiguous than those who do not.

It is as yet unclear which patients may go on to develop lingering cognitive, emotional, or physical sequelae.

Future Directions

The EBI literature is in many ways still in its infancy. A large proportion of studies rely on case reports and subjective ratings of symptoms rather than controlled experimental designs utilizing objective functional indicators such as neuropsychological testing. Most EBI is sustained by young men, resulting in a dearth of information regarding the effects of EBI on women and other age groups.

Of the experimental studies, sample sizes are routinely small and longitudinal studies have not been performed. Many studies group EBI and lightning injuries together, despite evidence that symptom presentation and recovery may be different for the two conditions. Similarly, peripheral EBI is not always distinguished from more central EBI. Potential premorbid-mediating effects on outcome, such as education, intelligence, and emotional adjustment, have not been systematically studied. Acute versus long-term recovery and its relationship to injury variables (e.g., type of current, direct versus indirect injury, etc.) and other pertinent factors (e.g., symptom validity, litigation, the presence of pain, etc.) is also needed.

References and Readings

Duff, K., & McCaffrey, R. (2001). Electrical injury and lightning injury: A review of their mechanisms and neuropsychological, psychiatric, and neurological sequelae. *Neuropsychology Review, 11*(2), 101–116.

Gatewood, M., & Zane, R. (2004). Lightning injuries. *Emergency Medicine Clinics of North America, 22*, 369–403.

Koumbourlis, A. (2002). Electrical injuries. *Critical Care Medicine, 30*(11), S424–S430.

Pliskin, N., Ammar, A., Fink, J., Hill, K., Malina, A., Ramati, A., et al. (2006). Neuropsychological changes following electrical injury. *Journal of the International Neuropsychological Society, 12*, 17–23.

Pliskin, N., Capeli-Schellpfeffer, M., Law, R., Malina, A., Kelley, K., & Lee, R. (1998). Neuropsychological symptom presentation after electrical injury. *The Journal of Trauma, Injury, Infection, and Critical Care, 44*(4), 709–715.

Pliskin, N., Fink, J., Malina, A., Moran, S., Kelley, K., Capeli-Schellpfeffer, M., et al. (1999). The Neuropsychological effects of electrical injury. *Annals of the New York Academy of Sciences, 888*, 140–149.

Primeau, M., Engelstatter, G., Bares, K. (1995). Behavioral consequences of lightning and electrical injury. *Seminars in Neurology, 15(3)*, 279–285.

Electrical Injury

► Electrical Brain Injury

Electrical Stimulation Mapping (ESM)

► Cortical Mapping

Electro-Convulsive Therapy

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Definition

Electro-convulsive therapy is a psychiatric treatment involving the use of electrically induced seizures in anesthetized patients for therapeutic purposes. Currently, ECT is most commonly used to treat patients suffering from a severe, major depression that has failed to respond to other treatments (Benbow, 2004). However, ECT is also used for the treatment of mania in bipolar disorder as well as catatonia (Benbow, 2004). Typically, ECT is administered two to three times per week and consists of a regime of 6–12 treatment sessions.

Current Knowledge

Historically, the induction of seizures for therapeutic relief dates as far back as 1785, when it was first documented in the London Medical Journal (Rudorfer, Henry, & Sackeim, 2003). However, it was not until 1937 that electricity was used to invoke seizures in humans (Fink, 1984). ECT was subsequently popularized in the 1940s and 1950s as it was inexpensive, convenient, and less invasive than other forms of psychiatric treatment such as a prefrontal lobotomy (Fink, 1984; Cerletti, 1956).

Today, the use of ECT, like all other medical procedures, requires informed consent and relies on several factors, such as the severity and length of illness, the effectiveness of alternative treatments, patient's preference, and the potential benefits and adverse effects of the procedure (Lisanby, 2007).

ECT is generally regarded as a short-term treatment for acute episodes of illness, and is often followed by pharmacological therapy or further ECT. Additionally, estimates of remission range from 30% to 70%, with the

greatest risk of relapse occurring in the first 6 months (Surgeon General, 1999; Prudic, Olfson, Marcus, Fuller, & Sackeim, 2004; Sackeim et al., 2001). While most of the published literature suggests ECT is effective against depression, it has also been argued that no study has shown a significant difference between real and placebo ECT at 1 month post-treatment (Ross, 2006). Additionally, recent research has demonstrated that some forms of ECT cause significant, chronic cognitive decline. These studies report significant global cognitive deficits, as well as memory loss, which may be long-lasting or even permanent (Sackeim et al., 2007; Breggin, 2007; Lisanby, Maddox, Prudic, Devanand, & Sackeim, 2000; MacQueen et al., 2007). In particular, bilateral electrode placement has been linked to the greatest cognitive impairment (Sackeim et al., 2008; Stoppe, Louza, Rosa, Gil, & Rigonatti, 2006; O'Connor, Gardner, Eppingstall, & Tofler, 2010). Thus, many clinicians typically utilize bilateral ECT only subsequent to the failure of unilateral treatment (O'Connor, Gardner, Eppingstall, & Tofler, 2010).

References and Readings

Benbow, S. M. (2004). Adverse effects of ECT. In A. I. F. Scott (Ed.), *The ECT handbook,* (2nd ed., pp. 170–174). London: The Royal College of Psychiatrists.

Breggin, P. (2007). ECT damages the brain: Disturbing news for patients and shock doctors alike. *Ethical Human Psychology and Psychiatry, 9*(2), 83–86.

Cerletti, U. (1956). Electroshock therapy. In A. M. Sackler et al. (Eds.), *The great physiodynamic therapies in psychiatry: An historical appraisal* (pp. 91–120). New York: Hoeber-Harper.

Fink, M. (1984). The origins of convulsive therapy. *American Journal of Psychiatry, 141*, 1034–1041.

Lisanby, S. H. (2007). Electroconvulsive therapy for depression. *The New England Journal of Medicine, 357*(19), 1939–1945.

Lisanby, S. H., Maddox, J. H., Prudic, J., Devanand, D. P., & Sackeim, H. A. (2000). The effects of electroconvulsive therapy on memory of autobiographical and public events. *Archives of General Psychiatry, 57*, 581–590.

MacQueen, G., et al. (2007). The long-term impact of treatment with electroconvulsive therapy on discrete memory systems in patients with bipolar disorder. *Journal of Psychiatry and Neuroscience, 32*, 241–249.

Mental Health: A Surgeon General (1999). *Report of the Surgeon General*, Chapter 4.

O'Connor, D. W., Gardner, B., Eppingstall, B., & Tofler, D. (2010). Cognition in elderly patients receiving unilateral and bilateral electroconvulsive therapy: A prospective, naturalistic comparison. *Journal of Affective Disorders*. PMID: 20053457.

Prudic, J., Olfson, M., Marcus, S. C., Fuller, R. B., & Sackeim, H. A. (2004). Effectiveness of electroconvulsive therapy in community settings. *Biological Psychiatry, 55*, 301–312.

Ross, C. A. (2006). The sham ECT literature: Implications for consent to ECT. *Ethical Human Psychiatry and Psychology, 8*, 17–28.

Rudorfer, M. V., Henry, M. E., & Sackeim, H. A. (2003). Electroconvulsive therapy. In A. Tasman, J. Kay, & J. A. Lieberman (Eds.), *Psychiatry* (2nd ed., pp. 1865–1901). Chichester: Wiley.

Sackeim, H. A., Haskett, R. F., Mulsant, B. H., Thase, M. E., Mann, J. J., Pettinati, H. M., et al. (2001). Continuation pharmacotherapy in the prevention of relapse following electroconvulsive therapy: A randomized controlled trial. *JAMA, 285*, 1299–1307.

Sackeim, H. A., Prudic, J., Fuller, R., Keilp, J., Lavori, P. W., & Olfson, M. (2007). The cognitive effects of electroconvulsive therapy in community settings. *Neuropsychopharmacology, 32*, 244–254.

Sackeim, H. A., Prudic, J., Nobler, M. S., Fitzsimons, L., Lisanby, S. H., Payne, N., Berman, R. M., Brakemeier, E. L., Perera, T., & Devanand, D. P. (2008). Effects of pulse width and electrode placement on the efficacy and cognitive effects of electroconvulsive therapy. *Brain Stimulation, 1*, 71–83.

Stoppe, A., Louza, M., Rosa, M., Gil, G., & Rigonatti, S. (2006). Fixed high-dose electroconvulsive therapy in the elderly with depression: A double-blind, randomized comparison of efficacy and tolerability between unilateral and bilateral electrode placement. *The Journal of ECT, 22*, 92–99.

World Health Organization (2005). *WHO Resource Book on Mental Health, Human Rights and Legislation*. Geneva, 64.

Electrocution

► Electrical Brain Injury

Electrodiagnosis

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Synonyms

EDX; Electromyography (EMG); Nerve conduction studies (NCS)

Definition

Electrodiagnosis is a neurophysiological study used to detect problems involving nerves or muscles.

Current Knowledge

Electrodiagnosis as an extension of the neuromuscular/physical examination employs the anatomical principles

of localization, searching for evidence of motor and sensory injury. It is most helpful in the evaluation of patients with problems of weakness, paresthesia, pain, or fatigue. Electrodiagnosis not only can support or confirm clinical diagnosis, but may have an important prognostic role in guiding management. EMG of neuromuscular disease applies knowledge of basic physiological processes involved in impulse transmission in peripheral nerves, the motor unit and muscles and changes produced in these processes by disease. Complications are minimal and include mild discomfort or small amount of ecchymosis or bleeding at the needle electrode insertion site. Equipment used may be digital or analog type instrument with amplifier for precise and adequate recording of the waves and signals.

There are two components in the electrodiagnostic testing of the motor unit: the nerve conduction study and the EMG test. The combination of these may help clarify different aspects of motor unit functioning and in this way assists in understanding the physiology of nerve and muscle. The nerve conduction test involves the stimulation of nerves and muscle by means of applied electric currents (microvolt) and recording of their potential (latency, amplitude, and conduction velocity). The EMG portion involves the insertion of a needle electrode into one or more muscles resulting in a brief burst of electrical activity which, after amplification, are displayed on a cathode-ray tube and frequently reproduced through a loudspeaker. This burst of activity is generally recorded as motor unit action potentials (MUAP). MUAP represents a sample of the activity of fibers of the motor unit and its characteristics in relation to the position of the needle. Particular attention is paid to the morphology, amplitude, duration, and recruitment of the motor fibers.

Cross References

► Neuron
► Neuropathy
► Paresthesia

References and Readings

Dumitru, D., Zwarts, M. J., & Amato, A. A. (Eds). (2002). *Electrodiagnostic medicine* (2nd ed.). Philadelphia, PA: Hanley & Belfus, Inc.

Johnson, E. W., & Pease, W. S. (Eds.) (1977). *Practical electromyography* (3rd ed.). Philadelphia, PA: Williams Wilkins.

Kimura, J. (Ed.). (1989). *Electrodiagnosis in disease of nerve and muscle: Principles and practice* (2nd ed.). Philadelphia, PA: FA Davis Company.

EDX

► Electrodiagnosis

Electroencephalography

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Definition

Electroencephalography (EEG) most commonly refers to the surface recording of the brain's ongoing spontaneous electrical activity, although recordings can be made from electrodes placed on the dura or within the brain parenchyma for specialized purposes.

Historical Background

The presence of electrical activity in the brain was discovered by Richard Caton, in 1875. It was not until 1924 that methods of amplifying these electrical potentials were developed, which allowed ongoing recording of EEG rhythms via surface recordings.

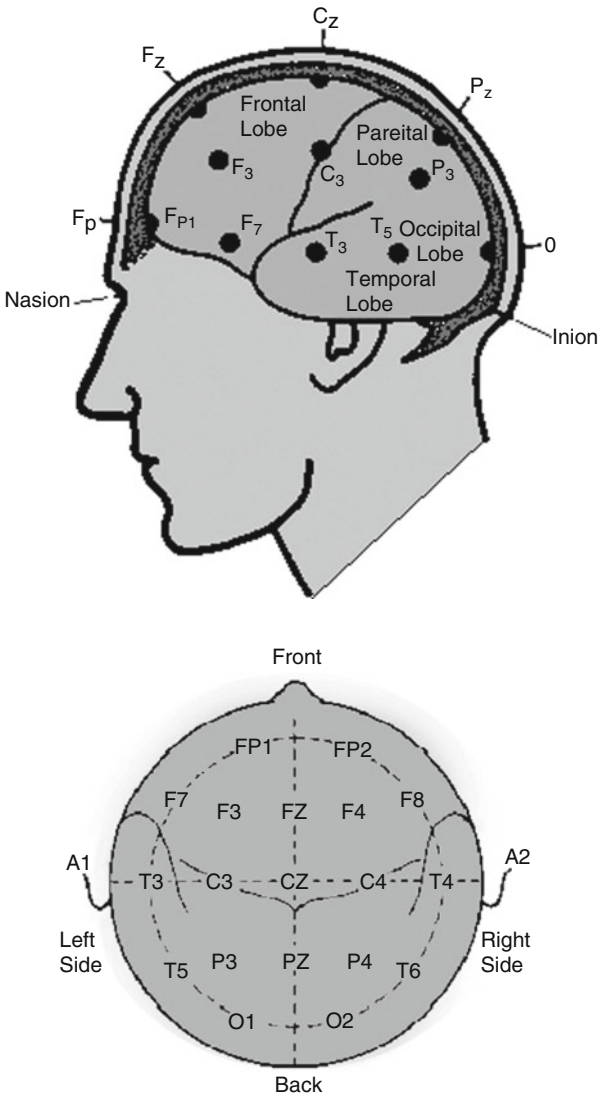
Current Knowledge

Measurement of the EEG signal has been applied to multiple clinical problems, including epilepsy, sleep disorders, and the diagnosis of brain death. All of these applications depend on knowledge of normative patterns of brain electrical activity, including the predominant frequencies, amplitudes, and degree of coherence.

Surface EEGs are generally recorded from a set of small electrodes glued to the scalp (typically 19 active electrodes), after preparing the skin to lower its impedance. In order to improve reproducibility across laboratories and sessions, the electrodes are placed in standard locations measured from the nasion (junction between the bridge of the nose and the forehead) to the inion (bump at the posterior base of the skull) and from ear to ear. These standard locations are referred to as the International 10–20 System, because the spaces between

electrodes are measured in percentages (10 or 20%) of the distances between the primary landmarks. (See EEG Figs. 1 and 2)

In standard clinical EEGs, the voltage is measured from each electrode over time, and displayed as a waveform with voltage on the vertical axis and time on the horizontal axis. In order to produce interpretable tracings, filtering of the raw signal is generally required to eliminate interference from other environmental electrical sources, muscle contraction (including heart), eye movements, etc. Resting EEG patterns generally display rhythmic activity at varying frequencies, referred to as the "background rhythm." These are arbitrarily divided into the following frequency bands: Delta (<4 Hz, associated with slow wave sleep and various forms of encephalopathy), Theta (4–7 Hz, seen in drowsiness or meditation in adults), Alpha (8–12 Hz, seen in awake and relaxed states and some forms of coma), and Beta (12–30 Hz, seen in concentration and mental activity). All of these rhythms indicate a degree of synchronization among neuronal populations, but synchrony generally decreases as rhythm increases and mental activity becomes more intense.



Electroencephalography. Figures 1 and 2 EEG Figures 1 and 2 show the locations of the major coordinates of the International 10–20 system with respect to the anatomy of the head and brain

The use of EEG monitoring for diagnosing seizures depends on the identification of either *interictal* abnormalities (i.e., unusual features of the recording present when the patient is not having a seizure, referred to as spikes or sharp waves) or *ictal* activity, in which the high amplitude synchronized wave forms characteristic of seizure activity are seen across a brain region or the whole brain. Unfortunately, the sensitivity of interictal EEG abnormalities is low (i.e., many patients with documented epilepsy have normal interictal EEGs), although it can be increased by various maneuvers including sleep deprivation, photic stimulation, or hyperventilation. Sleep deprivation may be the most effective maneuver to increase the yield of positive studies in epilepsy. On the other hand, the probability of sampling an actual seizure during routine EEG recording is low unless seizures are very frequent or very predictable. Where necessary, prolonged continuous EEG recording often coupled with videotaping can be used to increase the yield of ictal EEGs.

The EEG also shows characteristic changes in the presence of focal or diffuse brain damage, most typically a corresponding focal or diffuse slowing of the typical frequency spectrum. Generally, however, the presence of such brain damage is clinically evident and the EEG has a minor role in its diagnosis.

Normal sleep has a characteristic "architecture" – meaning a set of characteristic sleep stages that occur with some structured ordering and which are accompanied by characteristic EEG frequencies and the appearance of sleep-specific features such as sleep spindles. (See Polysomnography). Thus, EEG recording generally plays a key role, along with measures of muscle activity, respiration, and eye movements, in the diagnosis of sleep disorders.

The general phenomenon of surface EEG recording has been adapted into a number of more sophisticated and specialized techniques for particular purposes. For example, the raw EEG signal can be subjected to spectral

analysis, using Fourier transformation, to quantitatively measure the relative amplitudes of activity at different frequencies. One can also record EEG activity time-locked to specific sensory events (See evoked potentials, event-related potentials) in order to isolate the magnitude, timing, and predominant location of the brain's responses to those events. More recently, aspects of the EEG signal have been delivered back to the person from whom they are recorded to provide EEG biofeedback (i.e., an attempt to allow the individual to voluntarily modify his/her pattern of brain activity), or to control some interface device such as a computer mouse, allowing individuals with impaired motor control to interact with the environment.

All of these uses are limited by the fact that the EEG signal is normally recorded from the scalp's surface. Thus, the raw EEG signal is biased toward cortical activity nearer to the surface of the brain. Medial temporal lobe activity can be recorded more sensitively by including nasopharyngeal electrodes, but deep gray matter activity is difficult to sample. In addition, although the temporal resolution of the EEG signal is excellent, spatial resolution is imprecise because of volume conduction of signals originating from many parts of the brain. In addition, pathology that alters this conduction (e.g., lobectomy, large hematoma, etc.) may distort the signal.

Cross References

- ▶ Alpha Rhythm
- ▶ BAER
- ▶ Event Related Potential
- ▶ Evoked Potentials
- ▶ Magnetoencephalography
- ▶ P300
- ▶ Polysomnography
- ▶ Quantitative Electroencephalography
- ▶ Somatosensory Evoked Potentials
- ▶ Visual Evoked Potentials

References and Readings

EEG or Electroencephalography. Retrieved October 15, 2007 from http://bio-medical.com/news_display.cfm?mode=EEG&newsid=5

Goodin, D. S., Aminoff, M. J., & Laxer, K. D. (1990). Detection of epileptiform activity by different noninvasive EEG methods in complex partial epilepsy. *Annals of Neurology, 27*(3), 330–334.

Leach, J. P., Stephen, L. J., Salveta, C., & Brodie, M. J. (2006). Which electroencephalography (EEG) for epilepsy? The relative usefulness of different EEG protocols in patients with possible epilepsy. *Journal of Neurology, Neurosurgery, and Psychiatry, 77*(9), 1040–1042.

Electromyography (EMG)

▶ Electrodiagnosis

Embedded Figures Test

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Synonyms

Figure ground test; Group embedded figures test; Hidden figures test

Description

In these tasks participants must locate the presence of a target figure embedded within a complex background. In the Embedded Figures Test (Witkin, 1971) the participants are asked to trace with their finger the outline of a figure located within an obscuring complex scene. Participants complete 12 trials and 2 forms are available for test-retest use. The simple figure and complex scene cannot be viewed at the same time. Participants are allotted 3 min to complete the task. Spreen and Benton (1969) developed a similar task in which the participant uses a pencil to identify the embedded target shape. Participants complete 16 items presented within a non-reusable paper booklet. Unlike Witkin's EFT, in this version the target figure is located on the left side of the page, while the complex visual scene with the target embedded within is located to the right. There is also a version for left-handed participants in which the location of the target and complex background are reversed. Participants have 30 s to complete each item. The Group Embedded Figures test (Witkin et al., 2002) is an adaptation of the EFT with 18 items

that can be administered in a group format. The instructions for this test have also been translated into Dutch, German, Mandarin, and Turkish. Additionally, the Developmental Test of Visual Perception-Adolescent and Adult (DTVP-A) has a figure-ground discrimination task (Reynolds, Pearson, & Voress, 2002). In this task the participant views a complex scene with multiple (2–4) figures embedded within. The participant's task is to identify which of the multiple choice figures located at the bottom of the stimulus page are present in the complex scene.

Historical Background

Embedded figure tasks have been in use since the early twentieth century to measure perceptual processing and general problem solving. Witkin expanded the application of the EFT into the area of personality psychology by using the task as a measure of cognitive style. Briefly summarized, individuals who achieve relatively low scores on the EFT can be described as field-dependent: being pulled to the overarching organization of the figure rather than the subcomponents. By contrast, individuals who achieve higher scores on this task can be described as field-independent as they are able to separate figure from ground more easily (Witkin et al., 2002). Witkin's EFT manual cites a number of studies that have found an association between other measures of field-dependence/independence and EFT performance.

In terms of perceptual skills, figure-ground processing is believed to be mediated by cortical regions important for both object recognition as well as spatial localization. The ability to perform feature detection (target identification) requires abilities mediated by the ventral visual pathway ("what" visual system) specialized for object perception and identification (Likova & Tyler, 2008; Ring et al., 1999). However, the dorsal visual processing pathway ("where" visual system), important for spatial localization of the target amidst a distracting background, also contributes to figure-ground discrimination (Likova & Tyler, 2008). Figure-ground perception can also be examined with Navon stimuli (1977) in which small letters (local stimuli) are spatially configured to form a larger letter (global stimulus). Convergent evidence from neuroimaging and behavioral studies of patients with focal brain lesions have revealed that the right superior temporal gyrus (STG) is specialized for detecting information at the global level, whereas the left STG is important for detecting information at the local level (reviewed in Ivry & Robertson, 1998).

Psychometric Data

Normative data and psychometric test properties vary depending upon the particular embedded figures task used. The normative data for the EFT task developed by Spreen and Benton (1969) provide mean and standard deviations for ages 16–79 in approximately 10-year divisions. Sample sizes for these data, however, small (less than 25 participants per decade). Witkin's normative data for the EFT, reported in the manual, are based on larger sample sizes and are separated by gender but limited by the range of ages (10-approximately 40). Test-retest reliability with up to a 3-year interval is reportedly high (.89) as is reported odd-even reliability (.90 or greater, see manual). Construct validity has been examined and performance on Witkin's EFT is associated with other perceptual measures including block design, object assembly, picture completion, and disembedding tasks, but not verbal measures (WAIS or WISC verbal comprehension tests and sustained attention, see manual for details). The Group Embedded Figures test provides normative data in quartiles as well as the mean and SD for college aged men and women (Witkin et al., 2002). Importantly, others have expanded upon the normative data provided in the manual and there are published means and standard deviations for older female adults (see Panek et al., 1980).

Normative data is available for ages 11 through 74, with large sample sizes for each age group ($n > 75$) for the figure-ground task from the DTVP-A (Reynolds, Pearson, & Voress, 2002). For this particular task, gender was not found to be associated with figure-ground performance. Normative data is therefore not separated by gender. Internal reliability for the DTVP-A ranges from .70–.86 depending upon the age range. Internal reliability collapsed across age was found to be high for men, women, and African American, Hispanic, and European American groups (>.75). Test-retest reliability has been examined less rigorously but was found to be sufficiently high ($r = .71$) in a sample of 30 participants. Construct validity using factor analysis indicates that the figure ground task significantly loaded on the expected motor reduced visual perception construct.

Clinical Uses

Research involving clinical populations provides further support for the involvement of visuospatial abilities mediated by higher-level visual association areas in EFT performance. For instance, Corkin and colleagues (1989) observed that patients with brain lesions in the right

parietal lobe demonstrated a significant decline, over a 30-year period, on a task of Hidden Figures. In patients with Alzheimer's disease, who have known visual association cortex involvement, Mendez and colleagues (1990) found that all 30 of their participants demonstrated disturbances in figure-ground analysis despite documented preserved visual acuity and color recognition. Parkinson's disease (PD) is associated with a disruption of higher level visuospatial functions likely due to disruption of striatal-parietal circuits and PD patients with mild (Levin et al.,1989), moderate and severe disease stage (Flowers & Robertson, 1995) have been found to perform more poorly on a figure-ground task relative to control participants. Patients with Autism or Asperger syndrome are faster at performing the EFT relative to control participants (Jolliffe & Baron Cohen, 1997). This finding may also support a specialized role for the left hemisphere in disembedding the figure from the ground as Autism and Asperger's syndrome have been associated with compromise of right hemisphere functions.

Cross References

- ▶ Dorsal Visual Pathway
- ▶ Figure-Ground Discrimination
- ▶ Global Versus Local Processing
- ▶ Ventral Visual Pathway

References and Readings

Corkin, S., Rosen, T. J., Sullivan, E. V., & Clegg, R. A. (1989). Penetrating head injury in young adulthood exacerbates cognitive decline in later years. *Journal of Neuroscience, 9*, 3876–3883.

Flowers, K. A., & Robertson, C. (1995). Perceptual abnormalities in Parkinson's disease: Top-down or bottom-up processes? *Perception, 24*, 1201–1021.

Ivry, R. B., & Robertson, L. C. (1998). *The two side of perception.* Cambridge, MA: MIT Press.

Jolliffe, T., & Baron-Cohen, S. (1997). Are people with autism and Asperger syndrome faster than normal on the Embedded Figures Test? *Journal of Child Psychology and Psychiatry, 38*, 527–534.

Levin, B. E., Llabre, M. M., & Weiner, W. J. (1989). Cognitive impairments associated with early Parkinson's disease. *Neurology, 39*(4), 557–561.

Likova, L. T., & Tyler, C. W. (2008). Occipital network for figure/ground organization. *Experimental Brain Research, 189*, 257–267.

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

Navon, D. (1977). Forrest before trees: The precedence of global features in visual perception. *Cognitive Psychology, 9*, 353–383.

Panek, P. E., Funk, L. G., Nelson, P. K. (1980). Reliability and validity of the group embedded figures test across the life span. *Percept Mot Skills, 50*(3 Pt 2), 1171–1174.

Reynolds, C. R., Pearson, N. A., & Voress, J. K. (2002). *Developmental test of visual perception adolescent and adult.* Austin: Pro-Ed.

Ring, H. A., Baron-Cohen, S., Wheelwright, S., Williams, S. C., Brammer, M., Andrew, C., et al. (1999). Cerebral correlates of preserved cognitive skills in autism: A functional MRI study of embedded figures task performance. *Brain, 122*, 1305–1315.

Spreen, O., & Benton, A. L. (1969). *Embedded figures test; Manual of instructions.* Neuropsychology Laboratory, Department of Psychology, Victoria: University of Victoria.

Witkin, H. A., Oltman, P. K., Raskin, E., & Karp, S. A. (1971). *A manual for the embedded figures test.* Palo Alto, CA: Consulting Psychologist Press.

Witkin, H. A., Oltman, P. K., Raskin, E., & Karp, S. A. (2002). *Group embedded figures test manual.* Palo Alto, CA: Consulting Psychologist Press.

Embolic Stroke

▶ Cerebral Embolism

Embolism

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Definition

An embolism occurs when an object or embolus migrates from one part of the body through the blood vessels and causes blockage in a blood vessel in another part of the body. An embolus that migrates through the vascular system to the brain will likely cause an ischemic stroke. Foreign substances that can cause an embolism include a blood clot, an air bubble, amniotic fluid, a globule of fat, a clump of bacteria, chemicals (such as talc), and drugs (mainly illicit ones). Blood clots are the most common cause of embolism. Embolism can be contrasted with a thrombus which is the formation of a clot within a blood vessel, rather than being carried from somewhere else. Prevention and treatment for embolism vary depending on specific pathology (e.g., fat, air, bacteria, and blood clot) and source (e.g., bone fracture, cardiac, surgical procedure, and atherosclerotic plaque).

Cross References

► Stroke

Emergency Mental Health Treatment

► Crisis Intervention

Emotion

► Emotional Intelligence

Emotional Disorder

► Affective Disorder
► Depressive Disorder

Emotional Disturbance

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Description/Definition

An emotional disturbance (ED) is a major uncontrollable emotional or behavioral condition, which impairs the individual to a significant degree and does not allow the child or youth to profit from conventional educational services. It is most often used to refer to children and youth in school systems. An emotional disturbance is not caused by any obvious physical abnormalities of the brain, although research in the last 2 decades has begun to show a neuropsychological base for these disorders (Hartlage & D'Amato, 2008). Children and youth with ED are called by many similar names including students who are behavioral disabled (BD), emotionally disabled, psychologically and emotionally handicapped, emotionally disordered, and emotionally/behaviorally disabled (EBD).

While services have been provided from some public schools for more than a century, it was in 1975 that PL 94–142 required all schools to serve students with an ED. At this time, many new programs were begun although this population has traditionally been underserved. This term is one of the 12 disabilities specified by the Individuals with Disabilities Education Act (IDEA). It mainly covers the age group from birth to age 21. Based on the IDEA (1997), it is a condition demonstrating one or more of the following characteristics over a *long* period of time and to a *marked* degree, which adversely affects educational performance:

- An inability to learn which cannot be explained by intellectual, sensory, or health factors;
- An inability to build or maintain satisfactory interpersonal relationships with peers and teachers;
- Inappropriate types of behavior or feelings under normal circumstances;
- A general pervasive mood of unhappiness of depression; or
- A tendency to develop physical symptoms or fears associated with personal or school problems. [Code of Federal Regulations, Title 34, Section 300.7(c)(4)(i)]
- A student who is diagnosed with an ED is entitled to special education services. As defined by IDEA Emotional Disturbance includes Schizophrenia but does not apply to children who are socially maladjusted unless it is determined that they have an emotional disturbance. [Code of Federal Regulation, Title 34, Section 300.7(c)(4)(ii)]

Evaluation

The definition of ED is not clear and changes from state to state in the USA. Since no specific diagnostic instruments are required to qualify for services, the diagnosis of precise conditions such as what is a *general* and *pervasive* mood of unhappiness or depression requires a highly trained specialist such as a psychiatrist or school psychologist. Students who exhibit dramatic externalizing behaviors (e.g., aggression) are more easily diagnosed, but to those who display internalizing behaviors (e.g., depression) are more difficult to identify. There are more essential criteria to label a student as emotionally disturbed. One, a student's behavior must

be unusual and problematic for his parents and/or teacher across settings. Moreover, at least two documented interventions must also show that the behavior is not amenable to change. Two, because of the complexity of these disorders, a multidisciplinary team approach is required made up of various members such as regular education teachers, special education teachers, principals, parents, and a psychologist.

Treatment

For educators, in order to decide what kind of special education services are suitable for eligible students who manifest an ED, a functional behavioral assessment is the most popular technique currently in use. Using a variety of data and strategies, educators are able to develop a comprehensive behavior intervention plan which usually has school and home components. Recent laws as well as research have suggested that only evidence-based interventions should be used (e.g., Traughber & D'Amato, 2005).

Intervention plans typically cover five distinct components – including cognitive (e.g., poor memory, short-attention span), academic (e.g., poor educational performance not matching cognitive abilities), physical (e.g., stomachache, absenteeism), behavioral (e.g., aggression, depression, compulsive behaviors, attention seeking), and communication (e.g., echolalia, illogical speech difficulties) areas. In addition, many students who manifest an ED also are treated using a psychopharmacological intervention.

Cross References

► Emotional and Behavioral Disorders
► Social Problems

References and Readings

Algozzine, R., & Ysseldyke, J. (2006). *Teaching students with emotional disturbance*. CA: Corwin.

Council for Exceptional Children (CEC) (2010). *Behavior disorders/emotional disturbances*. Retrieved January 15, 2010, from http://www.cec.sped.org

Department of Education (1999). 34 CFR Parts 300 and 303 Assistance to states for the education of children with disabilities and the early intervention program for infants and toddlers with disabilities. *Federal Register, 64*(48).

Forness, S. R., & Knitzer, J. (1992). A new proposed definition and terminology to replace "serious emotion disturbance" in individuals with disabilities act. *School Psychology Review, 21*(1), 12–20.

Hartlage, L. C., & D'Amato, R. C. (2008). Understanding the etiology of neurological and psychiatric disorders. In A. MacNeil Horton Jr., & D. Wedding (Eds.), *The neuropsychology handbook* (3rd ed., pp. 87–108). New York: Springer.

Individuals with Disabilities Education Act (2010). In *Wikipedia encyclopedia online*. Retrieved February 10, 2010, from http://en.wikipedia.org

Kutash, K., Duchnowski, A. J., & Friedman, R. M. (2005). The system of care twenty years later. In M. H. Epstein, K. Kutash, & A. J. Duchnowski (Eds.), *Outcomes for children with emotional and behavioral disorders and their families: Program and evaluation best practices* (2nd ed., pp. 3–22). Austin, TX: Pro-Ed.

Miller, M. A., Fellbaum, C., Tengi, R., & Langone, H. (n.d.). *Emotional disturbance*. Retrieved January 20, 2010, from http://wordnetweb.princeton.edu

NICHCY (2004). Disability fact sheet No.5 – Emotional disturbance. *National Dissemination Center for Children with Disabilities*. Retrieved January 20, 2010, from http://www.nichcy.org

Traughber, M. C., & D'Amato, R. C. (2005). Integrating evidence-based neuropsychological services into school settings: Issues and challenges for the future. In R. C. D'Amato, E. Fletcher-Janzen, & C. R. Reynolds (Eds.), *Handbook of school neuropsychology* (pp. 827–858). New York: Wiley.

Emotional Incontinence

► Emotional Lability

Emotional Intelligence

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Synonyms

Emotion; Intelligence

Definition

Emotional intelligence (EI) is defined as "the ability to carry out accurate reasoning about emotions and the ability to use emotions and emotional knowledge to enhance thought" (Mayer, Roberts, & Barsade, 2007).

Historical Background

Intelligence in regards to emotions was discussed by Wechsler in the 1940s as the "aggregate or global capacity of the individual to act purposefully, to think rationally and to deal effectively with his environment" (Mayer, Salovey, & Caruso, 2000). Piaget also mentioned emotional intelligence within his stage model of development. Psychologists felt that intellect incorporated more than just knowledge and expressed interest in multiple intelligences by the 1980s. Studies by John Mayer and Peter Salovey began theoretical research on emotional intelligence in the early 1990s. During this decade, Goleman (1995) published the book *Emotional Intelligence* which popularized the term and it received national media attention. Daniel Goleman made strong claims that emotional intelligence made major contributions to individuals and society. This claim evoked controversy among other EI researchers. Also during this time early measures of EI were developed. Emotional Intelligence is now an active research area in psychology, business, and education.

Current Knowledge

Theorists believe that EI is distinct from other cognitive mental processes because it involves a primary focus on a more specific area of problem solving (Mayer et al., 2007). Emotional intelligence theorists have focused their research on emotional ability itself and attempted to quantify EI as a separate processing domain from other intelligences with tests. Most of the current research has focused on three approaches (specific-ability, integrative-models, and mixed models) to explain emotional intelligence along with their theoretical and methodological perspectives (Mayer et al., 2007). Specific-ability approach focuses on particular skill(s) that can be considered essential to EI. These skills and abilities are accuracy in emotional perception, ways in which emotions facilitate thinking, emotional reasoning and understanding, and emotional self-management. The second approach, the integrative-model, connects several specific abilities to obtain an overall EI, including Izard's Emotional Knowledge Approach (Izard et al., 2001) and the four-branch model (Mayer & Salovey, 1997) of EI. The final approach to explain EI is Mixed Model which uses broad definitions such as non-ability competencies, personality dispositions, and emotionally and socially intelligent behavior to describe EI (Mayer et al., 2007). Most measures under this category assess emotional intelligence attributes but interject other variables which are not considered part of EI. Many of the mixed-model methods are self-report scales and seem to assess personality traits rather than EI. Mayer et al. (2007) believe that a clearer approach to research would be to consider EI a discrete variable and then study its related variables. A major critique of mixed model scales suggests that they lack an EI definition that is consistent with scientific theory (Mayer et al., 2007).

Measurement of Emotional Intelligence

Measures of EI are based on specific-ability and integrative-model theories. The measures discussed are performance tests, rather than self-report measures, where subjects are asked to perform a specific skill (Mayer et al., 2007). Specific-ability measures include the Diagnostic Analysis of Nonverbal Accuracy 2 (DANVA2), the Japanese and Caucasian Brief Affect Recognition Test (JACBART), and the Levels of Emotional Awareness Scale (LEAS). The DANVA2 and JACBART include scales of emotional perception while the LEAS measures emotional understanding. Integrative-model measures include Izard's Emotional Knowledge Test (EKT; Izard et al., 2001) which provides an integrative measure of EI focusing on emotional perception and understanding. The Four-branch model connects abilities from the following areas: accurately perceiving emotions; using emotions to facilitate thought; understanding emotion; and managing emotions (Mayer & Salovey, 1997; Mayer, Salovey, Caruso, & Sitarenios, 2003). These abilities are measured by the Mayer–Salovey–Caruso Emotional Intelligence Test (MSCEIT) (Mayer, Salovey, & Caruso, 2002). Content and response-process validity were adequate for the Specific-ability and integrative-model measures (Mayer et al., 2007). Internal consistency reliabilities for the measures fall in the moderate-to-high range. The reliabilities for most full-scale measures are adequate for research and at the higher numbers, a reliable assessment of an individual (Mayer et al., 2007). Through a factor analysis study, a single, global EI factor was found to describe MSCEIT data (Mayer et al., 2003). Additionally, two factor models indicate loadings on Experiential and Strategic EI while three or four models emphasize Emotional Perception, Understanding, and Management. Factor analysis studies have suggested that EI measures outline recognizable factors making these findings consistent with a hierarchical view of intelligence (Mayer et al., 2007). However, convergent validity across EI measures is lacking and it continues to be a concern for EI researchers. Overall, research has shown that measures

of EI are relatively reliable and valid, but more research needs to be conducted.

Based on studies with EI measures, EI has been related to biopsychosocial processes, other related mental abilities, emotions and empathy, and personality traits. A study conducted by Reis et al. (2007) indicated that while solving MSCEIT problems the brain areas activated were the left frontal polar and left anterior temporal regions which are linked to social cooperation. Another study (Lane, Quinlan, Schwartz, Walker, & Zeitlin, 1998) with the LEAS indicated that subject with higher scores exhibited greater responsiveness to stimuli in area 24 of the anterior cingulate cortex, which is linked to emotional processing. Researchers have also found that individuals higher in EI utilize less brain activity to solve emotional problems (Jausovec & Jausovec, 2005). In addition, factor analysis studies indicate that EI seems to be related to socio-emotional reasoning but not to creativity (Mayer et al., 2007). Individuals with higher EI scores on several measures correlated with self-judgments of empathic feeling (Mayer et al., 2000). Scores from the MSCEIT Emotional Understanding show the strongest individual relationship with verbal/crystallized intelligence measures across many studies (Mayer et al., 2007).

Emotional Intelligence and Life Effects

Research on emotional intelligence demonstrates that it correlates with social relationships in individuals' environments, their performance in school and work settings, and their well-being. In childhood and adolescence, EI consistently predicts positive social and academic outcomes in children. Skills in emotional regulation and emotional knowledge also affect their well-being (Denham et al., 2003). Studies also suggest that adults with high EI are seen more positively and chosen as friends (Mayer et al., 2007). Negative correlations were found with EI and social deviance, critical remarks, destructive responses. In family and intimate relationships, research outcomes suggest that parental warmth and parental support were slightly correlated with some scales of EI. Several studies on intimate relations show a strong correlation between EI, relationship health and perceived quality of relationships. When comparing EI and academic performance, research shows very little correlation with academic achievement, especially when cognitive intelligence and personality measures are controlled. However, Izard et al. (2001) found that emotional knowledge in preschoolers did predict third grade teachers' ratings of academic aptitude. In the work settings, individuals with high EI scores correlate with successful problem solving, positive working relationships, workplace effectiveness and performance, higher wages, and effective leadership (Mayer et al., 2007). Gender differences in emotional abilities have been shown in many of the aforementioned studies. Emotional Intelligence is correlated with positive life satisfaction, higher self-esteem, and lower evaluations of depression.

Future Directions

Further research should be conducted to determine if emotional and social intelligences could be included in the taxonomy of mental abilities (Mayer et al., 2007). Additional empirical studies could be conducted to determine if emotional intelligence predicts a wider range of school and work outcomes than discussed above. Further meta-analyses of effects of EI, focusing in particular on the correlates of measures based on specific-ability and integrative-model approaches need to be conducted. Additional research should be performed on gender differences in EI. The effects of teaching emotional knowledge and reasoning in school, work, and home settings should be evaluated. Since EI research has only been conducted over the last 2 decades, information gathered from future research will continue to direct emotional intelligence theory and measurement.

References and Readings

Denham, S. A., Blair, K. A., DeMulder, E., Levitas, J., Sawyer, K., & Auerbach-Major, S. (2003). Preschool emotional competence: Pathway to social competence. *Child Development, 74*, 238–256.

Goleman, D. (1995). *Emotional intelligence.* New York: Bantam.

Izard, C. E., Fine, S., Schultz, D., Mostow, A. J., Ackerman, B., & Youngstrom, E. (2001). Emotion knowledge as a predictor of social behavior and academic competence in children at risk. *Psychological Science, 12*, 18–23.

Jausovec, N., & Jausovec, K. (2005). Differences in induced gamma and upper alpha oscillations in the human brain related to verbal/performance and emotional intelligence. *International Journal of Psychophysiology, 56*, 223–235.

Lane, R. D., Quinlan, D. M., Schwartz, G. E., Walker, P. A., & Zeitlin, S. B. (1998). Neural correlates of levels of emotional awareness: Evidence of an interaction between emotion and attention in the anterior cingulate cortex. *Journal of Cognitive Neuroscience, 10*, 525–535.

Mayer, J. D., Roberts, R. D., & Barsade, S. G. (2007). Human abilities: Emotional intelligence. *Annual Review of Psychology, 59*, 507–536.

Mayer, J. D., & Salovey, P. (1997). What is emotional intelligence? In P. Salovey & D. Sluyter (Eds.), *Emotional development and emotional intelligence: Educational implications* (pp. 3–31). New York: Basic Books.

Mayer, J. D., Salovey, P., & Caruso, D. (2000). Models of emotional intelligence. In R. Sternberg (Ed.), *Handbook of intelligence* (pp. 396–420). Cambridge, UK: Cambridge University Press.

Mayer, J. D., Salovey, P., & Caruso, D. (2002). *Mayer-Salovey-Caruso emotional intelligence test (MSCEIT) user's manual.* Toronto, ON: MHS Publication.

Mayer, J. D., Salovey, P., Caruso, D., & Sitarenios, G. (2003). Measuring emotional intelligence with the MSCEIT V2.0. *Emotion, 3*, 97–105.

Reis, D. L., Brackett, M. A., Shamosh, N. A., Kent, A. K., Salovey, P., & Gray, J. R. (2007). Emotional intelligence predicts individual differences in social exchange reasoning. *NeuroImage, 35*, 1385–1391.

Emotional Lability

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Synonyms

Emotional incontinence

Definition

Emotional lability is defined as rapid emotional change, most often associated with changes in affect. Lability is observed conditions such as CVA, traumatic brain injury, senile dementia, schizophrenia, bipolar disorder, or borderline personality disorder. Treatment with various antidepressant medications appears to be the most effective intervention.

Cross References

► Pseudobulbar Palsy
► Traumatic Brain Injury

References and Readings

Caplan, B. (2009). Rehabilitation psychology and neuropsychology with stroke survivors. In R. G. Frank, M. Rosenthal, & B. Caplan, *Handbook of rehabilitation psychology, 2nd Edition* (pp. 63–94). Washington, D.C.: American Psychological Association.

Morris, P., Robinson, R. G., & Raphael, B. (1993). Emotional lability after stroke. *Australian and New Zealand Journal of Psychiatry, 27*(4), 601–605.

Silver, J., Arciniegas, D., & Yudovsky, S. (2005). Psychopharmacology. In J. Silver, T. McAllister, & S. Yudovsky (Eds.), *Textbook of traumatic brain injury.* Arlington, VA: American Psychiatric.

Emotionality

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Definition

Emotionality includes a variety of subjective feeling states that predictably influence observable behavior and physiological responses for functional purposes related to adaptation. Emotions typically involve multiple components including autonomic, hormonal, behavioral, and cognitive component. Physiological signs of emotions may include change in autonomic nervous system activity which includes changes in heart rate, muscle tension, perspiration, and metabolic changes. Ekman's research on the cross-cultural invariance of emotional identification and expression is suggestive of emotions as a species-typical response. The coordination of the emotional state is facilitated by the amygdala. The amygdala, a cluster of nuclei in the limbic system near the temporal lobes, has been found to be important for eliciting a cascade of physiological changes involved in emotional behavior.

Various theories of emotions have been proposed. The James–Lange theory postulates that emotions are the by-product of the physiological changes occurring in the body. In contrast, the Cannon–Bard theory proposed that emotional experience precedes the physiological responses corresponding to emotions. Most contemporary models of emotion provide a synthesis of these two views that include the interacting influence of both cognitive appraisal and physiological events.

Assessment of emotional behavior is of critical importance in clinical neuropsychology as many emotional behaviors are symptomatic of clinical conditions. The Diagnostic and Statistics Manual of Mental Disorders Fourth Edition (DSM-IV) includes emotional behaviors or symptoms as part of many disorders including anxiety, depression, schizoaffective, and a variety of other disorders. A variety of instruments are available to screen or assess the emotional symptoms of particular disorders, such as the Beck Depression Inventory, or to widely screen multiple disorders, such as the Emotional Status Exam of the Dean–Woodcock.

Cross References

► Emotion
► Emotional Intelligence

References and Readings

Dean, R. S., & Woodcock, R. W. (2003). *Dean-Woodcock neuropsychological battery.* Itasca, IL: Riverside Publishing.

Ekman, P. (1980). *The face of man: Expressions of universal emotions in a New Guinea Village.* New York: Garland STPM Press.

Emphysema

► Chronic Obstructive Pulmonary Disease

Employment Coach

► Employment Specialist

Employment Facilitation

► Job Advocacy

Employment Specialist

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Synonyms

Employment coach; Job coach; Job development specialist; Job placement specialist; Vocational specialist; Work specialist

Definition

A provider or facilitator of services in the work context and community to locate and secure support options for persons with disabilities for the purpose of optimizing full career potential. Employment specialists function as a consultant and work in partnership with others to build on existing supports or develop new supports that meet individualized needs in maximizing career potential.

Employment specialists are primarily responsible for providing or securing an assessment of the person with the disability, job development, job placement, job-site training, and ongoing follow along services. The goal of an employment specialist is to train the person with the disability to perform and be successful on the job, then gradually fade out of the picture over time. Employment specialists have the ultimate goal of assisting individuals with disabilities to obtain and succeed in work settings of the person's choice.

Cross References

► Job Coach
► Supported Employment

References and Readings

Blanck, P. D. (2000). *Employment, disability, and the Americans with Disabilities Act: Issues in law, public policy, and research.* Evanston: Northwestern University Press, Inc.

Griffin, C., Hammis, D., & Geary, T. (2007). *The job developer's handbook: Practical tactics for customized employment.* Baltimore: Paul H. Brookes Publishing Co., Inc.

Unger, D., Kregel, J., Wehman, P., & Brooke, V. (2002). *Employers' views of workplace supports: VCU charter business roundtable's national study of employers' experiences with workers with disabilities.* Richmond: VCU RRTC on Workplace Supports and Job Retention Monograph.

Wehman, P., Inge, K. J., Revell, W. G., & Brooke, V. A. (2006). *Real work for real pay: Inclusive employment for people with disabilities.* Baltimore: Paul H. Brookes Publishing Co., Inc.

Encephalitis (Viral)

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Synonyms

Herpes simplex encephalitis; Meningoencephalitis

*The authors are not related by blood or marriage.

Short Description

Viral encephalitis is a condition in which a virus infects the brain, causing immune mediated inflammatory changes and destruction of gray matter that is usually accompanied by inflammation of the adjacent meninges. (Anderson, 2001; Boos & Esiri, 2003; Halperin, 2007; Nath & Berger, 2000; Roos & Tyler, 2008). Viral encephalitis sometimes causes irreversible impairments in brain function. Therefore, it is critical to reach the appropriate diagnosis and begin treatment in a timely manner.

Categorization

Viral encephalitis is categorized as (1) acute, (2) subacute/chronic, or (3) parainfectious (Boos & Esiri, 2003). Symptom profiles that deteriorate rapidly over a period of two to three days show an acute onset and require immediate medical intervention. Herpes Simplex Virus (HSV) is the most common cause of viral encephalitis and follows this temporal pattern (Anderson, 2001).

By contrast, symptoms that develop over a period of weeks or months are characteristic of subacute/chronic disease processes, such as HIV/AIDS and the prion disorder – Creutzfeldt-Jakob disease. Parainfectious or post-vaccination encephalitis is a complication that occurs following a systemic viral infection (e.g., measles), non-specific febrile illness, or antigen challenge (i.e., immunizations). Individuals often report an asymptomatic period prior to the onset of neurological symptoms which may affect multiple regions of the central nervous system (CNS).

Epidemiology

Encephalitis can be sporadic or epidemic, and differences in epidemiological and etiological characteristics further help define the various categories of encephalitis (Anderson, 2001; Boos & Esiri, 2003). The clustering of similar cases, which may range from pairs into hundreds of persons, indicates a common transmission vector and points toward particular viral agents (Boos & Esiri, 2003). Transmission mechanisms in epidemics can involve human-to-human transmission, as with influenza and the enteroviruses; insect-to-human transmission, as is the case with arboviruses; and animal-to-human transmission, as seen in Nipah virus encephalitis (Boos & Esiri, 2003).

Encephalitis may be a secondary CNS complication in cases where the primary epidemic infection initially targeted other organs (Anderson, 2001). Therefore, encephalitis falls into categories and subcategories that are characterized by different viruses and underlying disease processes, including acute sporadic, acute epidemic, subacute/chronic sporadic, and subacute/chronic epidemic. Herpes Simplex encephalitis (HSE) is an example of acute sporadic encephalitis, and the AIDS dementia complex is the most frequent form of chronic epidemic encephalitis (Boos & Esiri, 2003).

A variety of viral pathogens can invade the CNS and produce encephalitis (see Tables 1 and 2). Often disabling or fatal, the most common cause of acute sporadic viral encephalitis worldwide is the herpes simplex virus (Anderson, 2001; Halperin, 2007). It accounts for 10–20% of viral encephalitis cases in the US and Europe with an estimated prevalence of 0.1–0.4 per 100,000 population per year (Anderson, 2001).

Epidemics of viral encephalitis in the US are usually caused by the arboviruses, or arthropod borne viruses, whose principal vector is the mosquito (see Table 2). Within the US, West Nile Virus (WNV) recently became the most common form of vector-borne epidemic encephalitis (Halperin, 2007). The Centers for Disease Control (CDC) reported 687 cases of WNV encephalitis in 2008, with 1,356 total cases of WNV infection resulting in approximately 44 fatalities (CDC, 2009). In addition to WNV, there are four seasonally active arboviruses that are geographically specific and account for most other

Encephalitis (Viral). Table 1 Non-arthropod borne viruses associated with encephalitis

Type	Virus
DNA viruses	
Herpesviruses	Herpes simplex type 1, herpes simplex type 2, varicella-zoster, cytomegalovirus, epstein-barr virus
Adenoviruses	Adenovirus types 6, 7, 11, 12
RNA viruses	
Retroviruses	Human immunodeficiency virus type 1
Enteroviruses	Polioviruses, coxsackieviruses group A, coxsackieviruses group- B, echoviruses, enteroviruses 70, 71
Rubivirus	Rubella
Arenavirus	Lymphocytic choriomeningitis
Paramyxoviruses	Measles, mumps
Orthomyxoviruses	Influenza, parainfluenza
Rhabdovirus	Rabies

Encephalitis (Viral). Table 2 Arthropod-borne[a] viruses associated with encephalitis

Type	Virus
RNA viruses	
Bunyaviruses	California encephalitis, jamestown canyon virus, laCrosse- virus, rift valley fever, snowshoe hare virus (Canada)
Flaviviruses	
Mosquito-borne	West nile virus, Japanese encephalitis (Asia), St. Louis encephalitis, Murray valley encephalitis (Australia, New Guinea)
Tick-borne	Powassan virus, central European encephalitis, Russian spring-summer encephalitis, louping Ill (British islands), Kyassanur forest virus (India)
Orbivirus	Colorado tick fever
Togaviruses	Venezuelan equine encephalitis, western equine encephalitis, eastern equine encephalitis

[a]Principal transmission vectors are ticks or mosquitoes

cases of encephalitis in the US: The Eastern Equine, Western Equine, St. Louis, and LaCrosse Viruses (Anderson, 2001; Halperin, 2007). The Powassan virus is present in the US and causes a small number of cases in the Northeast annually.

The Japanese arbovirus is the most common cause of epidemic viral encephalitis worldwide. Clusters of infection occur in Eastern Asia from Siberia to India. In Central and South America, the Venezuelan equine virus is the major pathogen. The most common arboviruses in Europe are principally transmitted via ticks during the spring and summer months. The Russian Spring-Summer and Central European viral strains cause the greatest number of human cases of encephalitis, with incidences ranging from 0.9 to 72.5 per 100,000 depending on location.

Natural History, Prognostic Factors, Outcomes

The first accounts of inflammatory changes in the brain were reported during the nineteenth century (Boos & Esiri, 2003). It was not until the epidemic of encephalitis lethargica during World War I that detailed case reports noting pathology were recorded. Knowledge of the pathological features associated with encephalitis is based upon examination of the brain at autopsy, neuroimaging studies, electroencephalography (EEG), and studies of brain biopsy specimen (Boos & Esiri, 2003).

The onset and progression of viral encephalitis is most commonly acute, with encephalitic symptoms usually developing within a 24 h period following a non-specific febrile prodrome (Anderson, 2001; Boos & Esiri, 2003; Halperin, 2007). In most cases, virus enters the body through the skin (i.e., insect bite, abrasion) or is transmitted via the mucosal, respiratory or gastrointestinal route (Anderson, 2001; Halperin, 2007). Virus replicates at the entry site and travels to the blood, causing a systemic viremia. Thereafter, a secondary viremia ensues as the virus concentrates in various locations such as the lymph tissue, liver, and spleen. The secondary viremia produces a quantity of virus sufficient to breach the vessels of the choroid plexus, the meninges and the brain (Anderson, 2001). Thus, with the exception of rabies virus and neurotropic herpes viruses, virus reaches the CNS hematogenously by passing through the endothelium of the blood-brain barrier (Nath & Berger, 2000). By contrast, the herpes viruses and rabies spread centripetally from the site of infection through the sensory and cranial nerves via axonal transport to the CNS (Anderson, 2001; Halperin, 2007).

Adults and older children usually present with symptoms of an acute febrile illness, with evidence of some meningeal inflammation, abnormal mental state, an altered level of consciousness, and either focal or diffuse neurologic signs (Roos & Tyler, 2008). Evidence of systemic infection (i.e., fever, leukocytosis) is not always present, especially in older or immunocompromised individuals (Halperin, 2007). The inclusion of encephalitis in the differential diagnosis will depend upon clinical evidence of brain dysfunction (Halperin, 2007). The generalized or focal neurologic aberrations of encephalitis may be stationary, progressive, or fluctuating, with neurologic signs reflecting the location(s) of the infection and inflammation (Roos & Tyler, 2008).

Both focal and generalized seizures occur in more than 50% of patients and may be difficult to control (Nath & Berger, 2000; Roos & Tyler, 2008). The most commonly observed focal symptoms are ataxia, aphasia, hemiparesis, cranial nerve deficits, and involuntary movements (Roos & Tyler, 2008). In some cases, involvement of the hypothalamic-pituitary axis may result in hypothermia or hyperthermia, diabetes insipidus, or autonomic dysfunction (Nath & Berger, 2000; Roos & Tyler, 2008). Level of consciousness may range from mild lethargy to deep coma (Anderson, 2001; Boos & Esiri, 2003; Halperin, 2007; Roos & Tyler, 2008).

Neuropsychological and Psychological Outcomes

Neuropsychological

Viral Encephalitis can include motor deficits, aphasia, amnesia, global cognitive decline, and epilepsy (Hokkanen & Launes, 2007). Due to the effects of the virus in the temporal lobes, the most commonly reported neuropsychological impairments, which may occur together or in isolation, are anterograde and retrograde amnesia (Hokkanen & Launes, 2007; Pewter, Williams, Haslam, & Kay, 2007). The retrograde amnesia can include impairments in semantic or episodic memory and in the ability to recall names, faces, and episodes involving familiar or famous people. Less widespread damage to the temporal lobes and fronto-basal areas may result in less severe amnestic disorders (Hokkanen & Launes, 2007).

While not typical in HSE, isolated amnesia or category specific anomia has been reported (Hokkanen & Launes, 2007). In addition to aphasia, anomia (i.e., encompassing visual identification, drawing, or sorting of pictures which may suggest widespread loss of semantic knowledge), executive dysfunction (e.g., the Stroop task, letter fluency, card sorting tasks and the Cognitive Estimations Test), and impairments in visual perception (Hokkanen & Launes, 2007; Pewter, Williams, Haslam, & Kay, 2007) have also been reported. Cognitive impairments may remain stable or diminish over time. However, some HSE patients may show a gradual, global cognitive deterioration (Hokkanen & Launes, 2007). Another common sequel of encephalitis is epilepsy, which when accompanied by frequent seizure activity may have a detrimental effect on cognitive functioning, with memory performance particularly vulnerable due to neuronal loss in hippocampal regions (Hokkanen & Launes, 2007).

In general, the cognitive deficits in non-HSE encephalitis are less frequent as well as less severe than those in HSE (Hokkanen & Launes, 2007). However, while other forms of encephalitis have received less thorough neuropsychological investigation, cognitive impairments have been reported following herpes simplex virus type 2, St Louis encephalitis, influenza virus types A and B, and Japanese encephalitis (Pewter, Williams, Haslam, & Kay, 2007).

Psychological

The patient's mental state is frequently confused and disoriented with neuropsychiatric symptoms that may include agitation, emotional lability, behavioral disorders, personality changes, hallucinations, and frank psychosis (Roos & Tyler, 2008). In HSE, psychiatric and behavioral symptoms have been reported to precede, accompany, and follow the acute illness (Hokkanen & Launes, 2007). In some patients, the altered behavior and psychotic episodes are first observed long before the onset of neurological symptoms, which may delay the process of arriving at the correct diagnosis (Hokkanen & Launes, 2007). Following treatment with acyclovir, 40–60% of the HSE survivors demonstrate persistent personality and behavioral abnormalities and instability, but the emotional symptoms appear to be milder (Hokkanen & Launes, 2007). Common symptoms include panic and anxiety disorders, phobic anxiety, major affective disorders (e.g., bipolar), manic behavior, aggressive outbursts, irritability, depression, and obsessive-compulsive behaviors (Hokkanen & Launes, 2007; Pewter, Williams, Haslam, & Kay, 2007).

Single-case studies have reported more severe cases with schizophreniform disorder (Pewter, Williams, Haslam, & Kay, 2007). The medical literature suggesting encephalitis as a cause of secondary psychosis appears to consist primarily of case reports or anecdotal observations, with the only reliable finding of psychosis occurring during the degenerative process of chronic HIV encephalitis (Pewter, Williams, Haslam, & Kay, 2007).

While psychiatric symptoms may reflect an emotional reaction to a possibly fatal illness, damage to the limbic system and amygdalo-frontal pathways may be key etiological factors (Hokkanen & Launes, 2007). The neuropsychiatric symptoms arising from limbic lobe involvement are well-known and can include emotional or mood disorders (e.g., rage, aggression, depression and mania), delusions, hallucinations, anxiety and dissociative disorders as well as sexuality changes, and hyperoral behaviors (Hokkanen & Launes, 2007).

Prognosis

The prognosis for encephalitis depends upon the strain of virus, the degree to which level of consciousness is altered, and the age of the patient (Boos & Esiri, 2003; Nath & Berger, 2000; Roos & Tyler, 2008). Arbovirus-associated encephalitis has a variable mortality rate. Eastern equine encephalitis is the most virulent strain and thus has the highest mortality rates. California virus encephalitis has the lowest mortality rate. Among the population, children younger than age 4 and the elderly have the highest mortality rates. Encephalitis caused by eastern, western, and St. Louis viruses has a comparatively high rate of neurologic sequelae (Roos & Tyler, 2008). Approximately 80% of survivors of Eastern equine encephalitis have severe neurologic deficits (Roos & Tyler, 2008). However, Epstein-Barr viral encephalitis, California encephalitis virus, and Venezuelan equine encephalitis rarely result in

sequelae (Roos & Tyler, 2008). Patients who present with severe neurologic impairments (Glasgow coma score ≤ 6) either die or survive with severe sequelae. Younger patients ($\leq$30), with a relatively low degree of altered consciousness at the initiation of therapy, do significantly better (100% survival, 62% with no or mild sequelae) than older ($\geq$30) patients (64% survival, 57% with no or mild sequelae) (Roos & Tyler, 2008). Overall, better long-term outcomes in HSE are associated with right-lateralized effects (Laurent et al., 1990).

Evaluation

Clinical evidence of both inflammation and of brain involvement is required for the diagnosis (Halperin, 2007). Diagnosis requires an explanation of the patient's systemic inflammatory response (if present) and encephalopathy by systematically excluding infectious and noninfectious mimics of encephalitis (Boos & Esiri, 2003; Halperin, 2007; Roos & Tyler, 2008). Differential diagnosis is complicated by the fact that other illnesses may produce the symptoms of viral encephalitis, such as the vascular diseases, tumors, abscess, fungal, parasitic, rickettsial and tuberculous infections, Reye's syndrome, toxic encephalopathy, subdural hematoma, and systemic lupus erythematosus (Roos & Tyler, 2008). The main diagnostic priority is to identify treatable causes of acute encephalitis, and determination of etiology is also essential from prognostic, therapeutic, and public health perspectives (Halperin, 2007; Roos & Tyler, 2008). The three treatable agents responsible for encephalitis are HSV-1, HSV-2, and varicella-zoster virus (Halperin, 2007).

A careful epidemiological investigation is part of the case history and may help provide a focus for the clinical and laboratory assessments (Boos & Esiri, 2003; Roos & Tyler, 2008). Consideration should be given to the possibility of exposure to infectious diseases at home, work, and during travel and to season of the year, patient age, geographic region, and possible exposure to animal or insect bites. If viral encephalitis or other parenchymal brain disease is suspected, the diagnostic approach to the patient will include neuroimaging, lumbar puncture, cerebrospinal fluid (CSF) examination, serological studies, electroencephalography (EEG), and (rarely) brain biopsy (Boos & Esiri, 2003; Halperin, 2007; Roos & Tyler, 2008).

Computed Tomography (CT) may be used to identify mass effect, but MRI using contrast agents is more sensitive in demonstrating changes in cerebral edema, white matter disturbances, infarction, and blood-brain barrier irregularities (Anderson, 2001; Halperin, 2007). ICP monitoring should be implemented if the Glasgow Coma Scale (GCS) is ≤ 8, or if there is imaging evidence of significant cerebral edema that cannot be controlled (Anderson, 2001; Boos & Esiri, 2003; Halperin, 2007). If imaging demonstrates a lack of posterior fossa or supratentorial mass effect and intracranial pressure is not dangerously elevated, lumbar puncture can be performed with minimal risk of herniation (Anderson, 2001; Halperin, 2007; Roos & Tyler, 2008).

The characteristic encephalitic CSF findings are a lymphocytic pleocytosis (10–1,000 cells/mm^3), a slightly elevated protein level, and normal glucose level (Anderson, 2001; Boos & Esiri, 2003; Halperin, 2007; Nath & Berger, 2000; Roos & Tyler, 2008). Early examination may show polymorphonuclear leukocytes, and around 20% of patients will have a significant number of red blood cells (>500/L) in the CSF (Anderson, 2001; Nath & Berger, 2000; Roos & Tyler, 2008). Bacterial cultures and polymerase chain reaction (PCR), along with CSF and serum antibody estimation, should be performed for herpes and other etiologic agents as deemed necessary based upon case history and epidemiologic factors (Anderson, 2001; Boos & Esiri, 2003; Halperin, 2007; Nath & Berger, 2000; Roos & Tyler, 2008).

EEG is used infrequently as a diagnostic tool. However, it can help corroborate the Encephalitis diagnosis. Patients often exhibit an abnormal EEG, with diffuse slow wave activity that is directly related to the severity of the infection (Anderson, 2001). Herpes simplex encephalitis has a somewhat characteristic EEG profile involving abnormal activity from a predominantly temporal focus that may include spike and slow wave activity, and periodic lateralized epileptiform discharges (Anderson, 2001; Boos & Esiri, 2003; Halperin, 2007; Nath & Berger, 2000; Roos & Tyler, 2008).

The need for brain biopsy to definitively diagnose encephalitis has greatly declined with the development and availability of CSF PCR amplification techniques for HSV and other viruses (Boos & Esiri, 2003; Roos & Tyler, 2008). However, biopsy is considered when clinical and diagnostic studies are inconclusive and/or suggest the possibility of another illness (Boos & Esiri, 2003).

Treatment

Early stage severe encephalitis may require the resources of an intensive care unit (Anderson, 2001; Boos & Esiri, 2003; Halperin, 2007; Nath & Berger, 2000; Roos & Tyler, 2008). As with any delirious or unconscious patient, basic management and supportive therapy include monitoring and maintaining satisfactory nutrition, hydration,

electrolyte balance, respiration, blood pressure, and body temperature (Anderson, 2001; Boos & Esiri, 2003; Halperin, 2007; Nath & Berger, 2000; Roos & Tyler, 2008).

Because of the risk of raised intracranial pressure (ICP), ICP must be repeatedly assessed to ensure proper brain perfusion and avoid secondary ischemia, infarction, and tissue shifting (Anderson, 2001; Boos & Esiri, 2003; Halperin, 2007; Roos & Tyler, 2008). The typical first line treatment for increased ICP is administration of Mannitol (1–2 g/kg) in a 20% solution (Halperin, 2007).

Seizures, which develop in approximately 50% of severe cases (Roos & Tyler, 2008), can be controlled with anticonvulsants, and prophylactic therapy should be considered if ICP is elevated and/or if there is evidence of substantial cerebral swelling or mass effect (Anderson, 2001; Boos & Esiri, 2003; Halperin, 2007; Nath & Berger, 2000; Roos & Tyler, 2008). Drug therapy for viral encephalitis is limited to specific pathogens, but should be introduced as soon as a presumptive diagnosis is reached (Boos & Esiri, 2003; Griffin, 2000; Roos & Tyler, 2008). Acyclovir is an effective treatment for HSV encephalitis, as well as infections due to the Epstein-Barr and Varicella-Zoster viruses (Anderson, 2001; Boos & Esiri, 2003; Griffin, 2000; Halperin, 2007; Nath & Berger, 2000; Roos & Tyler, 2008). The recommended dosage is 10–15 mg/kg of acyclovir i.v. every 8 h for 14–21 days (Boos & Esiri, 2003; Griffin, 2000; Halperin, 2007; Roos & Tyler, 2008). The relatively alkaline pH of acyclovir may result in phlebitis (Roos & Tyler, 2008). Other side effects may include elevated creatinine levels, thrombocytopenia, gastrointestinal disturbances, and neurotoxicity (Roos & Tyler, 2008).

Ganciclovir and foscarnet have proven to be effective against cytomegalovirus infections of the CNS. The usual induction dose of ganciclovir is 5 mg/kg i.v. every 12 h, followed by a 5 mg/kg daily maintenance dose (Roos & Tyler, 2008). Foscarnet is administered at an induction dosage of 60 mg/kg i.v. every 8 h, switching to a maintenance dose of 60–120 mg/kg each day (Roos & Tyler, 2008).

Therapy of acute parainfectious encephalitis involves immunomodulating or immunosuppressive drug regimens (Boos & Esiri, 2003). The first line treatment is usually pulse steroids, which in non-responders may be followed by i.v. immunoglobulin (Ig), plasmapheresis, or repeat steroid treatment (Boos & Esiri, 2003). The neurological complications of HIV infection, categorized as a chronic encephalitic condition, are managed with antiretroviral treatments (Anderson, 2001; Boos & Esiri, 2003; Griffin, 2000; Halperin, 2007).

Encephalitis patients who are comatose should receive chest physical therapy and passive range of motion exercises (Halperin, 2007). Stretching exercises reduce the likelihood of contractures (Halperin, 2007). Neurorehabilitation can help mitigate impairments due to brain injury (Halperin, 2007). Speech therapy may be indicated in certain cases, and pharmacological intervention with a combination of zolpidem and selegiline or bromocriptine may be beneficial for abulic patients with severe catatonia and rigidity (Halperin, 2007).

Cross References

- ▶ Acquired Immunodeficiency Syndrome (AIDS)
- ▶ Brain Swelling
- ▶ Cerebral Edema
- ▶ Cerebral Perfusion Pressure
- ▶ Epstein-Barr Virus
- ▶ Intracranial Pressure
- ▶ Lumbar Puncture
- ▶ Mass Effect
- ▶ Meningitis
- ▶ Prion Disease

References and Readings

Anderson, M. (2001). Encephalitis and other brain infections. In M. Donaghy (Ed.), *Brain diseases of the nervous system* (pp. 1117–1180). New York: Oxford University Press.

Boos, J., & Esiri, M. M. (2003). *Viral encephalitis in humans.* Washington, DC: ASM Press.

Centers for Disease Control. (Apr. 2009). 2008 West Nile virus activity in the United States (Reported to CDC as of April 10, 2009). Retrieved July 5, 2009 from http://www.cdc.gov/ncidod/dvbid/westnile/surv & controlCaseCount08_detailed.htm

Griffin, D. E. (2000). Encephalitis, myelitis, and neuritis. In L. G. Mandell, J. E. Bennett, & R. Dolin (Eds.), *Principles and practice of infectious diseases* (pp. 1143). Philadelphia, PA: Churchill Livingstone.

Halperin, J. J. (Ed.). (2007). *Encephalitis: Diagnosis and treatment.* New York: Informa Healthcare.

Hokkanen, L., & Launes, J. (2007). Neuropsychological sequelae of acute-onset sporadic viral encephalitis. *Neuropsychological Rehabilitation, 17*(4/5), 450–477.

Laurent, B., Allegri, R. F., Michel, D., Trillet, M., Naegele-Faure, B., Foyatier, N., et al. (1990). Primarily unilateral herpes encephalitis. Long-term neuropsychological study of 9 cases. *Revue Neurologique (Paris), 146*, 671–681.

Nath, A., & Berger, J. R. (2000). Acute viral meningitis and encephalitis. In L. Goldman & J. C. Bennett (Eds.), *Cecil textbook of medicine* (pp. 2123–2126). Philadelphia, PA: W. B. Saunders.

Pewter, S. M., Williams, W. H., Haslam, C., & Kay, J. M. (2007). Neuropsychological and psychiatric profiles in acute encephalitis in adults. *Neuropsychological Rehabilitation, 17*(4/5), 478–505.

Roos, K. L., & Tyler, K. L. (2008). Meningitis, encephalitis, brain abscess and empyema. In B. Fauci, H. Kasper, J. Longo, & Loscalzo (Eds.), *Harrison's principles of internal medicine* (17th ed., pp. 2621–2640). New York: McGraw Hill.

Encephalopathy

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Synonyms

Brain disease

Definition

Encephalopathy is a nonspecific term that refers to diffuse dysfunction within the brain that causes disturbances in function and mental status. Encephalopathy can be associated with either morphological changes within the brain or metabolic imbalances. It originates from a variety of factors including, but not limited to, genetic susceptibility/mutation, traumatic brain injury, cerebrovascular accident, psychiatric disorders, toxic agents, and systemic disease. Encephalopathy can either be acute (i.e., sudden onset such as metabolic encephalopathy or traumatic brain injury), chronic (i.e., gradual deterioration such as dementia, HIV, or schizophrenia), or static (i.e., a nonprogressive abnormality such as mental retardation).

Cross References

- ▶ Alzheimer's Disease
- ▶ Binswanger's Disease
- ▶ Brain Tumor
- ▶ Brainstem Glioma
- ▶ Dementia
- ▶ Huntington's Disease
- ▶ Hypertensive Encephalopathy
- ▶ Parkinson's Disease
- ▶ Toxic-Metabolic Encephalopathy

References and Readings

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

Kumar, V., Fausto, N., & Abbas, A. (2004). *Robbins & Cotran pathologic basis of disease* (7th ed.). Philadelphia, PA: Saunders.

Encephalopathy of Prematurity

▶ Periventricular Leukomalacia

Endolymphatic Hydrops

▶ Ménière's Syndrome

Endothelial Proliferation

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Synonyms

Vascular endothelial proliferation

Definition

Endothelial proliferation is an increase in vascular endothelial cells needed for the growth of new or existing blood vessels. It is stimulated by solid tumors that need to generate blood vessels to continue growth. It contributes to angiogenesis in tumor formation (Louis & Cavenee, 2005), and results from overexpression of vascular endothelial growth factor (VEGF). Endothelial proliferation is one of the characteristics of malignancy in gliomas as defined by the World Health Organization's primary central nervous system tumor grading system (Kleihues, Burger & Scheithauer, 1993). Endothelial proliferation is associated with breakdown in the blood–brain barrier (Kracht et al., 2004). It is a feature of malignancy that indicates poor prognosis, but is considered in combination with other features which is not unique.

References and Readings

Kleihues, P., Burger, P. C., & Scheithauer, B. W. (1993). The new WHO classification of brain tumours. *Brain Pathology, 3*(3), 255–268.

Kracht, L. W., Miletic, H., Busch, S., Jacobs, A. H., Voges, J., Hoevels, M., et al. (2004). Delineation of brain tumor extent with [^{11}C]L-methionine positron emission tomography: Local comparison with stereotactic histopathology. *Clinical Cancer Research, 10*, 7163–7170.

Louis, D. N., & Cavenee, W. K. (2005). *Molecular biology of central nervous system tumors* (pp. http://brain.mgh.harvard.edu/MolecularGenetics.htm). Boston, MA: Massachusetts General Hospital, MGH Neurosurgical Service, Brain Tumor Center.

Endotracheal Tube

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Synonyms

Breathing tube; ETT

Definition

Endotracheal tube (ETT) is a tube placed into the trachea for mechanical ventilation and management of a patient's airway. The tube is placed in the patient's trachea to ensure a patent airway allowing oxygen, anesthetics, or gaseous medications to reach the lungs.

Current Knowledge

Intubation, usually performed with general anesthesia, is the process of inserting an ETT. A laryngoscope, which consists of a blade of varying shapes and sizes, a handle, and a light source, is an instrument used to help visualize the larynx and surrounding structures. A stylet, a malleable piece of metal placed inside the ETT, can also be used to aid intubation by providing stiffness and curvature at the end of the tube allowing for easier insertion. It is removed after intubation and the ETT is attached to a ventilator or self-inflating bag. Placement of the ETT is confirmed by auscultating both sides of the chest with a stethoscope.

Indications for ETT

Endotracheal intubation should be considered when establishment of a definitive airway is needed for a number of circumstances: respiratory arrest; respiratory failure; airway obstruction; inadequate air exchange; need for prolonged ventilatory support; severe flail chest or pulmonary contusion; multiple trauma, head injury, and abnormal mental status; status epilepticus; inhalation injury; protection from aspiration; patient unable to protect his airway; when other means of ventilation are not possible or not effective; or when undergoing general anesthesia.

Types

The different types of ETT are cuffed or uncuffed, oral or nasal, reinforced tubes, double lumen tubes, and tracheostomy tubes. The internal diameter of these tubes range from 2–10.5 mm and are chosen based on the patient's body size, with smaller sizes used in neonates and pediatric patients. While most tubes used are cuffed, uncuffed tubes are often used in pediatric patients under eight years of age. Nasal tubes are often utilized for intubation during faciomaxillary surgeries. Reinforced tubes are used when there is a concern that the ETT will be damaged during intubation. However, these tubes cannot be cut due to an internal metal ring. Also, these tubes often slide down the right main bronchus resulting in the ventilation of only one lung. Conversely, double-lumen endo-bronchial tubes are often used during thoracic surgery to preferentially ventilate one lung. The other lung is purposefully collapsed making it easier to operate on the deflated lung. A tracheostomy tube is a shortened tube inserted into the trachea through an opening in the neck.

Complications

During ETT intubation, trauma can occur to teeth, oropharynx, or vocal cords and may result in edema or bleeding. Also, if the ETT is misplaced deep into either the right of left main bronchus, only one lung will be adequately ventilated. This may result in a pneumothorax of the ventilated lung. Inadequate ventilation will also occur if the ETT is incorrectly placed in the esophagus. Additionally, aspiration of stomach contents can occur resulting in pneumonia or acute respiratory distress syndrome. Other rare but more severe complications include tracheal or esophageal perforation, spinal cord and vertebral column injury, cardiorespiratory arrest, brain damage, and death.

Cross References

▶ Polytrauma
▶ Traumatic Brain Injury

References and Readings

Divatia, J. V., & Bhowmick, K. (2005). Complications of endotracheal intubations and other airway management procedures. Indian Journal Anaesthesia, *49*(4), 308–318.

Finucane, B. T., & A. H. Santora. (2003). *Principles of airway management.* New York: Springer.
Stewart, C. E. (2002). *Advanced airway management.* Upper Saddle River, NJ: Prentice Hall Publishing.

Enhancement

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Definition

Enhancement is the neural process by which the intensity of focus is increased for specific locations to facilitate spatial-selective attention.

Current Knowledge

A relationship is known to exist between selective attention and an increase in the response of neurons in visual areas when a laboratory animal focuses on stimuli in the receptive field. This was first discovered for neurons in the superior colliculus of monkeys using electrophysiological methods (Goldberg & Wurtz, 1972), as neurons responded more intensely to the onset of a stimulus when saccadic movement to the stimulus was required, compared with when they maintained fixation. Subsequently, similar neuronal enhancement was demonstrated across a variety of other brain regions containing higher-order visual processing areas, including the inferior parietal and prefrontal cortex, though similar responses have also been found in other extrastriatal areas (e.g., V4) and even the primary visual cortex. These studies were considered to provide an essential foundation for explaining the neural mechanisms by which increased attention to a stimulus occurs triggered by initial stimulation, which results in an "enhanced" response to the stimulus and its spatial context. This model of visual-selective attention assumes a bottom-up approach, in which attention is driven by neuronal events caused by prior stimulation. Considerable visual neuroscience research has focused on the mechanisms that drive enhancement, and spatial, temporal, and other factors that constrain the response.

In studies conducted over the past decade, there has been increasing evidence that "top-down" processes may have greater influence on the neuronal response to novel stimuli rather than stimulus-evoked responses. Attentional biases created by constantly changing "salience maps" in the parietal lobes influence the probability of response to particular locations or features at the moment when the stimulus occurs (Gee, Ipata, Gottlieb, Bisley & Goldberg, 2008). Ultimately, visual-selective attention seems to be governed by enhancement occurring as a result of the preexisting response biases and the effects of cueing resulting from information associated with new stimuli as they are processed. Enhancement has direct implications for explaining the covert shifts of attention that have been the subject of extensive cognitive neuroscientific inquiry (Posner, Walker, Friedrich & Rafal, 1987).

Cross References

▶ Inferior Parietal Cortex
▶ Selective Attention

References and Readings

Gee, A. L., Ipata, A. E., Gottlieb, J., Bisley, J. W., & Goldberg, M. E. (2008). Neural enhancement and pre-emptive perception: the genesis of attention and the attentional maintenance of the cortical salience map. *Perception, 37*(3), 389–400.
Goldberg, M. E., & Wurtz, R. H. (1972). Activity of superior colliculus in behaving monkey. II. Effect of attention on neuronal responses. *Journal of Neurophysiology, 35*(4), 560–574.
Posner, M. I., Walker, J. A., Friedrich, F. A., & Rafal, R. D. (1987). How do the parietal lobes direct covert attention? *Neuropsychologia, 25*(1A), 135–145.

Environmental Adaptation

▶ Environmental Modifications

Environmental Dependency

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Synonyms

Imitation behavior; Pull to stimulus; Utilization behavior

Short Description or Definition

Environmental dependency refers to instances when the visual or visuo-tactile presence of objects "compels" patients to either grasp and/or use the objects.

Categorization

Environmental dependency is usually best understood as part of greater, superordinate frontal lobe syndrome.

Epidemiology

The incidence and prevalence of environmental dependency syndrome is unknown.

Natural History, Prognostic Factors, Outcomes

Environmental dependency syndrome or utilization behavior has not been extensively studied. Therefore, there is little information regarding natural history, prognostic factors, and outcomes.

Neuropsychology and Psychology of Environmental Dependency Syndrome

As noted above, environmental dependency refers to instances when the mere visual or visuo-tactile presence of objects "compels" patients to either grasp the object and/or use it. When patients with environmental dependency syndrome or utilization behavior are pulled to grasp and use objects, patients general show intact recognition of the object and its proper use. In this sense, environmental dependency or utilization behavior is not associated with an appreciative or associative agnosia.

Lhermitte (1983) noted that elements of an environmental dependency syndrome were first described by Wilson and Walshe (1914) and Adie and Critchley (1927). These authors described patients with "forced grasping" of objects in association with either left or right frontal lobe lesions. Denney-Brown (1958) is generally credited with coining the term *magnetic apraxia* to describe a complex manual response or activity where patients not only grasp but may also use objects correctly though in an inappropriate context. For example, presented with the examiners glasses and merely asked to name the object, a patient with *magnetic apraxia* may take the object from the examiner and put them on. Denney-Brown (1958) interpreted this phenomenon as representing a release behavior mediated by the parietal lobe due to the suppression of inhibitory mechanisms that would be otherwise governed by the frontal lobes.

Lhermitte (1983) described a series of patients who exhibited environmental dependency syndrome/utilization behavior. The aetiology of the underlying neurological condition was variable. However, when the examiner displayed common objects, all patients grasped the object from the examiner and would demonstrate its correct use. In this series of case studies, Lhermitte commented that other aspects of a frontal lobe syndrome were invariably present. In several patients, environmental dependency syndrome/utilization behavior was present acutely but then resolved. All patients described by Lhermitte (1983) presented with evidence of left or right frontal lobe lesions; however, he was reluctant to associate these behaviors to a precise locale within the frontal lobes solely on the basis of this handful of cases.

In addition to this series of case studies, Lhermitte (1983) described a formal examination procedure for the presence of environmental dependency/utilization behavior whereby the examiner sits in front of the patient with common objects. The first test condition is designed to establish a baseline for the presence of environmental dependency/utilization behavior, in that patients are shown objects and utilization behavior is observed. In the second test condition, patients are specifically told *not* to either grasp or use objects that are presented. If patients once again grasp the displayed objects in this second test condition, the examiner may consider the patient as having an environmental dependency/utilization behavior. In his 1983 report, Lhermitte describes testing a neurologically normal control group where neither test condition elicited this type of grasping behavior.

Better known, perhaps, are a series of two papers authored by Lhermitte and colleagues in 1986. In part one of this series, Lhermitte, Pillon, and Serdaru (1986) prospectively tested 75 patients who were divided into three groups presenting with focal lesions involving the frontal lobes, as well as non-focal frontal lesions: *group 1* demonstrated what Lhermitte et al. (1986) called imitation behavior. Using testing procedures described by Lhermitte (1983), these patients imitated the examiner's gestures despite being told not to do so; *group 2* demonstrated both imitation behavior and utilization behavior as described by Lhermitte (1983); *group 3* presented with

neither imitation nor utilization behavior. Neuropsychological and neurological assessment was obtained on all participants, as well as laboratory studies such as CT scans.

Among patients with focal lesions, neuropsychological testing found greater perseveration in groups 1 and 2 compared to group 3 on tests such as Luria's Graphical Sequences (see Luria, 1980, pp. 296–309; Goldberg, 1986; Lamar, Podell, Carew, Cloud, Kennedy, & Goldberg, 1997), Luria's three-step hand sequence (fist-palm-side; Luria, 1980), and the Wisconsin Card Sorting Test. No differences were noted when groups 1 and 2 were compared. Lhermitte et al., (1986) also commented on differences in behavior such that groups 1 and 2 exhibited greater indifference, disinterestedness, indifference to social rules, and apathy compared to group 3. Less specificity with respect to impairment on executive or frontal lobe tests was found in patients with nonfocal lesions. An analysis of CT scan found that "the lower half of the frontal lobe was affected in all patients" with less imitation and utilization behavior in "upper region" of the frontal lobes (p. 330). Also, few patients with posterior or retro-rolandic lesions exhibited either imitation or utilization behavior. Finally, imitation and utilization behavior could be found in patients with caudate and/or thalamic lesions.

Lhermitte et al., (1986) describe imitation behavior as a less severe presentation of either utilization behavior or an environmental dependency syndrome. The authors stress an "imbalance in patients between dependence on and independence from external stimuli, which leads them to become dependent on these stimuli. "The sight of an object implies the order to use it" (p. 331). Lhermitte et al., (1986) assert both imitation and utilization behavior can be present in patients with dementia, but that general intellectual deterioration fails to either predict the presence or severity of imitation or utilization behavior. These authors went on to describe several patients in whom imitation or utilization behavior could be elicited who were able to return to relatively normal activities.

Lhermitte (1986) studied in vivo two patients with utilization behavior/environmental dependency syndrome; that is, patients were studied in natural settings including a "doctor's office, a lecture room, a car, a garden, an apartment where various activities were possible, and a gift shop" (p. 335). Both patients had documented frontal lobe lesions. In this paper, Lhermitte (1986) accompanied both patients while they engaged in natural or normal daily activates. The degree each patient either imitated behavior or engaged in behavior that was inappropriate, but contextually consistent with their environment, is quite striking. Lhermitte's description of the behavior of his patients is quoted below:

> On the same day on which Patient 1 had walked around the apartment as if it were part of a museum, we returned to the bedroom. The bedspread had been taken off and the top sheet had been turned back in the usual way. When the patient saw this, he immediately began to get undressed. He got into bed, pulled the sheet up to his neck, and prepared to go to sleep. Later, when I picked up an article of clothing, the patient got up and dressed in an orderly fashion. (p. 339)
>
> As soon as Patient 2 saw the bed, she tucked in the covers on both sides. She did not get undressed. I walked toward the bed carrying my stethoscope. The patient lay down immediately. Realizing that the clothes were getting in my way, she helped me unbutton her blouse and undo her brassiere, so that her chest was completely bare. After this test, she accompanied me to a table where various items used for intramuscular injections were laid out. I went through the movements of preparing an injection. She immediately lifted her dress and pulled down her pantyhose to bare her right buttock. Later, I showed her the syringe. She took it. When I took off my jacket and shirt, she picked up the needle and a cotton ball, which she soaked in antiseptic, then bent down to my buttock to give the injection. (p. 340)

In describing this very striking behavior, Lhermitte (1986) stresses the environmental context in which behavior occurs. Patients are unable to desist or reframe from action, that is, objects in the environment and/or the context of the situation appears to 'demand' action on the part of the patient despite the inappropriate nature of their action or behavior. This is clearly indicated by patient 2 who, upon seeing a syringe and related equipment, prepares to administer an injection to Dr. Lhermitte.

Lhermitte (1983, 1986) and Lhermitte et al., (1986) clearly document an association between utilization behavior/environmental dependency syndrome and frontal lobe lesions. However, these behaviors have also been described in dementia and other medical/neurological conditions (Conchiglia, Della Rocca, & Grossi, 2007; Hoffmann, 2007; Hoffmann & Bill, 1992; Tanaka, Albert, Hara, Miyashita, & Kotani, 2000).

Evaluation

No formal, normative-based assessment is available for the presence and/or severity of environmental dependency syndrome.

Treatment

No formal treatment studies for environmental dependency are available.

Cross References

► Perseveration

References and Readings

Adie, W. J., & Critchley, M. (1927). Forced grasping and groping. *Brain, 50*, 142–170.

Conchiglia, G., Della Rocca, G., & Grossi, D. (2007). On a peculiar environmental dependency syndrome in a case with frontal–temporal damage: Zelig-like syndrome. *Neurocase, 13*, 1–5.

Denney-Brown, D. (1958). Nature of apraxia. *Journal of Nervous and Mental Disease, 126*, 9–32.

Goldberg, E. (1986). Varieties of perseveration: A comparison of two taxonomies. *Journal of Clinical and Experimental Neuropsychology, 8*, 710–726.

Hoffmann, M. (2007). Transient environmental dependency syndrome due to phendimetrazine tartrate. *European Neurology, 58*, 49–50.

Hoffmann, M. W., & Bill, P. L. (1992). The environmental dependency syndrome, imitation behaviour and utilisation behaviour as presenting symptoms of bilateral frontal lobe infarction due to moyamoya disease. *South African Medical Journal, 81*, 271–273.

Lamar, M., Podell, K., Carew, T. G., Cloud, B. S., Kennedy, C., & Goldberg, E. (1997). Perseverative behavior in Alzheimer's disease and subcortical ischaemic vascular dementia. *Neuropsychology, 11*, 523–534.

Lhermitte, F. (1983). 'Utilization behavior' and its relation to lesions of the frontal lobes. *Brain, 106*, 237–255.

Lhermitte, F., Pillon, B., & Serdaru, M. (1986). Human autonomy and the frontal lobes. Part I. Imitation and utilization behavior: A neuropsychological study of 75 patients. *Annals of Neurology, 19*, 326–334.

Lhermitte, F. (1986). Human autonomy and the frontal lobes. Part II. Patient behavior in complex and social situations: The "environmental dependency syndrome". *Annals of Neurology, 19*, 335–343.

Luria, A. R. (1980). *Higher cortical functions in man*. New York: Basic Books.

Paquier, C. R., & Assal, F. (2007). A case of oral spelling behavior: Another environmental dependency syndrome. *Cognitive and Behavioral Neurology, 20*, 235–237.

Tanaka, Y., Albert, M. L., Hara, H., Miyashita, T., & Kotani, N. (2000). Forced hyperphasia and environmental dependency syndrome. *Journal of Neurology, Neurosurgery, and Psychiatry, 68*, 224–226.

Wilson, S. A. K., & Walshe, F. M. R. (1914). The phenomenon of tonic innervation and its relation to motor apraxia. *Brain, 37*, 199–246.

Environmental Illness

► Multiple Chemical Sensitivity

Environmental Intervention

► Environmental Modifications

Environmental Modifications

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Synonyms

Environmental adaptation; Environmental intervention; Home modification

Definition

Environmental modifications (EM) entail the rearranging, rebuilding, or retrofitting of an existing space or building to make it accessible, usable, and safe either for a specific individual or for people with disabilities, in general. This type of modification can involve installing equipment, simple structural changes to a single room or significant alterations to an entire structure.

Current Knowledge

Modifications may be undertaken (1) out of an individual's desire to optimize mobility and independence; (2) family's concern about enhancing the safety of a physically or cognitively compromised individual; (3) under direction from a rehabilitation professional; or (4) under a legal mandate such as the American Disabilities Act. Whatever the case, EM can entail simple installation of prefabricated adaptive devices (durable medical equipment, DME) such as grab bars and a shower bench, significant structural changes such as adding a ramp or widening a doorway or a combination of the two. DME can be fabricated to an individual's needs or specifications but is much more commonly mass produced with limited capacity for adjustment. EM within one's own home or other permanent residence typically are most extensive. However, the principles and technical changes associated with home modification may be applied to the workplace and other places of public accommodation. Although restraints and other

devices that manage the mobility of ambulatory but cognitively compromised individuals (such as gates between rooms) typically are not considered as comparable to mobility-enhancing EM, the concept of restructuring a preexisting space for an individual's well being certainly extends to this class of modifications.

Goals for EM: Although a desire to optimize safety, mobility, and independence underpins most EM projects, there is considerable variability in the pragmatic goals of any given set of modifications. Such goals may include facilitating completion of basic activities of daily living (ADL) such as feeding, grooming, and toileting oneself. At higher levels, independent ADL's (IADL) such as meal preparation and household chores such as laundry may be integrated into planned EM. Finally, environmental modifications may be undertaken to facilitate community reentry and return to work, a class of adaptations that typically entails changes beyond the home.

Finances: Generally, a narrowly defined subset of DME is covered by Medicare, Medicaid, and many private insurance companies. Moreover, some DME companies have programs to provide this equipment to individuals with limited financial resources. Beyond DME, EM are not directly paid for by insurers. Although some State, Federal, and private assistance is available, the costs of EM typically are borne by the individual, rendering some types of EM inaccessible to significant numbers of people with disabilities.

Trend and Counter-trend: Historically, DME and mass produced accessibility hardware has been medical in appearance, utilitarian in design, and uniform in size and shape. However, with an aging population and assertive disability communities, the design and appearance of EM have become increasingly consumer driven and tailored for specific individuals, spaces, and tastes. In the United States, there has been a recent trend toward newly constructed housing for older adults and to a lesser extent, people with disabilities. These new structures are designed to be accessible from the outset thus obviating any need for modification in the future.

Cross References

► Community Reentry
► Discharge Planning

References and Readings

Fielo, S. B., & Warren, S.A. (2001). Home adaptation: Helping older people age in place. *Geriatric Nursing. 22*(5), 239–247.

Tse, T. (2005). The environment and falls prevention: Do environmental modifications make a difference? *Australian Occupational Therapy Journal. 52*, 271–281.

Enzyme

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Definition

Enzymes are protein molecules that are necessary to cellular processes and are potential pharmacological targets for the treatment of various diseases. The purpose of an enzyme is to act as a catalyst and lower the activation energy needed to carry out a biochemical reaction, and they often greatly increase the rate of a reaction as well. Most enzyme names have the suffix "-ase" attached to the substrate of the reaction or to describe the action performed. Enzymes assist in converting a given substrate into a different product. This process may result in breaking down an existing molecule (catabolism) into more basic components, or through synthesis, the combination and creation of a larger product (anabolism). When the substrate, and other molecules, interact with the enzyme at its active site, a conformational (or allosteric) change in the enzyme's shape may occur to influence (induce or inhibit) the activity of the enzyme. Factors potentially affecting enzyme specificity for a substrate include charge, shape, or hydrophilic qualities of both the enzyme and the complimentary substrate. Examples of enzymes that are targets of psychopharmacological agents include acetylcholinesterase and monoamine oxidase types A and B. In these examples, the goal of pharmacotherapy is to increase the synaptic concentration of the respective neurotransmitter that is naturally metabolized (broken down) by these enzymes. Enzyme inhibition may be either reversible or irreversible and either competitive or noncompetitive. The family of oxidative enzymes known as cytochrome P450 (e.g., CYP-2D6), which are a necessary part of phase I metabolism of many psychopharmacological agents, may be involved in drug–drug interactions when inhibited (e.g., fluoxetine) or induced (e.g., carbamazepine) by other pharmacological agents.

References and Readings

Brunton, L. L., Lazo, J. S., & Parker, K. L. (Eds.). (2006). *Goodman & Gilman's The pharmacological basis of therapeutics* (11th ed.). New York: McGraw-Hill.

Cooper, J. R., Bloom, F. E., & Roth, R. H. (Eds.). (2003). *The biochemical basis of neuropharmacology* (8th ed.). New York: Oxford University Press.
Davis, K. L., Charney, D., Coyle, J. T., & Nermeroff, C. (Eds.). (2002). *Neuropsychopharmacology: The fifth generation of progress.* Philadelphia, PA: Lippincott Williams & Wilkins.
Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd ed.). New York: Cambridge University Press.
Voet, D., Voet, J. G., & Pratt, C. W. (Eds.). (2008). *Fundamentals of biochemistry* (3rd ed.). New Jersey: Wiley.

EOWPVT

► Expressive One-Word Picture Vocabulary Test

Ependymoma

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Definition

Ependymomas are tumors that arise from the ependymal cells that line the ventricular system in the brain or spinal cord. There are four main types, classified according to tumor grade: myxopapillary ependymomas and subependymomas (grade I), ependymomas (grade II), and anaplastic ependymomas (grade III). Ependymomas are one of the most common types of tumors seen in children, especially those aged 3 years and younger. In children, ependymomas are usually located intracranially, primarily in the posterior fossa, while in adults, they are usually located spinally. Ependymomas are typically slow growing and the main symptoms are due primarily to raised intracranial pressure. A shunt may be necessary. Depending on the location, symptoms can include nausea, headaches, vomiting, papilledema, paresthesias, ataxia, spinal pain, and changes in mood and personality. Supratentorial ependymomas are often accompanied by hemiparesis, sensory disturbances, aphasia, and other types of cognitive impairment. Seizures and focal neurological deficits can also occur. Surgical intervention is usually first-line intervention, followed by radiotherapy. In young children, chemotherapy is the treatment of choice over radiotherapy because of the possible long-term effects of radiotherapy.

Ependymoma. Figure 1 Courtesy Michael Fisher, MD, Peter C. Phillips, MD. The Children's Hospital of Philadelphia

Cross References

► Brain Tumor
► Edema
► Neoplasms
► Posterior Fossa
► Radiotherapy
► Tumor Grade

References and Readings

Applegate, G. L., & Marymont, M. H. (1998). Intracranial Ependymomas: a review. *Cancer Investigations, 16*(8), 588–593.
Moynihan, T. J. (2003). Ependymal tumors. *Current Treatment Options in Oncology, 4*(6), 517–523.

EPI

► Eysenck Personality Inventory

Epicritic Pain

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Definition

A variant of *nocioceptive pain* (pain resulting from ongoing activation of primary afferent neurons by noxious stimuli), *epicritic pain* is transmitted to the spinal cord by A Delta (δ) fibers. These fibers are sparsely myelinated, large-diameter, and fast-conducting, which transmit sharp, well-localized pain. A δ fibers are mostly sensitive to mechanical and thermal stimuli. Epicritic pain is less responsive to opioid therapy than is *protopathic pain.*

Cross References

► Protopathic Pain

References and Readings

McCaffery, M., & Pasero, C. (1999). *Pain clinical manual* (2nd ed.). St. Louis: Mosby.

Epidemiology

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Definition

Epidemiology is a branch or subspecialty of medical studies and sciences that examines the incidence, causes, distribution, control, and transmission of a disease within a population of interest. It can also examine the genesis and developmental characteristics of a specific disease. For example, in examining the epidemiology of traumatic brain injury (TBI), one would expect to learn more about the incidence, prevalence, annual rates, risk factors (including sex, race, and age), and causes of TBI.

Cross References

► TBI

References and Readings

Centers for Disease Control and Prevention. (2007). *What is epidemiology?*. Retrieved December 26, 2007, from http://www.cdc.gov/malaria/distribution_epi/epidemiology.htm.

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

Epidural Hematoma

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Synonyms

EDH

Definition

Epidural hematoma is a traumatic accumulation of blood that forms following blunt force on the head (i.e., baseball bat, hammer). The force of impact is strong enough to tear the dural cover of the brain from the skull. This tearing of the dural cover disrupts the nearby arteries and veins, as well as dural branches of these blood vessels. One of the most common sites for epidural hematoma is the tempoparietal area due to skull fracture disrupting the middle meningeal artery or its dural branches. Motor vehicle accidents and physical assaults are common causes of epidural hematoma. Frequently neurosurgical intervention is required on an emergent basis to prevent an increase in intracranial pressure from rapidly accumulating blood. If intracranial pressure elevates too quickly or too high, the patient can become comatose. Most commonly, craniotomy with evacuation is used to intervene in cases of epidural hematoma. On some occasions, an epidural drain is placed to assist in the funneling off of extra fluid from the surface of the brain.

Cross References

► Cerebral Hemorrhage
► Skull Fracture

References and Readings

Ammerman, J. M., Jarrell, S. T., & Polin, R. S. (2006). Traumatic intracranial hemorrhage. In R. W. Evans (Eds.), *Neurology and trauma* (2nd ed., pp. 156–166). New York: Oxford University Press.

Epilepsy

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Synonyms

Cerebral seizures; Convulsive disorder; Seizure disorder

Short Description or Definition

Epilepsy is a common neurological disorder characterized by recurrent seizures of cerebral origin. Neither a specific disease nor a single syndrome, epilepsy encompasses a variety of symptom complexes arising from multifarious brain dysfunctions from various pathologic processes that produce seizures from a flurry of abnormal electrical activity in the brain. The hallmark of epilepsy is two or more unprovoked seizures (excluding e.g., seizures secondary to alcohol withdrawal) occurring more than 24 h apart. Seizures may be accompanied by a wide range of behavioral disturbances, and may or may not entail a loss of consciousness. They sometimes involve an "aura" (see entry for aura), a subjective sensation signaling the onset of a seizure that may include sensory experiences such as smells and visual distortions. The term aura, which is actually a delimited simple partial seizure, is attributed to the Roman physician Galen (130–200 AD) who reportedly overheard a boy say just before a seizure that he felt a cool breeze, "aura" in Latin (Bennett, 1992). There is a great deal of heterogeneity in epilepsy's presentation, any accompanying auras, behavioral components, seizure control, the degree to which the affected individual's quality of life is impacted, and the underlying etiology. Some seizures are not the result of epileptic discharges generated in the brain and are termed NES for non-epileptic seizures (the term "pseudoseizures" is generally avoided because of the pejorative tone and lack of precision). NES can arise from medical disorders such as syncope, migraine, stroke/TIA, movement disorders, sleep disorders, or metabolic disturbances such as diabetes. NES can also have psychological origins, including conversion disorder, somatization disorder, or Munchausen syndrome. Some patients may have episodes of both NES and epileptic seizures, which can greatly complicate accurate diagnosis. Finally, some patients feign seizures for primary gain, usually within the context of personal injury litigation or seeking financial support such as long-term disability.

Categorization

Seizures are classified according to the extent of presumed underlying neural involvement. Broadly speaking, they are characterized as generalized (involving the whole brain) or partial (starting in a specific area, or seizure focus; for more on this see Seizure entry). Electroencephalographic (EEG) monitoring through the use of scalp electrodes and the clinical history are generally employed for diagnosis. However, positive EEG findings do not occur in all patients with epilepsy, and having a negative EEG does not rule out epilepsy. EEG can be helpful although not necessarily definitive as a means of determining whether seizures originate across the whole brain or from a localized seizure focus. EEG localization is especially important in treatment planning for surgical intervention.

Epilepsy can also be classified as one of three etiologies describing the presumptive cause: idiopathic, symptomatic, and cryptogenic. Whereas symptomatic and cryptogenic epilepsies are caused by a lesion (not readily apparent in cryptogenic types), in idiopathic epilepsy, the underlying pathology is unknown. In addition to being characterized by different types or etiologies of seizures, some cases of epilepsy are associated with specific syndromes. These syndromes may become evident in early childhood, such as Lennox–Gastaut, syndrome, West syndrome, benign Rolandic epilepsy, and Landau–Kleffner syndrome. Others include Rasmussen's encephalitis juvenile myoclonic epilepsy, temporal lobe epilepsy (TLE), frontal lobe epilepsy, and neurocutaneous disorders including Type 1 neurofibromitosis, Sturge–Weber syndrome, and

tuberous sclerosis. Chronic TLE arising from mesial temporal sclerosis may also be regarded as a syndrome. Finally, some classification schemes combine various aspects of seizure type, semiology, etiology, and associated syndrome features (Engel, 2006; Loddenkemper et al., 2005).

Epidemiology

Present estimates of the prevalence of epilepsy range from 0.5 to 1% of the population; these estimates are generally consistent across geographic regions and ethnicity. Seizure onset most commonly occurs during childhood with a second smaller peak in senescence. Almost any type of brain pathology can cause seizures, including cerebrovascular disease, prenatal or perinatal insults, head injury, tumor, CNS infections, meningitis, encephalitis, and developmental migrational or dysplastic defects (e.g., heterotopias). Comorbidities include psychiatric disorders, which are present in an estimated 25–50% of individuals with epilepsy, with higher prevalence among patients with poorly controlled seizures. These include depression, anxiety, psychotic disorders, as well as cognitive and personality changes occurring in the interictal or ictal/post-ictal states. Furthermore, research in epilepsy and depression suggests a possible shared pathogenic mechanism between seizures and mood disorders (Jests & Friedman, 2006). In addition, behavioral disorders may arise in response to neurobiological effects, treatment effects, and psychosocial effects; these can include general psychopathology, personality changes, aggression, sexual dysfunction, affective disorders, and psychosis.

Natural History, Prognostic Factors, Outcomes

The natural history of epilepsy varies widely, based on age of onset, seizure type, frequency, etiology, and associated syndrome features. Generalized tonic–clonic seizures (GTCs) are usually well controlled with medication unless they are part of the more complex epilepsy syndromes. Complex partial seizures tend to be more uncontrolled; typically, patients suffering from these disorders undergo multiple trials of AEDs to find a combination that will prevent breakthrough seizures.

Prognostic factors are complicated by the heterogeneity of the syndrome. While approximately 30% of individuals with epilepsy have seizures that are well controlled through antiepileptic medications (AEDs), other individuals have chronic unremitting epilepsy that is treatment refractory, and still others have chronic epilepsy that shows only partial response to AEDs. It is also important to consider the differences in AED responsiveness that are seen in generalized versus partial cases, as well as the epilepsy syndromes, which tend to be very poorly controlled by AEDs. Idiopathic GTCs and absence seizures generally tend to show the best response to medication. If medication trials fail patients may proceed to epilepsy surgery; more than 80% of patients with complex partial seizures of mesial temporal lobe origin achieve postsurgical seizure freedom. Less success is seen when surgery is in neocortical or extra-temporal regions. Presurgical neuropsychological testing combined with demographic variables and other neurodiagnostic findings have been shown to be helpful in determining which patients are at greatest risk for postoperative decline (Bennett, 1992). Across all types of epilepsies and treatments, greater seizure frequency with poorer control is associated with the most deleterious impact on cognitive abilities and quality of life. Multiple episodes of status epilepticus (a flurry of continuous seizures or many seizures with little seizure-free time between episodes) also produce significant declines in cognition, mood, and quality of life.

Sudden unexplained death in epilepsy (SUDEP) occurs in a very small minority of patients (approximately 1 in 1,000 individuals with epilepsy per year). There are no known precursors, and while it is generally associated with GTCs, it is otherwise poorly understood. Although research suggests that epilepsy involves an increased mortality risk, the risk is predominantly present at a younger age and early after diagnosis, and the absolute risk is moderate.

Neuropsychology and Psychology of Epilepsy

Cognitive impairments are sometimes seen in individuals with epilepsy, and are related to seizure type, seizure frequency and severity, age of seizure onset, medication variables, and the underlying pathology. Generalized epilepsies cause more cognitive impairment if they are not well controlled; frequency and other factors contribute to the severity of impairment. Generalized syndromes are associated with the most severe cognitive difficulties. Idiopathic generalized epilepsy is usually well controlled and does not tend to have as many cognitive implications.

In cases of partial epilepsy, if seizures are well controlled minimal impact on cognitive functioning is generally seen. However, when seizures are not well controlled, there are more localization factors; in such cases, a degree of concordance tends to be seen between cognitive deficits and site of seizure generation. However, individuals with severe and

frequent refractory partial epilepsy may fail to show an expected lateralization pattern.

TLE accounts for 70% of chronic symptomatic partial cases; therefore, the cognitive deficits related to TLE are common. In cases involving a left-sided seizure focus, these deficits may include verbal learning and memory deficits including consolidation, retrieval, and delayed recall, as well as naming deficits (i.e., semantic memory). With right TLE, deficits may involve nonverbal memory deficits, although traditional measures for these deficits tend to be relatively insensitive to the effects of right temporal lobe dysfunction (for more on this, see Barr, 1997). Frontal deficits can also be seen in TLE, as involvement of areas distal to the seizure onset zone is not uncommon; these may include deficits of executive functions such as attention, working memory, mental flexibility, response inhibition, and planning.

AEDs also contribute to cognitive deficits in epilepsy (see Meador, 2006). The cognitive deficits most commonly associated with AEDs are attention, concentration, and psychomotor speed. However, the presence and extent of cognitive effects of AEDs on cognition can vary by whether older or newer generation medications are being taken. Generally, the barbiturates have the most deleterious effects on cognitive abilities. Fewer but sometimes still significant cognitive side effects can be seen in other older generation drugs such as phenytoin, carbamazepine, and valproate. Newer generation drugs approved in the 1990s and beyond tend to have a less severe impact on cognition. Polypharmacy is often associated with greater cognitive impairment. However, effective seizure control is usually more contributory to cognitive status than the specific AED(s) being taken.

Evaluation

The neuropsychological evaluation can be important for several reasons. First, it allows for an understanding of the extent of cognitive impairment in individuals with epilepsy as well as aides in characterizing psychosocial and quality of life issues that may be amenable to intervention. Secondly, it can be an important means of providing corroborating evidence for localizing the epileptogenic zone, which is particularly critical for presurgical evaluations. This information can help with presurgical treatment planning and prediction of postsurgical sequelae, including appropriateness of cognitive remediation.

There are specialized procedures in which neuropsychologists participate as part of neurosurgical interventions for epilepsy. Prior to surgery, many centers conduct the Wada test to identify the laterality of language or to identify language dominance and to identify the memory capability of each hemisphere separately. The test, named after Dr. Juhn Wada who developed the procedure at the Montreal Neurological Institute, involves anesthetizing one hemisphere at a time and then conducting brief language and memory testing of the intact hemisphere. This is accomplished by injection of sodium amobarbital into the internal carotid artery as part of an angiogram. The Wada test can also help predict possible postoperative memory declines and assist in corroborating seizure localization by examining the disparity of memory scores between the two hemispheres. Although functional MRI is being used by many centers for language lateralization, it has not yet supplanted the Wada test and is less effective in assessing memory. Neuropsychologists also frequently assist in cortical mapping with electrical stimulation of the brain to more precisely localize language, other cognitive abilities, and sensorimotor areas. This mapping is conducted during stimulation through chronically implanted (days to weeks) subdural grids and strips involving two craniotomies, or during intraoperative stimulation with the patient awake after the craniotomy opening in a one-stage procedure.

Treatment

Antiepileptic drugs (AEDs), also known as anticonvulsants (a misnomer because not all seizures are convulsive), are a common treatment. The primary mechanism of action of these drugs varies, but includes sodium channels, calcium channels, GABA, glutamate, calcium inhibitors, and hormones. The side effects of AEDs vary considerably depending on the specific medication. They can include diplopia, cognitive effects, somnolence, weight gain/loss, gingival hyperplasia, osteoporosis, gastrointestinal complaints, headache, and rash. In addition to AEDs, other interventions include hormonal therapy and a ketogenic diet. Surgical interventions include resective surgery, corpus collosotomy, hemispherectomy, and implantation of stimulating devices, e.g., vagus nerve stimulator implantation. Alternative techniques such as yoga and neurofeedback are controversial and have not been subject to controlled clinical trials.

Cross References

- ▶ Absence Seizure
- ▶ Anticonvulsants
- ▶ Aura

- ▶ Clonazepam
- ▶ Generalized Seizure
- ▶ Grand Mal Seizure
- ▶ Juvenile Myoclonic Epilepsy
- ▶ Myoclonic Epilepsy of Infancy
- ▶ Nonepileptic Seizures
- ▶ Partial Seizure
- ▶ Phenobarbital
- ▶ Seizure
- ▶ Wada Test

References and Readings

Allen Hauser, W. (1990). *Epilepsy: Frequency, causes, and consequences.* New York: Demos Publications.

Barr, W. B. (1997). Examining the right temporal lobe's role in nonverbal memory. *Brain and Cognition, 35*(1), 26–41.

Bennett, T. L. (Ed.). (1992). *The neuropsychology of epilepsy.* New York: Plenum Press.

Engle, J., Jr. (2006). Report of the ILAE classification core group. *Epilepsia, 47*(9), 1558–1568.

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

Jests, D. V., & Friedman, J. H. (2006). *Psychiatry for neurologists.* Totowa, NJ: Humana Press.

Loddenkemper, T., Kellinghaus, C., Wyllie, E., Najm, I. M., Gupta, A., Rosenow, F., et al. (2005). A proposal for a five-dimensional patient-oriented epilepsy classification. *Epileptic Disorders, 7*(4), 308–316.

Meador, K. J. (2006). Cognitive and memory effects of the new antiepileptic drugs. *Epilepsy Research, 68*(1), 63–67.

Epinephrine

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Synonyms

Adrenalin; Adrenaline

Indications

Epinephrine (E), also called adrenalin, is a sympathomimetic monoamine neurotransmitter that acts as a hormone. It is a catecholamine, derived from the amino acids phenylalanine and tyrosine, and released from the adrenal medulla. In 1895, the Polish physiologist, Napoleon Cybulski discovered E in the adrenal gland. In 1901, a Japanese chemist, Jokichi Takamine isolated the same hormone from cow glands. And in 1904, Friedrich Stolz first artificially synthesized E.

E shares common pathways with catecholamines, dopamine, and norepinephrine. During times of stress, the splanchnic nerves in the adrenal medulla stimulate the sympathetic nervous system (SNS) to release E and adrenocorticotropic hormone (ACTH). ACTH activates the adrenal cortex to produce the stress-reactive hormone, cortisol. This process interacts with the synthesis of E. Tyrosine hydroxylase converts tyrosine to L-dopa. L-dopa synthesizes dopamine via dopa decarboxylase, and dopamine β-hydroxylase converts dopamine into norepinephrine. Norepinephrine then synthesizes E. Specialized neurons secrete E and norepinephrine when modified preganglionic sympathetic fibers synapse onto neuroendocrine cells. Sympathomimetic or adrenergic drugs bind to E receptors, also called adrenoceptors.

Mechanisms of Action

When E is secreted into the bloodstream, it rapidly activates the sympathetic nervous system to prepare the body for emergencies. Although E is not itself considered psychoactive, SNS arousal also releases norepinephrine which is the precursor to E, is psychoactive, and has similar actions in the body. In the liver, E binds to both α and β receptors. The effects of E are mediated as a nonselective agonist through sympathetic adrenoceptors, α_1, α_2, β_1, and β_2. When bound to α_1 receptors, E activates the inositol-phospholipid signaling pathway. Glycogen synthase is then inactivated and phosphorylation activates phosphorylase kinase. This triggers the enzyme, glycogen phosphorylase to stimulate glycogenolysis. In the liver and muscle cells, E catalyzes the adenylate cyclase signaling pathway to activate glycogenolysis via β-adrenergic receptors. Glycogenolysis results in the release of glucose into the bloodstream via the breakdown of glycogen.

Blood glucose levels increase as a function of glycogen catabolism in the liver. Lipolysis in fat cells is activated and glucose supplies to the brain increase. This contributes to heightened mental alertness. When E is released, it acts as a hormone, increasing SNS activity. SNS activation leads to an elevated metabolic rate, respiration, and heart rate, increased stroke volume and blood pressure, dilation of respiratory passageways and pupilary muscles, and activation of sweat glands. E also causes arterioles in skeletal muscle to dilate which increases energy, but constricts arterioles in the gastrointestinal tract and the skin

which decreases digestion and elimination. Prolonged sympathetic activation and the associated release of stress hormones E and cortisol can lead to immune suppression (Epinephrine, 2009b).

Specific Compounds and Properties

E is an organic, endogenous central nervous system sympathomimetic amine and derivative of the biologically active group, the catecholamines. It has an average molecular weight of 182.2044 and its chemical formula is $C_9H_{13}NO_3$ (Metabolomics Toolbox, 2009).

Clinical Use (Including Side Effects)

Therapeutically, E and norepinephrine are central to many actions of sympathomimetic agents such as alpha (α)- and beta (β)-adrenergic agonists, MAOI, and COMT Inhibitors. E activation in various preparations is used as a cardiac stimulant to treat cardiac arrest, to relax bronchial tissue in asthmatics and anaphylaxis, as a vasoconstrictor in cases of hemorrhage, or to reduce the rate of absorption of anaesthetic agents. It is also used to treat sepsis and prepared as a borate salt for ophthalmological use.

Monoamine oxidase inhibitors (MAOIs) and catechol-*O*-methyl transferase (COMT) inhibitors act as indirect-acting adrenergic sympathomimetics. Monoamine oxidase (MAO) is the enzyme primarily responsible for the metabolism of E and norepinephrine. MAOIs stimulate the production and release of catecholamines in the CNS, and inhibit the normal process of monoamine oxidase degrading and removing E, norepinephrine, and dopamine from the presynaptic terminal. Preventing the metabolism of these catecholamines increases their concentration in the CNS (Hoffman, 2004; Katzung, 2004).

MAOIs such as tranylcypromine and phenelzine are used to treat resistant depressions and panic disorder. By negative feedback at presynaptic α_2- or β-adrenergic receptors, chronic use of MAOIs has been shown to lead to down-regulation of the synthesis of E and NE. Other side effects include sedation, insomnia, anxiety, agitation, dizziness, constipation, diarrhea, nausea, orthostatic hypotension, syncope, sexual dysfunction, appetite changes, and in rare instances hypertensive crisis, mania, seizures, and hepatotoxicity. The severe symptoms are of concern if tyramine-containing foods or drugs are ingested.

COMT Inhibitors inhibit the actions of the enzyme, catechol-*O*-methyl transferase (COMT). As is the function of normal MAO in the presynatic terminal, COMT degrades E and NE postsynaptically. COMT inhibitors interfere with this degradation and transformation process which enables these catecholamines to remain active. Examples of COMT inhibitors include entacapone and tolcapone used adjunctively with levodopa to treat Parkinson's disease. Side effects include dyskinesias, diarrhea, nausea, abdominal pain, red-brown urine, and hepatotoxicity.

Mixed α/β-agonists relax smooth muscle in the lungs and uterus, and in skeletal muscle vasculature. Their actions increase cardiac output and dilate bronchials and pupils. The normal action of β receptors involves E-induced stimulation. β_1-adrenergic receptors are located mainly in the heart and kidneys. Stimulation increases cardiac conduction automaticity and velocity, and causes the kidneys to release renin. β_2-adrenergic receptors are located primarily in the lungs, gastrointestinal tract, liver, uterus, vascular smooth, and skeletal muscle. Stimulation of β_2 receptors induces glycogenolysis in the liver, and relaxes skeletal and smooth muscle in the lungs. β_3 receptors are primarily in fat cells and when stimulated activate lipolysis.

E, arbutamine, mabuterol, mephentermine, phenylpropanolamine, and synephrine are examples of mixed α/β-agonists prescribed for anaphylaxis, acute asthmatic reactions, cardiac arrest, and glaucoma. Side effects include dry mouth, anxiety, nervousness, insomnia, nausea, vomiting, elevated heart rate, hypertension, and hives.

Nonselective and β_1-selective adrenergic antagonists or blockers are used therapeutically to inhibit symptoms of physiologic hyperarousal known as "the fight/flight response." β-antagonists have intrinsic sympathomimetic actions. As anxiolytics, their effects are a function of blocking actions of E and norepinephrine, decreasing sympathetic over-activation. The sympathetic antagonism of β-blockers, such as atenolol, metoprolol, and nebivolol, appears to be the primary therapeutic mechanism enabling diminished performance and social anxiety. They are also used to treat angina, aortic aneurysm, arrhythmias, essential tremor, hyperhidrosis, hypertension, hyperthyroidism, mitral valve prolapse, and ventricular tachycardia. Nonselective or mixed α/β-antagonist such as celiprolol, inderolol, mepindolol, nadolol, sotalol, and timolol can also be used to reduce intraocular pressure in glaucoma treatment. Side effects can include abnormal vision, bradycardia, bronchospasm, cold extremities, depression, diarrhea, dizziness, edema, fatigue, hallucinations, headaches, heart block, heart failure, hypotension, insomnia, muscle cramps,

nausea, sexual dysfunction, sleep disturbances, and problematic glucose/lipid metabolism.

Cross References

- ▶ Autonomic Nervous System
- ▶ Catecholamine
- ▶ Dopamine
- ▶ Norepinephrine

References and Readings

Epinephrine. (2008). In *Merriam-Webster Online Dictionary*. Retrieved January 13, 2009, from http://www.merriam-webster.com/dictionary/Epinephrine.

Epinephrine. (2009a). In *MedicineNet*. Retrieved January 13, 2009, from http://www.medterms.com.

Epinephrine. (2009b). In *Wikipedia*. Retrieved January 13, 2009, from http://en.wikipedia.org/wiki/epinephrine.

Hoffman, B. B. (2004). Adrenoceptor-activating & other sympathomimetic drugs. In B. Katzung (Ed.), *Basic and clinical pharmacology* (9th ed., pp. 122–141). New York: Lange Medical Books/McGraw-Hill.

Katzung, B. G. (2004). Introduction to autonomic pharmacology. In B. Katzung (Ed.), *Basic and clinical pharmacology* (9th ed., pp. 75–93). New York: Lange Medical Books/McGraw-Hill.

Lu, D. (2009). Sites of drugs acting at the adrenergic synapses. In *PHAR 402*. Retrieved January 21, 2009, from http://www.uic.edu/classes/phar/phar402.

Metabolomics Toolbox. (2009). In *Human Metabolome Database*. Retrieved January 19, 2009, from http://hmdb.ca/scripts/show_card.cgi?METABOCARD = HMDB00216.txt.

Nicol, R. A. (2004). Introduction to the pharmacology of CNS drugs. In B. Katzung (Ed.), *Basic and clinical pharmacology* (9th ed., pp. 336–350). New York: Lange Medical Books/McGraw-Hill.

Episodic Memory

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Synonyms

Autobiographical memory; Declarative memory; Explicit memory; Relational memory

Definition

Episodic memory is a type of associative learning where relationships between stimuli (e.g., people, places and things) form an autobiographical register of events as they occur in time. Episodic memory is the kind of memory that allows us to recollect our own past and the experiences we have lived through. Episodic memory formation is one of the primary functions of the medial temporal lobe, which is why episodic memory is often referred to as a hippocampal-dependent memory function. Damage to the hippocampus, or the cerebral cortex surrounding it, results in a loss of episodic memory known as amnesia. Episodic memory should not be confused with semantic memory, which refers to the understanding of meanings and the recollection of factual information about the world. Semantic memory is context-independent knowledge, and together with episodic memory make up the two types of declarative or explicit memory, so named because memories of this type can be brought to the forefront of consciousness and verbalized.

Current Knowledge

In 1972, a Canadian psychologist Endel Tulving proposed that episodic memory is different from other kinds of memory (Tulving, 1983; 2002). According to Tulving, two closely related features that distinguish episodic from other forms of memory are autonoetic consciousness and chronesthesia. Autonoetic consciousness refers to mental "time-travel", or the cognitive reenactment of previous events, hypothesized to give rise to the subjective experience of remembering. Chronesthesia refers to the subjective experience of time, as in the perception of past, present, and future.

Episodic memory encoding is an automatic process and occurs without effort. It is single-trial learning. A brief experience that occurred decades earlier can be involuntarily recalled if the appropriate eliciting stimuli are encountered. During memory encoding, attention plays an important role in the organization of the information being encoded by the hippocampal system, and emotion can have a profound effect on the memory encoding. The emotional modulation of memory is thought to involve interactions between the hippocampal memory system and inputs from the amygdala. For example, post-traumatic stress disorder (PTSD) is a condition of uncontrollable automatic memory recall, usually of a

very frightening or threatening event. Although other memory mechanisms (e.g., Pavlovian conditioning) are likely to also be involved, PTSD is an example of how emotional arousal and attention can influence episodic memory function.

Sensory stimuli are processed in multimodal association regions of the parietal, temporal, and occipital cortices, brain areas thought to give rise to perceptual experience. These multisensory channels are "funneled" into the medial temporal lobe along separate multi-synaptic pathways, and are passed into the associative networks of the hippocampal system. The hippopampal system gives rise to projections that return to the same multimodal association regions outside the medial temporal lobe. The organization of this circuitry – funneling sensory information into the hippocampus, and hippocampal projections back to the sensory regions giving rise to the initial input – is thought to allow for the consolidation of episodic memory. The term "consolidation" refers to the time-dependent process of long-term memory storage.

Our understanding of memory consolidation comes from observation of patients with retrograde amnesia (the inability to recall things that happened in the time prior to the medial temporal lobe damage). Patients with amnesia resulting from medial temporal lobe damage are typically still able to recall facts and remote memories from the years before the injury. The "temporally graded" nature of retrograde amnesia suggests that memory consolidation is a protracted process that can take years, and that eventually the retrieval of that memory is no longer dependent on the medial temporal lobe. However, the long-standing hypothesis that long-term memory eventually becomes entirely independent of the medial temporal lobe has recently been challenged (Nadel & Moscovitch, 2001).

Brain imaging studies of normal subjects provides evidence that the right prefrontal cortex is activated during the retrieval of episodic memories (Buckner, Raichle, Miezin, & Petersen, 1996; Nyberg, 1998). The frontal lobes are thought to be critical for associating the content of an event (semantic knowledge) with its source episodic memory (i.e., when and where the event occurred). Although episodic memory and semantic memory are both affected by medial temporal lobe damage, episodic memory and semantic memory may be dissociable in amnesic patients with severe frontal lobe damage (Squire & Zola, 1998). For example, patient K.C., who has been studied for many years by Tulving and his colleagues, has no episodic memory but does show spared semantic memory (Rosenbaum et al., 2005).

Future Directions

In 1998, Clayton and Dickinson demonstrated that animals might possess episodic memory. They showed that Western Scrub-Jays (Aphelocoma californica) can remember where they cache different types of food, and depending on the perishability of the item and the amount of time elapsed since caching, could discriminate among them. Clayton and Dickinson argued that while they could not demonstrate autonoetic consciousness, the "what-where-and-when" components of the Scrub-Jays' cashing behavior was evidence that the birds possessed episodic-like memory. Whether episodic memory is a uniquely human capacity remains a question of scientific debate.

Hyperthymestic syndrome is a term proposed by Parker and her colleagues for a clinical case of superior autobiographical memory (Parker, Cahill, & McGaugh, 2006). The woman, known as "A.J.," spends an abnormally large amount of time thinking about her personal past, and has the extraordinary capacity to recall specific events from it. Since the first report of hyperthymesia was published, several other cases have been reported, and as more of these cases of extraordinary autobiographical memory surface over time, researches may be able to utilize brain imaging techniques to reveal the mechanisms underlying both superior, as well as normal, episodic memory.

Cross References

- ▶ Hippocampus
- ▶ Medial Temporal Lobe
- ▶ Paired-Associate Learning

References and Readings

Buckner, R. L., Raichle, M. C., Miezin, F. M., & Petersen S. E. (1996). Functional anatomic studies of memory retrieval for auditory words and visual pictures. *The Journal of Neuroscience, 16*(19), 6219–6235.

Clayton, N. S., Griffiths, D. P., Emery, N. J., & Dickinson, A. (2001). Elements of episodic-like memory in animals. *Philosophical Transactions of the Royal Society of London – Series B: Biological Sciences, 356* (1413), 1483–1491.

Nadel, L., & Moscovitch, M. (2001). The hippocampal complex and long-term memory revisited. *Trends in Cognitive Sciences, 5*(6), 228–230.

Nyberg, L. (1998). Mapping episodic memory. *Behavioural Brain Research, 90*(2), 107–114.

Parker, E. S., Cahill, L., & McGaugh, J. L. (2006). A case of unusual autobiographical remembering. *Neurocase, 12*(1), 35–49.

Rosenbaum, R. S., Kohler, S., Schacter, D. L., Moscovitch, M., Westmacott, R., Black, E. S., et al. (2005). The case of K.C.: Contributions

of a memory-impaired person to memory theory. *Neuropsychologia, 43*(7), 989–1021.
Squire, L. R., & Zola, S. M. (1998). Episodic memory, semantic memory, and amnesia. *Hippocampus, 8*(3), 205–211.
Tulving, E. (1983). *Elements of episodic memory.* London: Oxford University Press.
Tulving, E. (2002). Episodic memory: From mind to brain. *Annual Review of Psychology, 53*, 1–25.

Epithalamus-Pineal Gland, Habenular Nuclei

► Diencephalon

EPS

► Extrapyramidal Symptoms

Epstein–Barr Virus

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Definition

Epstein–Barr virus, frequently referred to as EBV, is a member of the herpesvirus family and one of the most common human viruses. The virus occurs worldwide, and most people become infected with EBV sometime during their lives. In the United States, as many as 95% of adults have been infected.

Infection with EBV often results in mononucleosis. Symptoms of infectious mononucleosis are fever, sore throat, and swollen lymph glands. Although the symptoms of infectious mononucleosis usually resolve quickly, EBV remains dormant or latent in blood cells for lifetime.

Evaluation and Treatment

The clinical diagnosis of infectious mononucleosis is based on symptoms of fever, sore throat, swollen lymph glands, and the age of the patient. Usually, laboratory tests are needed for confirmation. Serologic results for persons with infectious mononucleosis include an elevated white blood cell count, an increased percentage of certain atypical white blood cells, and a positive reaction to a "mono spot" test.

There is no specific treatment for infectious mononucleosis other than treating the symptoms. Symptoms related to infectious mononucleosis caused by EBV infection seldom last for more than 4 months. When the illness lasts for more than 6 months, it is called chronic EBV infection. However, valid laboratory evidence for continued active EBV infection is seldom found in these patients. Chronic symptoms should be investigated further to determine if they meet the criteria for chronic fatigue syndrome (CFS). This process includes ruling out other causes of chronic illness or fatigue.

Cross References

► Chronic Fatigue Syndrome

References and Readings

National Center for Infectious Diseases Center for Disease Control and Prevention webpage at www.cdc.gov/ncidod/diseases/ebv.htm, accessed July 17, 2008.

EQ-5D

► EuroQol/EQ-5D

Equipotentiality

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Definition

Equipotentiality – a notion developed by Karl Spencer Lashley (1890–1958) positing that all areas of the brain are equally able to perform a task. This contrasts with the theory of localization, according to which neurocognitive

functions are specifically referable to discrete areas of the brain; hence, damage to restricted regions would be expected to produce selective cognitive deficits. Equipotentiality theory, however, hypothesized that the severity of cognitive dysfunction was directly related to the total amount of tissue damage. For example, memory functioning was thought to be diffusely distributed throughout the cortex rather than related to defined circuits or pathways. Under this theory, intact areas of the cortex could assume responsibility for discrete cognitive functions following injury. The theory did allow, however, for localization related to sensory and motor processes. The related concept of "mass action" posited that cognitive functions are equally and widely distributed across brain areas and that the entire cortex participates in cognitive functioning.

Cross References

► Karl Lashley

References and Readings

Lashley, K. S. (1929). *Brain mechanisms and intelligence.* Chicago: University of Chicago Press.

Lashley, K. S. (1930). Basic neural mechanisms in behavior. *Psychological Review, 37*, 1–24.

Equivalent Forms

► Alternate Test Forms

Erethism

► Mercury Exposure

ERP's

► Event-Related Paradigms

Error Evaluation and Error Utilization

► Error Recognition and Correction

Error Handling

► Error Recognition and Correction

Error Recognition and Correction

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Synonyms

Error evaluation and error utilization; Error handling

Definition

Error recognition refers to the ability to recognize or detect the presence of an error; recognition may happen as the error is being made or after it has occurred. Error correction is the ability to use knowledge about the presence of an error to remedy or correct it, allowing for an error-free outcome.

Error recognition and correction are distinct cognitive processes that facilitate adaptive functional living.

Current Knowledge

Human error has been a topic of interest in both clinical and applied psychology for decades, appearing in research on industrial safety, driving accidents, and computer science. More recently, investigation into the process of error recognition and correction among individuals with impaired cognitive functioning has expanded our understanding of the cognitive skills and brain regions involved. Early case studies described patients with frontal lobe damage the ability of which to identify and remedy their own errors was disturbed. Although the terms "error evaluation" and "error utilization" were used synonymously in some cases (e.g., Luria, Pribram, & Homskaya, 1964), subsequent reports of patients who were unable to correct their errors despite having intact error recognition have indicated that these abilities are distinct cognitive processes. Moreover, error recognition is considered a necessary prerequisite for error correction, which suggests that increasing error recognition may be a useful focus for intervention among individuals with cognitive impairment.

Previous literature demonstrates that individuals with cognitive impairments secondary to traumatic brain injury recognize and correct significantly fewer errors during everyday tasks as compared to healthy controls (e.g., Hart, Giovannetti, Montgomery, & Schwartz, 1998). Similar findings have been documented among individuals with dementia (Bettcher, Giovannetti, Macmullen, & Libon, 2008; Giovannetti, Libon, & Hart, 2002). Errors occur in both the error recognition and correction stages of the process, depending on the cognitive functions and/or brain regions that have been impacted.

Several domains of cognitive functioning are recruited during the process of recognizing and correcting errors, from simple attention, visual perception, and processing speed to higher-order executive functions including mental flexibility, problem-solving, and self-monitoring. Awareness, or the ability to be perceptive and insightful of one's own condition, actions, or feelings, is particularly important for successful error handling (Sohlberg & Mateer, 2001). Multiple models of awareness of cognitive deficits exist, and such models provide a useful framework for conceptualizing the process of error recognition and correction. Crosson et al. (1989) suggest a pyramid model of awareness of which intellectual awareness forms the foundation for emergent awareness, each of which is necessary for anticipatory awareness. Intellectual awareness, or the ability to understand that a function is impaired, and emergent awareness, which refers to the ability to recognize a problem while it is happening, each play an important role in error recognition, and consequently error correction. Toglia and Kirk (2000) suggest a more dynamic model of awareness that distinguishes between stored metacognitive knowledge and task-specific awareness. On-line awareness, or the ability to monitor and regulate performance during a given task or situation (Toglia & Kirk), provides a more comprehensive framework for understanding the cognitive processes involved in the processes of error recognition and correction. These authors further argue that the ability to gauge the difficulty level of a task and anticipate pitfalls (emergent awareness) is related to the initiation of self-monitoring and strategy use, which allows for both error recognition and correction.

Multiple brain regions are involved in the process of error recognition and correction. The frontal and prefrontal regions of the brain are strongly implicated in executive functioning, both independently and through interconnections with other brain regions. Damage to the frontal lobes can result in impairments in self-monitoring, set-shifting, and problem-solving, and has long been associated with disruption of the error handling process (e.g., Luria et al., 1964). Additionally, damage to the medial prefrontal cortex and anterior cingulate cortex (ACC) is associated with disturbed ability to monitor performance and recognize errors (Gehring & Fencsik, 2001; Stemmer et al., 2004), and fMRI studies report activation of the ACC when cognitive errors are made (Carter et al., 1998). Event-related potentials (ERPs) document the brain's unique response to errors: a negative component of an event-related brain potential called an error-related negativity (ERN) is detected at the onset of an error, and peaks soon thereafter (Mathalon, Whitfield, & Ford, 2003). Finally, correlational analyses of ERP and fMRI data indicate that the rostral ACC is selectively activated during error processing (Mathalon et al.).

Cross References

- ▶ Anosognosia
- ▶ Awareness
- ▶ Cognitive Rehabilitation
- ▶ Error, Sources of

References and Readings

Bettcher, B. M., Giovannetti, T., Macmullen, L., & Libon, D. J. (2008). Error detection and correction patterns in dementia: A breakdown of error monitoring processes and their neuropsychological correlates. *Journal of the International Neuropsychological Society, 14*, 199–208.

Carter, C., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulated cortex, error detection, and the online monitoring of performance. *Science, 280*, 747–749.

Crosson, B., Barco, P. P., Velozo, C. A., Bolesta, M. M., Cooper, P. V., Werts, D., et al. (1989). Awareness and compensation in postacute head injury rehabilitation. *Journal of Head Trauma Rehabilitation, 4*, 46–54.

Giovannetti, T., Libon, D. J., & Hart, T. (2002). Awareness of naturalistic action errors in dementia. *Journal of the International Neuropsychological Society, 8*, 633–644.

Hart, T., Giovannetti, T., Montgomery, M. W., & Schwartz, M. F. (1998). Awareness of errors in naturalistic action after traumatic brain injury. *Journal of Head Trauma Rehabilitation, 13*, 16–28.

Luria, A. R., Pribram, K. M., & Homskaya, E. D. (1964). An experimental analysis of the behavioral disturbance produced by a left frontal arachnoidal endothelioma (meningioma). *Neuropsychologia, 2*, 257–280.

Mathalon, D. H., Whitfield, S. L., & Ford, J. M. (2003). Anatomy of an error: ERP and fMRI. *Biological Psychology, 64*, 119–141.

Sohlberg, M. M., & Mateer, C. A. (2001). Management of dysexecutive symptoms. In M. M. Sohlberg & C. A. Mateer (Eds.), *Cognitive rehabilitation: An integrative neuropsychological approach* (pp. 230–268). New York: The Guilford Press.

Toglia, J., & Kirk, U. (2000). Understanding awareness deficits following brain injury. *NeuroRehabilitation, 15*, 57–70.

Error, Sources of

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Definition

Sources of error are the factors that influence a test score or item performance that are extraneous to the construct of interest. In a memory test, sources of error might include examiner imprecision or subjectivity, fatigue, time of day, or level of hunger. The effects of these sources comprise the error term in classical test theory. Test construction is conducted so as to minimize or otherwise allow an estimate and correction of the error term so as to maximize the extent to which an observed score reflects the true value of the construct of interest.

Cross References

- ▶ Classical Test Theory
- ▶ Test Reliability
- ▶ Test Validity

References and Readings

Kline, P. (1986). *A handbook of test construction: Introduction to psychometric design.* New York: Methuen.

Errorless Learning

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Synonyms

Delayed prompting; Response prevention; Stimulus fading

Definition

Errorless learning refers to a type of training that reduces the learner's opportunity to make errors during the learning process. The aim of this approach is to prevent the learner from reinforcing errant behavior, which may occur with repeated mistakes. One of the earliest researched errorless learning techniques, *stimulus fading*, is best highlighted in animal research by Terrace (1963), the first research that demonstrated the benefits of errorless learning. Terrace demonstrated that pigeons were better able to discriminate between green and red lights using stimulus fading. First, Terrace introduced a red light, the correct response. Once the pigeons responded consistently to the red light, a green light (the incorrect response) was introduced gradually to the experiment. The green light was at first briefly presented at a dim intensity, but eventually reached the same intensity and duration as the red light exposure. Eventually, when both red and green lights were being presented simultaneously at the same intensity and for the same duration, the pigeons consistently pecked the red light, and not the green, thus demonstrating the benefit of an errorless learning technique. Other errorless learning techniques include stimulus shaping, response prevention, delayed prompting, superimposition with stimulus fading, and superimposition with stimulus shaping (*see* Mueller, Palkovic, & Maynard, 2007).

Current Knowledge

Errorless Learning: A Popular Theory

The process of errorless learning has been theorized to be based on Hebbian plasticity. According to Hebb (1961), the synaptic connection between two neurons will be strengthened if they fire together. Learning occurs if this particular pattern of neural activity is activated on subsequent occasions. Thus, the patterned response will occur with greater frequency, whether the response is correct or incorrect. The goal of errorless learning is to increase synaptic firing between neurons while making mostly correct responses.

Applications in Current Research

Research has shown that errorless learning can be useful for a number of different populations and has been demonstrated to be beneficial in the treatment of a variety of conditions. Errorless learning has been shown to be beneficial in the treatment of memory impairments due to dementia, Alzheimer's type (Clare, Wilson, Carter, Breen, Gosses, & Hodges, 2000) and Korsakoff's Syndrome

(Komatsu, Mimura, Kato, Wakamatsu, & Kashima, 2000), anomia (Fillingham, Sage, & Ralph, 2006) and aphasia (Fillingham, Hodgson, Sage, & Ralph, 2003) following stroke or traumatic brain injury, remediation of cognitive impairments for patients with schizophrenia (Mulholland, O'Donoghue, Meenagh, & Rushe, 2008), and has been used to enhance skill acquisition among typical school-age children and children with pervasive personality disorders. Current research continues to explore additional applications for errorless learning, such as treatment for brain injured patients with memory impairment and poor executive functioning (Pitel, Beaunieux, Lebaron, Joyeux, Desgranges, & Eustache, 2006), and the relearning of functional skills in patients after acquired brain injury (Martelli, Nicholson, & Zasler, 2008) and following acute stroke events (Mount, Pierce, Parker, DiEgidio, Woessner, & Spiegel, 2007).

Cross References

- ▶ Hebb, Donald (1904–1985)
- ▶ Learning
- ▶ Stimulus Control
- ▶ Stimulus Generalization

References and Readings

Clare, L., Wilson, B. A., Carter, G., Breen, K., Gosses, A., & Hodges, J. R. (2000). Intervening with everyday memory problems in Dementia of Alzheimer Type: An errorless learning approach. *Journal of Clinical and Experimental Neuropsychology, 22*(1), 132–146.

Fillingham, J. K., Hodgson, C., Sage, K., & Lambon Ralph, M. A. (2003). The application of errorless learning to aphasic disorders: A review of theory and practice. *Neuropsychological Rehabilitation, 13*(3), 337–363.

Fillingham, J. K., Sage, K., & Lambon Ralph, M. A. (2006). The treatment of anomia using errorless learning. *Neuropsychological Rehabilitation, 16*(2), 129–154.

Hebb, D. O. (1961). *The organization of behavior: A neuropsychological theory. Stimulus and response – and what occurs in the brain in the interval between them.* New York: Science Editions, Inc.

Komatsu, S., Mimura, M., Kato, M., Wakamatsu, N., & Kashima, H. (2000). Errorless and effortful processes involved in the learning of face-name associations by patients with alcoholic Korsakoff's syndrome. *Neuropsychological Rehabilitation, 10*(2), 113–132.

Maritelli, M. F., Nicholson, K., & Zasler, N. D. (2008). Skill reacquisition after acquired brain injury: A holistic habit retraining model of neurorehabilitation. *NeuroRehabilitation, 23*, 115–126.

Mount, J., Pierce, S. R., Parker, J., DiEgidio, R., Woessner, R., & Spiegel, L. (2007). Trial and error versus errorless learning of functional skills in patients with acute stroke. *NeuroRehabilitation, 22*, 123–132.

Mueller, M. M., Palkovic, C. M., & Maynard, C. S. (2007). Errorless learning: Review and practical application for teaching children with Pervasive Developmental Disorders. *Psychology in the Schools, 44*(7), 691–700.

Mulholland, C. C., O'Donoghue, D., Meenagh, C., & Rushe, T. M. (2008). Errorless learning and memory performance in schizophrenia. *Psychiatry Research, 159*(1–2), 180–188.

Pitel, A. L., Beaunieux, H., Lebaron, N., Joyeux, F., Desgranges, B., & Eutasche, F. (2006). Two case studies in the application of errorless learning techniques in memory impaired patients with additional executive deficits. *Brain Injury, 20*(10), 1099–1110.

Terrace, H. S. (1963). Discrimination learning with and without "errors." *Journal of the Experimental Analysis of Behavior, 6*(1), 1–27.

Erudition

▶ Learning

Escitalopram

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Generic Name

Escitalopram

Brand Name

Lexapro

Class

Selective Serotonin Reuptake Inhibitor. Escitalopram is the s-isomer of citalopram.

Proposed Mechanism(s) of Action

Escitalopram blocks the presynaptic serotonin reuptake and desensitizes serotonin receptors (especially 5HT1A).

Indication

Major Depressive Disorder and Generalized Anxiety Disorder.

Off Label Use

Panic disorder, obsessive compulsive disorder, social anxiety disorder, and premenstrual dysphoric disorder.

Side Effects

Serious

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

Common

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

References and Readings

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

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

Additional Information

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

Essential Tremor

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Synonyms

Shaking

Definition

Essential tremor (ET) is the most common movement disorder. It typically presents with bilaterally symmetric hand tremor of 4–8 Hz which is present during postural changes. The tremor is also present during action and absent at rest. A family history is positive in one-half of patients. It is felt to have an autosomal dominant inheritance. The tremor of ET is more symmetric than the tremor of Parkinson's disease (PD). Rigidity and bradykinesia are not seen. ET often involves the upper extremities, head, and voice. It can interfere with fine motor movements such as the use of eating utensils and handwriting. There is no significant response to dopaminergic medications. The tremor tends to worsen with time. It is worsened by emotional factors and, in some patients, temporarily improved by consumption of alcohol.

Cross References

► Action Tremor
► Postural Tremor

References and Readings

Cersosimo, M. C., & Koller, W. C. (2004). Essential tremor. In R. L. Watts, & W. C. Koller (Eds.), *Movement disorders* (2nd ed., pp. 431–458). New York: McGraw-Hill.

Estelle v. Smith (1981)

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Definition

On December 28, 1973, Ernest Benjamin Smith was arrested for murder as a result of an armed robbery in a grocery store in which a clerk was fatally shot by Smith's accomplice. The state of Texas decided to seek the death penalty. Smith was interviewed by Dr. Grigson, a psychiatrist, for approximately 90 min, to determine whether or not he was competent to stand trial. Dr. Grigson found that Smith was competent to stand trial. Smith was charged and convicted of capital murder. During the

sentencing phase, Dr. Grigson underwent direct examination before the jury and testified that Smith was a "very severe sociopath" and that "he will continue his previous behavior and that his sociopathic behavior will 'only get worse'." Dr. Grigson also testified that Smith had no "regard for another human being's property or for their life, regardless of who it may be," that there is "no treatment. . .that in any way at all modifies or changes this behavior," that he "is going to go ahead and commit other similar or same criminal acts if given the opportunity to do so," and that he "has no remorse or sorrow for what he has done." Smith was sentenced to death.

The entirety of Dr. Grigson's opinion and testimony was based solely on his 90-min mental status examination of Mr. Smith. However, Dr. Grigson never provided Smith with a full disclosure of the potential uses for the information gained during the competency evaluation; most notably, he did not indicate that such information could be used to generate predictions about his future behavior. Regardless, the Texas Court of Appeals upheld the conviction and death sentence. Despite this ruling, the Federal District Court removed the death penalty because it found a constitutional error with admitting Dr. Grigson's testimony during the sentencing phase. Specifically, the court ruled that admission of Dr. Grigson's testimony about Mr. Smith's mental status during the penalty phase of the trial violated his 5th Amendment right against self-incrimination because he was not informed during the pretrial mental status examination that he had a right to remain silent and that any statement he made could be used against him during capital sentencing proceedings. Also, to perform a mental health evaluation without defense counsel's knowledge is also a violation of the defendant's 6th Amendment right to effective counsel.

Cross References

► *Dusky v. United States (1960)*

References and Readings

Denney, R. L., & Sullivan, J. P. (2008). Constitutional, judicial, and practice foundations of criminal, forensic neuropsychology. In R. Denney, & J. Sullivan (Eds.), *Clinical neuropsychology in the criminal forensic setting*. New York: Guilford.

Dusky v. U.S. 262 U.S. 402 (1960).

Estelle v. Smith, 451 U.S. 454 (1981).

Weissman, H. N., & DeBow, D. M. (2003). Ethical principles and professional competencies. In A. Goldstein (Ed.), *Handbook of Psychology (Vol. 11). Forensic Psychology*, New Jersey: Wiley.

Estimated Premorbid Intelligence

► Premorbid Intelligence

Estimation Methods

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Synonyms

Premorbid abilities

Definition

An important role of neuropsychological assessment is to identify changes or impairments in cognitive functions from previous performance following an illness or injury. Since premorbid neuropsychological data are rarely available, methods of estimating premorbid abilities are utilized.

Current Knowledge

Some neuropsychologists utilize a clinical estimation of premorbid abilities drawing on information from school, military, and work records as well as family or even self-report to provide a global impression of the individuals' premorbid functioning. While this approach allows the neuropsychologist to incorporate much information from the individual's background and history, there is a lack of standardization to incorporate the information as well as limitations on specificity of premorbid abilities. Therefore, at best one could only conclude very general estimations of function such as above average, average, and below average.

Other clinicians utilize a more formal estimation of premorbid abilities based on either (1) demographic variables, (2) current performance on standardized measures, or (3) a combination of current performance and demographics.

Demographic variables commonly used to estimate premorbid ability include education, occupation, age,

gender, and race. In particular, education and then occupation have demonstrated the highest correlations with intelligence measures. Methods for predicting premorbid intelligence quotient (IQ) scores from demographic variables have been developed for the various Wechsler measures (see Strauss, Sherman, & Spreen, 2006 for a review).

A second method of estimating premorbid abilities involves using current test scores. This approach is based on the concept that an injury or illness will not impact all aspects of intelligence equally and there are certain abilities thought to be more resistant to the effects of injury or illness. These so-called "hold" tests typically assess crystallized knowledge, such as vocabulary. These functions are more localized in the brain and less dependent on distributed white matter pathways, and therefore remain relatively intact with many types of neurologic insult (e.g., Traumatic Brain Injury, Multiple Sclerosis). Several measures are commonly used to assess premorbid abilities and these include word reading tests such as the National Adult Reading Test (NART), the reading subtest of the Wide Range Achievement Test (WRAT), Wechsler Test of Adult Reading (WTAR), and Wechsler subtests such as Vocabulary, Information, and Picture Completion. Of course, these methods would be inappropriate in populations with known language or reading disability, such as persons with aphasia, history of dyslexia, or advanced Alzheimer's disease. These methods would also be inappropriate for persons whose primary language is different from the language of the test.

A third method of estimating premorbid abilities has been the combination of demographic variables with current test scores. This method of combining demographics and current performance has demonstrated increased ability to predict performance above what using either demographics or current performance alone. For example, using WTAR performance combined with demographics variables has demonstrated an additional 4–7% ability to predict IQ and memory performance than using the WTAR alone (Psychological Corporation, 2001).

Cross References

- ▶ National Adult Reading Test
- ▶ Wechsler Adult Intelligence Scale (All Versions)
- ▶ Wechsler Test of Adult Reading
- ▶ Wide Range Achievement Test

References and Readings

Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). New York: Oxford University Press.

Ethical Principles in Forensics

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Synonyms

Ethics

Definition

Recent surveys have demonstrated that neuropsychologists are increasingly being asked to consult in forensic cases. As a result, they need to be aware of, and vigilant about, the associated ethical issues that arise. Grote (2005) notes that neuropsychologists have to be constantly vigilant of the need to produce unbiased and appropriately informed decisions if courts can be expected to rely on their opinions and that a failure to maintain this neutrality could lead others to view the field in a negative light, with particular biases directed toward the needs of the retaining party. Secondly, because neuropsychologists may not be fully aware of all of the potential ethical and legal implications that arise when reports are used in forensic settings, they may be especially at risk of committing an ethical violation without even being aware that such a violation has occurred. Some areas that are especially at risk for ethical violations in forensic practice include: release of raw test data and other test security issues, third-party observers, informed consent for assessment, and use of interpreters. For these, and other, reasons, neuropsychologists make efforts to educate themselves about the ethical obstacles that arise in the practice of forensic neuropsychology. At the very least, psychologists should be familiar with the most recent "Ethical Principles of Psychologists and Code of Conduct" (American Psychological Association, 2002) as well as the "Specialty Guidelines for Forensic Psychologists" (American Psychological Association, 1991)

Cross References

► Specialty Guidelines for Forensic Psychologists

References and Readings

American Academy of Clinical Neuropsychology (2007). AACN practice guidelines for neuropsychological assessment and consultation. *The Clinical Neuropsychologist, 21*, 209–231.

American Psychological Association (2002). Ethical principles of psychologists and code of conduct. *American Psychologist, 57*, 1060–1073.

Committee on Ethical Guidelines for Forensic Psychologists (1991). Specialty guidelines for forensic psychologists. *Law and Human Behavior, 15*, 655–665.

Grote, C. (2005). Ethical practice of forensic neuropsychology. In G. Larrabee (Ed.), *Forensic neuropsychology: A scientific approach.* New York: Oxford University Press.

Melton, G. B., Petrila, J., Poythress, N. G., & Slobogin, C. (2007). *Psychological evaluations for the courts* (3rd ed.). New York: Guilford Press.

Weissman, H. N., & DeBow, D. M. (2003). Ethical principles and professional competencies. In A. Goldstein (Ed.), *Handbook of psychology (Vol. 11). Forensic psychology,* Wiley: New Jersey. The 2002 APA "Ethical Principles" can be viewed in their entirety at: http://www.apa.org/ethics/code2002.html

Ethics

► Ethical Principles in Forensics

Ethics in the Practice of Neuropsychology

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Definition

Ethics provides a framework for understanding and examining morality, which broadly defined includes socially accepted norms about appropriate and inappropriate human conduct and encompasses consensual virtues, rights, and governing principles and rules. Ethics codes reflect the morality of specific groups, such as health care professionals. The American Psychological Association's Ethical Principles of Psychologists and Code of Conduct (APA, 2002) offers both guiding principles and ethical standards. Guiding principles reflect the values of the profession and are aspirational in nature. Conversely, ethical standards establish minimally acceptable professional behaviors and are enforceable codes of conduct whose ultimate purpose is protection of consumer welfare.

Clinical neuropsychologists who are APA members or whose state associations or licensing boards have adopted the APA Ethics Code are bound by its standards for professional conduct. Additionally, members of the Association of Postdoctoral Programs in Clinical Neuropsychology (APPCN) are bound by the APPCN Code of Conduct, which is meant to be complementary to the APA Ethics Code and focuses on ethical conduct and the process for addressing ethics complaints.

History

In 1938, the American Psychological Association (APA) created the ad hoc Committee on Scientific and Professional Ethics to evaluate the need for an ethics code. Although the 1940 committee report noted an ethics code was premature, the committee recommended that APA establish a standing committee to review complaints and formulate ethics rules as needed. By 1947, the same committee reported that the time had come for a formal code governing psychology practice (Peak, 1947). A formal process was undertaken to establish an ethics code based on Hobbs' recommendation that the development of such a code must include broad participation by APA members. Hobbs (1948) believed that the ethics code should be useful in everyday decision-making and not simply represent a blueprint for avoiding sanction. Therefore, the Committee on Ethical Standards for Psychology requested examples of ethical issues experienced by its members. Through classification and revision of categories of the critical incidents provided (i.e., the critical incident method), the first ethics code was created, which was initially recommended for adoption at the 60th Annual Business Meeting of APA (Adkins, 1952).

The APA Ethics Code has been revised 9 times, with the most recent changes having been adopted by the APA Council of Representatives in 2002 and implemented in 2003. (Previous code revisions have been extensively reviewed by Canter et al., 1994). Examples of changes incorporated in the 2002 code include clarification of multiple relationships, acceptability of release of test data to the client or others listed on the release form,

and enhanced sensitivity to cultural factors. Professional ethics codes are by nature evolutionary, reflecting changes in the profession and the sociocultural context in which the profession functions. There have been numerous works in neuropsychology discussing the application of the Ethics Code to clinical practice issues (e.g., Binder and Thompson, 1994; Bush, 2005).

Education and Training

Formal training (coursework and practical experience) in professional ethics occurs in all APA-accredited graduate training programs in psychology. Professional ethics is also included as part of the generic clinical core in education and training for clinical neuropsychologists. Postdoctoral residency requirements dictate that, upon completion, students should be eligible for board certification in clinical neuropsychology (Hannay et al., 1998). Professional ethics is currently one of three main components (the others being work sample review and fact finding) of the oral examination for certification by the American Board of Clinical Neuropsychology. Examinees are expected to be knowledgeable about the content of the APA Ethics Code and to apply the code's concepts to their neuropsychological practice. In addition to the Ethics Code, psychologists must maintain awareness of practice guidelines recommended within their specialty, which are commonly reflected in policy statements and position papers by the relevant APA division or related organizations. The American Academy of Clinical Neuropsychology, for example, posts policy statements on its web site, and recently published guidelines regarding neuropsychological assessment and consultation (Board of Directors, 2007). Previous guidelines have been provided by AACN, APA Divisions 22 (Rehabilitation Psychology) and 40 (Clinical Neuropsychology), and the National Academy of Neuropsychology (NAN) on a broad range of issues, such as third party observers, functional magnetic resonance imaging, and working with military veterans with traumatic brain injury.

Proficiency in professional ethics is required for licensure in every state. In order to address these requirements, APA, other professional organizations (e.g. International Neuropsychological Society), state psychological associations, and private companies all offer continuing education credit in applied ethics. Beyond these basic training requirements, training and supervision from experienced clinical neuropsychologists is important to ensure competence in the use of unfamiliar neuropsychological assessment tools. Both empirical and anecdotal reports suggest that appropriate use of assessments (in addition to boundaries of competence and ethical-legal issues) is a primary ethical concern among clinical neuropsychologists (Brittain, Frances, and Barth, 1995; Bush, 2007). Clearly, proficiency in ethics is an ongoing and lifelong process. Participating in continuing education, being aware of one's own strengths and weaknesses, and using mentors are all critical to ensure ethical competence.

Enforcement

The APA Ethics Committee publishes an annual report of its activities in the *American Psychologist*, including ethics case data (comparative data across several years) regarding ethics complaint adjudication. Enforcement decisions can potentially affect membership in the professional association. The process of enforcement also requires ongoing cooperative relationships with state Boards of Psychology, and in some cases the legal system. Ethics committees nested within state Boards of Psychology conduct their own investigations regarding ethical requirements of licensure, and many publish an annual list of license suspensions and revocations in order to inform the public. In a very real sense, enforcement regarding the APA Ethics Code reflects different levels of processing. At the state level, decisions relate to adherence to state statute and requirements regarding licensure, while at the national level ethics committee decisions impact ethical requirements of APA membership. Professional boards are also empowered to control their membership, with ethical violations being potential grounds for rescinding membership status. For example, ABCN has such authority.

Ethical Decision-Making

Ethics is as much about process as it is about content. Ethics codes and policy statements inform practice, but it is the individual psychologist who must thoughtfully weigh the factors involved in each unique clinical situation to reach an ethical decision. Several models exist that offer guidance in ethical analysis (e.g., Kitchener, 2000; Hanson, Kerkhoff, and Bush, 2005). For example, Hanson et al. (2005) illustrated a model for applied ethical decision-making in the context of a casebook relevant to psychology practice in different health settings (see Table 1). Collegial consultation during the process is strongly encouraged, particularly when faced with ambiguous, challenging, or emotionally charged issues.

Ethics in the Practice of Neuropsychology. Table 1 Ethics decision-making model (Hanson et al., 2005)

Steps	Titles	Details
I.	Principles, concepts, and standards	Identify the specific ethical principles, concepts and standards in conflict
II.	Context, stakeholder issues	Understand the events leading up to the ethical conflict, including historical and social context, stakeholders' roles and attributions
III.	Organizational/legal issues	Consider organizational rules/regulations and legal issues related to the conflict
IV.	Resolution	Synthesize information gathered to generate possible solutions based on balancing benefits and risks associated with each and desired outcome(s)
V.	Disposition	Select a course of action and implement it
VI.	Evaluation	Review the actual outcome(s) to facilitate learning and modify if necessary

Applications in Neuropsychology

The rich clinical neuropsychology literature in applied ethics reflects efforts to (1) thoughtfully deliberate on complex issues; (2) identify areas of ethical concern that enhance sensitivity to one's own behavior and/or lead to the formulation of practice guidelines; (3) provide guidance in applying the Ethics Code; (4) define parameters of individual professional conduct; and (5) ground practice issues in empirical analysis. This literature includes both theoretical and empirical work as well as positions taken by APA Division 40 (Clinical Neuropsychology) and related organizations (e.g., other APA divisions, neuropsychology specialty boards, clinical practice organizations) regarding specific aspects of neuropsychological practice. Bush (2005) provides comprehensive collections of commentary on ethical issues in neuropsychology.

Because of the range of ethical issues involved in neuropsychological practice, the APA ethical principles are used to organize the discussion below. These principles include beneficence and nonmaleficence (prevent harm/facilitate good or benefit; do no harm); fidelity and responsibility (establish and preserve trust; professionally responsible to the individual, community, and society); integrity (accuracy, honesty, and truthfulness); justice (provision of fair access to and quality of service); and respect for people's rights and dignity (respect client self-determination and individual differences). While by no means all encompassing, the issues discussed both characterize the current state of the field and foreshadow future ethical challenges.

Beneficence and Nonmaleficence: Competent neuropsychological evaluation can lead to correct diagnosis, effective advocacy, appropriate treatment and follow up, as well as support successful litigation. Conversely, inappropriate evaluation and poorly defined role boundaries can compromise both short-term and long-term client welfare. For example, with the burgeoning opportunities for neuropsychologists in forensic evaluation, the roles available (client's evaluator, legal consultant/expert witness, friend of the court, social advocate, etc.) must be carefully navigated to avoid potentially harmful multiple relationships. Similarly, "contingency fees, attempts by retaining parties to control examination procedures and findings,... test security and third-party observers" represent other ethical risks (Bush et al., 2008, p. 340). Thorough foundational training, continuing education, experience applying the APA Ethics Code, and setting appropriate role boundaries are all required to minimize ethical risk, especially when one faces the potentially adversarial nature of the legal system (Bush, 2005; Bush et al., 2008).

Fidelity and Responsibility: Psychologists have an ethical responsibility to stay abreast of the changing field of practice as well as to carefully consider the implications of new technology and data. For example, given the literature of the past 10 years, it would be professionally irresponsible to omit symptom validity assessment from a neuropsychological evaluation without an extremely clear justification (Bush, 2005; Iverson, 2006). Similarly, psychologists must remain vigilant regarding whether specific tests are normed adequately for diverse populations of interest. As another example, the advent of the internet provides a potentially exciting methodology for developing and modifying assessment tools and reaching underserved populations. However, adherence to well articulated psychometric principles remain paramount (Naglieri et al., 2004). By extension, neuropsychologists are challenged to preserve the valuable interaction

between observed behavior during the assessment process and test results, especially when neurological conditions are significantly played out through behavioral pathways. Achieving this critical interaction may be threatened by technological barriers inherent in distance assessment via the internet.

Integrity: In addition to skills acquisition, honest, accurate therapeutic engagement is a foundation for trust. Examples of ethically questionable behaviors threatening integrity include intentionally misconstruing or not being forthright about the amount of control one has over professional records, suppressing information not favorable to one's client, or misrepresenting credentials or findings for either the client's or personal gain. In addition, biases, such as those held toward specific cultures, and lack of knowledge can lead to inaccurate representation of one's skills (e.g., failing to present limitations regarding one's ability to interpret test results). What one does not know can harm the client; therefore, achieving and maintaining professional competence (Bush et al., 2008) and self-evaluation to insure unintentional biases and inconsistent practices do not interfere with appropriate ethical behavior are necessary. Consultation with colleagues can be particularly helpful in identifying biases and determining their impact on practice.

Justice: Justice is placed at risk when psychologists compromise or inequitably deliver services or avoid obligations. Neuropsychologists who choose to limit evaluations in the interest of cost control under-managed care must ethically defend the adequacy of battery comprehensiveness to address questions of interest. Individuals choosing to avoid the contentious legal process in order to sidestep potential ethical conflicts may well serve to harm a client needing the special expertise or information held by the forensic neuropsychologist. In addition, changing one's approach based on who is being represented (defense or prosecution) brings into question fair practice. Thorough knowledge of the intricacies of building a legal case and courtroom procedures will help the neuropsychologist avoid ethical pitfalls. To the extent that preservation of a balanced presentation of properly interpreted neuropsychological assessment data is achieved, justice can be facilitated.

Respect for People's Rights and Dignity: Acquiring informed consent and protecting confidentiality remain primary methods of respecting the autonomous rights of individuals served by clinical neuropsychologists. Both the 2002 APA Ethics Code and NAN offer guidance regarding appropriate consent acquisition (Johnson-Greene, 2005). Adequate informed consent must account for the individual's cognitive processing ability, prioritizing the components of the communication pertinent to the professional service to be rendered, the nature of the biological impairment that may call capacity into question, and cultural factors that may impact effective communication of information vital to allow consent. Careful inquiry into the individual's understanding of the issues, deliberation of alternatives and consequences, and demonstrated ability to communicate a decision remain primary goals in achieving adequate informed consent (Appelbaum and Grisso, 1988). Other issues of relevance may be the referral source and limits of confidentiality. More broadly, protecting confidentiality in small communities requires extra consideration, from office location to proactively agreeing upon how to respond to chance encounters. Through ethically appropriate and proactive management of consent and confidentiality issues, the neuropsychologist facilitates the individual's rights and welfare.

Future Challenges

The proliferation of tests and rapid expansion of our understanding of brain–behavior relationships challenge even the most conscientious psychologist to stay abreast of field developments. Defining and redefining one's area of competence will remain a challenge for all psychologists. Indeed, new neurophysiological, neurochemical, and interventional neurological technology will place even higher standards of specialized professional competence as brain–behavior concomitants of these technologies are used in research and practice (Steinbock, Arras, and London, 2009). Additionally, the explosion of computer-based technology outpaces our response to ethical concerns associated with its use/potential use for professional assessment and intervention. Similarly, medical advances will present new opportunities for which ethical guidance will not be well formulated. Intriguing ethical challenges inherent in emerging technologies in neurobiology and human enhancement relevant to neuropsychological practice and research will test current limits of ethical procedures and analyses. Balancing consumers' rights with consumer protection in an increasingly regulated society, as illustrated by the complexities of the release of test data provision under the 2002 Ethics Code, will also pose unique challenges in aspiring to uphold all the ethical principles. Finally, the impact of increasingly culturally diverse populations on competent neuropsychological practice has yet to be fully realized.

References and Readings

Adkins, D. C. (1952). Proceedings of the sixtieth annual business meeting of the American Psychological Association, Inc., Washington, D.C. *American Psychologist, 7*, 645–670.

American Psychological Association. (2002). Ethical Principles of Psychologists and Code of Conduct. *American Psychologist, 57*, 1060–1073.

Appelbaum, P., & Grisso, T. (1988). Assessing patients' capacities to consent to treatment. *New England Journal of Medicine, 319*, 1635–1638.

Beauchamp, T., & Childress, J. (2001). *Principles of biomedical ethics* (5th ed.). New York: Oxford University Press.

Binder, L. M., & Thompson, L. L. (1994). The ethics code and neuropsychological assessment practices. *Archives of Clinical Neuropsychology, 10*(1), 27–36.

Board of Directors, American Academy of Clinical Neuropscychology. (2007). American Academy of Clinical Neuropsychology (AACN) practice guidelines for neuropsychological assessment and consultation. *The Clinical Neuropsychologist, 21*, 209–231.

Bush, S. S. (Ed.). (2005). *A casebook of ethical challenges in neuropsychology.* New York: Taylor & Francis Publishers.

Bush, S. S. (2007). *Ethical decision making in clinical neuropsychology.* American Academy of Clinical Neuropsychology: Oxford University Press.

Bush, S. S., Grote, C. L., Johnson-Greene, D. E., & Macartney-Filgate, M. (2008). A panel interview on the ethical practice of neuropsychology. *The Clinical Neuropsychologist, 22*(2), 321–344.

Bush, S. S., Ruff, R. M., Troster, A. I., Barth, J. T., Koffler, S. P., Pliskin, N. H., et al. (2005). Symptom validity assessment: Practice issues and medical necessity. NAN Policy and Planning Committee. *Archives of Clinical Neuropsychology, 20*, 419–426.

Canter, M., Bennett, B., Jones, S., & Nagy, T. (1994). *Ethics for psychologists: A commentary on the APA Ethics Code* (1st ed.). Washington, DC: American Psychological Association.

Hannay, H. J., Bieliauskas, L. A., Crosson, B. A., Hammeke, T. A., Hamsher, K. deS., & Koffler, S. P. (1998). The Houston conference on specialty education and training in clinical neuropsychology. *Archives of Clinical Neuropsychology, 13*, 157–250.

Hanson, S., Kerkhoff, T., & Bush, S. (2005). *Health care ethics for psychologists: a casebook.* Washington, DC: APA Press.

Hobbs, N. (1948). The development of a code of ethical standards for psychology. *American Psychologist, 3*, 80–84.

Iverson, G. L. (2006). Ethical issues associated with the assessment of exaggeration, poor effort, and malingering. *Applied Neuropsychology, 13*(2), 77–90.

Johnson-Greene, D. (2005). Informed consent in clinical neuropsychology practice. Official statement of the National Academy of Neuropsychology. *Archives of Clinical Neuropsychology, 20*, 335–340.

Kitchener, K. S. (2000). *Foundations of ethical practice, research, and teaching in psychology.* Mahawah, NJ: Lawrence Erlbaum Associates.

Naglieri, J., Drasgo, F., Schmit, M., Handler, L., Prifitera, A., Margolis, A., et al. (2004). Psychological testing on the internet. *American Psychologist, 59*(3), 150–162.

Peak, H. (1947). Proceedings of the fifty-fifth annual business meeting of the American Psychological Association, Inc., Detroit, Michigan. *American Psychologist, 2*, 468–510.

Steinbock, B., Arras, J., & London, A. (2009). *Ethical Issues in Modern Medicine* (7th ed.), Boston, Massachusetts: McGraw-Hill Publishers.

ETT

► Endotracheal Tube

EuroQol Measure

► EuroQol/EQ-5D

EuroQol/EQ-5D

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Synonyms

EuroQol measure; EQ-5D

Description

Format and Subsections of the EQ-5D

The EQ-5D is a global measure of health status that consists of two main sections: the EQ-5D descriptive system, and the EQ Visual Analog Scale (VAS). The former contains five items covering mobility, self-care; usual activities such as work, study, housework, family, or leisure; pain/discomfort; and anxiety/depression, with the respondent choosing one of the three statements that best describes their state of health in that domain. The three response options are "no problems", "some problems," and "severe problems." For example, in the mobility section, the response options are as follows:

1. I have no problems in walking around
2. I have some problems in walking around
3. I am confined to bed

The EQ VAS is a 20-cm vertical line marked at 100 equidistant points, with every ten lines marked with a number from 0 ("worst imaginable health state") to 100 ("best imaginable health state"). The respondent marks the line at the point that represents their health state on that 1 day.

There is a third, optional section of the EQ-5D that consists of nine demographic questions (the socio-demographic questions, SDQ). The entire EQ-5D takes only a few minutes to complete.

Administration of the EQ-5D

The EQ-5D can be either self-rated with an interviewer present, rated through an interview (face-to-face or by telephone) or completed by proxy report. An Interactive Voice Response system has also been developed, which allows participants to enter their responses over an automated telephone system, in a range of languages, to a toll-free number (see www.euroqol.org for details). As of June 2009, there were official translations of the EQ-5D available in 102 languages, with more in development. A sample version of the measure can be found online, but for other language versions and to use the EQ-5D, permission must be obtained from the EuroQol Executive Office, which may include a license fee.

Scoring Responses to the EQ-5D

Responses to each item on the EQ-5D descriptive system are given a one-digit value, with responses to all five domains giving a five-digit code, describing the respondent's health state. The code 11111 therefore indicates no reported problems in any domain; 55555 indicates severe problems in every domain; and 11222 indicates moderate problems in usual activities, pain/discomfort, and anxiety/depression. There are 243 possible codes, along with two additional codes not obtained by means of the self-report measure, which represent an unconscious state and death. The VAS is scored according to the number closest to the line drawn by the respondent – a value between 0 and 100.

Responses from the EQ-5D can be presented in EQ-5D code format as a "health profile" demonstrating functioning in each of the five domains, either in terms of VAS scores as self-rated overall health or as a "weighted index" score. The procedure for obtaining weighted index scores differs according to the country in which the measure is being used, as the weights themselves are derived from studies, where groups representative of the general population in a given country have been asked to "value" particular health states. This valuation is obtained either by asking participants to assign a VAS value to a variety of theoretical health states or by the time trade-off (TTO) technique in which participants are asked to imagine they live in a certain health state for a period of 10 years, and then to specify the amount of time they would sacrifice to live in state 11111 instead.

Historical Background

The EQ-5D was developed by the EuroQol group, an international network of researchers set up in 1987, with the sole focus of developing a standardized, non-disease-specific measure of health-related quality of life, with the hope that this would enable cross-national comparisons to be made. It was designed to be quick and easy to administer, so that it could be used in studies that also needed more detailed disease-specific measures. The EuroQol group maintains records of researchers using the EQ-5D, and stages annual meetings regarding the relevant research. Over the years, the measure has been translated into many different languages, employed in a great number of studies across the world, and is used in a wide variety of clinical areas (see www.euroqol.org for a list of such areas).

Psychometric Data

Brazier, Jones, and Kind (1993) compared the EQ-5D with the SF-36 (another more detailed health status measure) in a group of greater than 1,000 members of the general population in the UK to examine the validity of the EQ-5D. There was a significant association between EQ-5D and SF-36 total scores; EQ-5D scores differed significantly between groups with and without chronic health conditions. Hurst, Kind, Ruta, Hunter, and Stubbings (1997), in a study of patients with rheumatoid arthritis, found significant correlations between disease-specific measures of health status and the EQ-5D. All of these findings provide evidence for the EQ-5D's validity, and other studies have found similar evidence in a variety of clinical conditions. However, Brazier et al. (1993) also found that the SF-36 was more sensitive to health problems than the EQ-5D, with the latter showing large ceiling effects in the general population.

Dorman, Slattery, Farrell, Dennis, and Sandercock (1998) found good test–retest reliability of EQ-5D responses in a study with people who had experienced a stroke, with kappa values for individual items ranging from 0.63 to 0.8 at a 3-week interval. They also found the value-weighted index and VAS to be internally consistent, with intra-class correlation (ICC) coefficients of 0.86 for both scales. Hurst et al. (1997) also reported that the test–retest reliability of the EQ-5D at 2-week and 3-month intervals in a group of people with rheumatoid arthritis was good (ICCs > 0.70).

Population norms from which to determine value-weighted scores are available for 15 different countries, based on data from between 400 and 6,000 respondents per country. These norms can be obtained from the EuroQol office.

Clinical Uses

As the EQ-5D provides a broad overall measure of health status, its utility on an individual clinical basis may be limited; one would most likely require a more detailed condition-specific instrument as well. However, the overall indication of health provided, particularly on the EQ VAS, could be a relatively useful and informal means of measuring change in perceived health over time or in response to treatment (though it should be remembered that both parts need to be administered to constitute the official EQ-5D). The descriptive system may be a useful outcome measure in clinical trials, but again, its scope means it could well be insensitive to small changes in health status.

The EQ-5D has been quite heavily criticized in terms of its valuation system, with these criticisms principally relating to the methods of obtaining such valuations (the VAS and TTO methods, each having their own limitations), and the resulting validity of those valuations. However, these criticisms relate to the weighted index score and other EQ-5D-derived valuations of health status, most often relevant in health economics. They do not relate to the EQ-5D scores most relevant in clinical neuropsychology research and practice, that is, the descriptive system, and VAS ratings of one's current state of health.

Cross References

- ▶ General Well-Being Schedule
- ▶ SF-36/SF-12

References and Readings

Brazier, J., Jones, N., & Kind, P. (1993). Testing the validity of the EuroQol and comparing it with the SF-36 health survey questionnaire. *Quality of Life Research, 2*, 169–180.

Cheung, K., Oemar, M., Oppe, M., & Rabin, R. (2009). EQ-5D user guide. Downloaded from http://www.euroqol.org/eq-5d/what-is-eq-5d/how-to-use-eq-5d.html 13.07.2009 at 12.20 pm.

Dorman, P., Slattery, J., Farrell, B., Dennis, M., & Sandercock, P. (1998). Qualitative comparison of the reliability of health status assessments with the EuroQol and SF-36 questionnaires after stroke. *Stroke, 29*, 63–68.

Hurst, N. P., Kind, P., Ruta, D., Hunter, M., & Stubbings, A. (1997). Measuring health-related quality of life in rheumatoid arthritis: Validity, responsiveness and reliability of EuroQol (EQ-5D). *British Journal of Rheumatology, 36*, 551–559.

The EuroQol website (www.euroqol.org) contains much useful information on the EQ-5D, as well as guidance towards locating a variety of more in-depth sources of information.

Event-Related Paradigms

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Synonyms

Auditory and visual evoked potentials; ERP's; Evoked potentials

Definition

Event-related paradigms (ERPs) are time locked and stereotyped brain responses to some "event." The "event" is a stimulus that evokes an electrophysiological response. The stimulus can be a sound, a simple visual pattern, a mental event or thoughts such as recognition of a specific target stimulus or the absence of a stimulus as when an increased time elapses between stimuli.

The recorded brain response is a very small electrical voltage of between 1 and 100 millionths of a volt and is recorded over the scalp by a very sensitive amplifier and computer averaging equipment that can recognize it as a consistency in the seemingly random waveforms of the overlying EEG. The response can be a singular waveform or a series of waveforms that indicate the chain of processors that respond in sequence to the stimulus as the response travels along the pathway from the primary receptor to the cortex of the brain. The location of the activity on the scalp is related to the pathway taken by the response from the primary processor to the specific area of cortex that is processing that stimulus.

Responses that occur from 0 to 200 ms are called "exogenous" and are felt to be the direct result of the presented stimulus. Responses occurring after 200 ms are called 'endogenous' and are felt to be correlated with the conceptual manipulation of the stimulus, the thought.

Current Knowledge

There are both clinical and research areas where ERPs can be used. The clinical uses evaluate the integrity of the neural processors that respond to the stimulus. Abnormal timing or amplitude of the waveforms may indicate damage or dysfunction in the brain tissues that should be activated. Some of the more common clinical response patterns are the Auditory Brain Response (ABR), the Mid Latency Response (MLS), and the "slow" and "late" cortical auditory ERPs.

The ABR is seen 1.5–15 ms from the stimulus. This response is the generated activity of the brain stem auditory structures in the pathway from eighth cranial nerve to the lateral lemniscus and inferior colliculus of the upper brain stem. The MLS (25–50 ms) originates in the upper brain stem and goes to the auditory cortex. The "slow" cortical auditory ERPs (50–200 ms) are generated in the auditory cortex. The "later" cortical auditory ERPs, especially the "mismatch negativity," indicate a *change* in the characteristics of the stimulus.

ERPs are valuable in research. They are very high in 'temporal' resolution and can reveal minute changes in sensory and cognitive brain responses to stimulus tasks and can also detect results of a thought or perception. Some of the areas where ERPs have been studied include attention and information processing (P50), emergence from the vegetative and minimally conscious states, and receptive memory.

While ERPs are being recorded, fMRI can provide anatomic localization of these processes.

Cross References

► fMRI
► P300

References and Readings

Fabiani, M., Gratton, G., & Federmeier, K. D. (2007). Event-related brain potentials: Methods, theory, and applications. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 85–119). Cambridge: Cambridge University Press, ISBN 0-521-84471-0.

Faran, S., Vatine, J. J., Lazary, A., Ohry, A., Birbaumer, N., & Kotchoubey, B. (2006). Late recovery from permanent traumatic vegetative state heralded by event-related potentials. *Journal of Neurology, Neurosurgery, and Psychiatry, 77*, 998–1000, doi:10.1136/jnnp.2005.076554.

Handy, T. C. (2004). *Event-related potentials: A methods handbook.* Cambridge, MA: The MIT Press (B&T), ISBN 0262083337. http://www.emedicine.com/neuro/topic69.html.

Kemp, A. H., Hopkinson, P. J., Hermens, D. F., Rowe, D. L., Sumich, A. L., Clark, C. R., Drinkenburg, W., Abdi, N., Penrose, R., McFarlane, A., Boyce, P., Gordon, E., & Williams, L. M. (2008). Fronto-temporal alterations within the first 200 ms during an attentional task distinguish major depression, non-clinical participants with depressed mood and healthy controls: A potential biomarker? *Human Brain Mapping.*

Luck, S. J. (2005). *An introduction to the event-related potential technique.* Cambridge, MA: The MIT Press, ISBN 0262621967.

Vanhaudenhuyse, A., Laureys, S., & Perrin, F. (2007). Cognitive event-related potentials in comatose and post-comatose states. *Neurocritical Care,*

Woollams, A., Taylor, J. R., Karayanidis, F., & Henson, R. N. (2008). Event-related potentials associated with masked priming of test cues reveal multiple potential contributions to recognition memory. *Journal of Cognitive Neuroscience, 20*, 1114–1129.

Event-Related Potentials

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Synonyms

Cerebral evoked potentials

Definition

Usually called EVOKED POTENTIALS when the stimulus is regular, programmed using signal-averaging techniques. Signal-averaging techniques will eliminate background noise and preserve the potential generated. However, evoked potentials (EP) are but a subset of event-related potentials. Stimulation of special peripheral sensory nerve systems directly or indirectly generates cerebral potentials. The stimulation – at a regular, random, or irregular rate through time – is the event. The type of stimulation could be visual, auditory, mechanical, electric, or a combination. Event-related potentials are relatively noninvasive and complications/sequella of these procedures are infrequent and mild. The cost of these procedures is but a small fraction of MRI and CT scan studies. Both the latency and the pattern, shape of the EPs are associated with the type of stimulus and are typically recorded on the scalp. Normal values have long been established for each EP. However, as these EPs are

placed within background EEG activity and characteristically have low voltage, computer averaging of thousands of responses is required. Fortunately, digital equipment requires but the press of a button to be activated. Lesions within the central nervous system (CNS) which disrupt these special sensory pathways will generate an abnormal EP and thus help confirm both the presence and/or location of that lesion. Each EP has components which could be generated by a different part of the CNS and therefore could be employed in mapping the lesion. Somatosensory EPs have been used to determine nerve conduction velocity values. An EP can also be monitored during an invasive or surgical procedure to see if an affected spinal nerve root entity is still functioning. Irregular event-related potentials have also been used to determine normal and clinically abnormal dermatome patterns using tactile stroking movements. For example, after closed head injury with associated inner ear damage, absence of wave I using brainstem auditory EPs will occur. In patients with coma, using somatosensory EPs to demonstrate loss of activity after P15 will usually predict death.

Current Knowledge

Somatosensory cerebral evoked potentials are useful to determine sensory nerve conduction velocity within the CNS.

Cross References

► Cerebral Cortex
► Somatosensory System

References and Readings

American Electroencephalographic Society. (1994). Guidelines on evoked potentials. *Journal of Clinical Neurophysiology, 11*, 40–73.
Aminoff, M. J. (1990). Evoked potential studies in neurological diagnosis and management. *Annals of Neurology, 28*, 706–710.
Rosenfeld, J. V., & Lennarson, P. J. (2007). *Coma and brain death.* In Schapira, A. H. V. et al., (Eds.), *Neurology and clinical science* (pp. 97–116). Philadelphia: Mosby.

Evidence Based Medicine

► Evidence Based Practice

Evidence Based Practice

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Synonyms

Evidence based medicine

Definition

Evidence based practice has been defined as "the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients. It is the practice of integrating individual clinical expertise with the best available external clinical evidence from systematic research."

Current Knowledge

Evidence based medicine requires that a practitioner asks questions, researches the relevant information, evaluates the data for its validity and usefulness, and determines whether or not the information reviewed is appropriate for implementation in his/her specific clinical practice. Assessment of validity involves four questions: (1) Was there an independent, blind comparison with a reference standard of diagnosis? (2) Was the diagnostic test evaluated in an appropriate population of patients comparable to the patient of interest? (3) Was the reference standard applied regardless of the diagnostic test result? (4) Was the test validated in a second, independent group of patients?

The topic of EBM has been criticized by some because even the most well thought out controlled experimental research study cannot control for all variables. What has been found to be true for one subgroup of patients across studies may not be able to be applied to all patients because of differing ethnicity, age, cultural, social, and economic factors. Treatment recommendations also are no longer just based on levels of evidence, but also on risk benefit ratio and cost. Therefore, evidence based practice should not supplant the practitioner's own clinical judgment and expertise, but rather should be used, along with patient preference, as an adjunct in order to provide optimal care for their patient population.

References and Readings

Fisher, C., & Wood, K. (2007). Introduction to and techniques of evidence-based medicine. *Spine, 32*(Suppl. 19), 66–72.

Hawkins, R. (2005). The evidence based medicine approach to diagnostic testing: Practicalities and limitations. *The Clinical Biochemist Reviews, 26*, 7–18.

Marwick, C. (1997). Proponents gather to discuss practicing evidence-based medicine. *Journal of the American Medical Association, 278*(7), 531–532.

Sackett, D. L., Rosenberg, W. M. C., Gray, J. A. M., Hayne, R. B., & Richardson, W. S. (1996). Evidence based medicine: What it is and what it isn't. *BMJ, 312*(7023), 71–72.

Evoked Potentials

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Definition

Evoked potentials are waveforms that can be recorded from a peripheral nerve, spinal cord, or cerebral cortex after stimulation of a peripheral nerve. They are named based on the pathways stimulated. Somatosensory evoked potentials (SSEPs) are recorded after stimulation of upper or lower extremity peripheral nerves. Visual evoked potentials (VEPs) are cortical responses measured after visual input. Brainstem auditory evoked potentials (BAEPs) are cortical responses measured after timed auditory stimuli.

Historical Background

Current Knowledge

Evoked potentials have many clinical and research implications. Using sensory and motor pathways, intraoperative monitoring can be used to follow functional integrity of pathways during spinal and intracranial neurosurgical procedures. SSEPs can also be used diagnostically to delineate the location of lesions, such as peripheral nerve or spinal cord. The use of cortical evoked potentials, such as VEPs and BAEPs, can also supplement neuroimaging information in diagnostic working-up for diseases such as multiple sclerosis. Short latency evoked potentials, such as SSEPs, VEPs, and BAEPs, have been studied as prognostic indicators in traumatic and anoxic brain injury. Absence of responses early after anoxic brain injury accurately predicts mortality due to the injury. Abnormal responses are also predictive of failure to emerge from coma in traumatic brain injury. Normal responses can be predictive of good outcomes both in traumatic and anoxic brain injury. Electrophysiologic recovery often precedes clinical recovery, making these tests valuable.

Recent research has focused on the use of event-related potentials (ERPs). These are long latency responses, occurring more than 70 ms after sensory stimulation. In contrast to evoked potentials, ERPs are reflective of changes in the electrophysiology of the brain as it performs a cognitive task. They can be used to define areas of pathology in the diseases of central nervous system, such as Alzheimer's disease and mild cognitive impairment. Finally, ERPs may have clinical utility in diagnosis of psychiatric disease such as schizophrenia as well as assistance in prediction of response to pharmacotherapy.

Cross References

▶ Event-Related Paradigms
▶ Somatosensory Evoked Potentials
▶ Visual Evoked Potentials

References and Readings

Claussen, J., & Hansen, H. C. (2001). Early recovery after closed traumatic head injury: Somatosensory evoked potentials and clinical findings. *Critical Care Medicine, 29*, 675–677.

Lew, H. L., Poole, J. H., Castaneda, A., Salerno, R. M., & Gray, M. (2006). Prognostic value of evoked and event-related potentials in moderate to severe brain injury. The *Journal of Head Trauma Rehabilitation, 21*, 350–360.

Pogarell, O., Mulert, C., & Hegerl, U. (2007). Event-related potentials in psychiatry. *Clinical EEG and Neuroscience, 38*, 25–34.

Robinson, L. R., Micklesen, P. J., Tirschwell, D. L., & Lew, H. L. (2003). Predictive value of somatosensory evoked potentials for awakening from coma. *Critical Care Medicine, 31*, 960–967.

Sala, F., Manganotti, P., Tramontano, V., Bricolo, A., & Gerosa, M. (2007). *Neurophysiologie Clinique, 37*, 399–406.

Taylor, J. R., & Olichney, J. M. (2007). From amnesia to dementia: ERP studies of memory and language. *Clinical EEG and Neuroscience, 38*, 8–17.

Ewing Sarcoma Family Tumors (ESFT)

▶ Ewing's Sarcoma

Ewing's Sarcoma

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Synonyms

Ewing sarcoma family tumors (ESFT); Peripheral primitive neuroectodermal tumors (pPNET)

Definition

Ewing sarcoma is named after Dr. James Ewing, who first described this tumor in the 1920s. It occurs primarily in bone or soft-tissue and is most often found in the pelvis, femur, tibia, and ribs. It is one of the most common types of pediatric cancers and is usually found in children and young adults between the ages of 10 and 20, and rare past the age of 30. There is a slightly higher incidence in males than females. Pain is usually the earliest symptom and can be accompanied by swelling and fever. Nerve and spinal cord compression may cause additional symptoms such as numbness, tingling, incontinence, and paralysis. This type of tumor often metastasizes to other parts of the body. It is usually malignant with unfavorable prognosis, particularly in those with metastatic disease. Treatment often involves whole-body chemotherapy, localized surgery, and/or radiotherapy. In children, possible late delayed effects of radiotherapy may include slowed bone growth and infertility.

Cross References

- ▶ Late-Delayed Effects of Radiation
- ▶ Metastasis
- ▶ Neoplasm
- ▶ Primitive Neuroectodermal Tumor
- ▶ Radiotherapy
- ▶ Sarcoma

References and Readings

Ewing, J. (1921). Diffuse endothelioma of bone. *Proceedings of the New York Pathological Society, 21*, 17–24.

Exacerbating–Remitting Multiple Sclerosis

▶ Relapsing–Remitting Multiple Sclerosis

Exaggerating

▶ Malingering

EXAMINER

▶ Executive Abilities: Methods and Instruments for Neurobehavioral Evaluation and Research

Excessive Alcohol Use

▶ Alcohol Abuse

Excessive Sleepiness

▶ Hypersomnia

Executive Abilities: Methods and Instruments for Neurobehavioral Evaluation and Research

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Synonyms

EXAMINER

Description

The EXAMINER is an NIH-sponsored project to develop a neuropsychological test battery that reliably and validly

assesses executive functions (often defined as the ability to engage in goal-oriented behavior) for clinical investigations and clinical trials, and that is adaptable across a wide range of ages and disorders. Executive function (EF) refers to a constellation of cognitive abilities that include the ability to plan, organize, manage multiple tasks simultaneously, mental flexibility, inhibition, and self-monitoring. These important behaviors are compromised in healthy elders and in people with neurological, mental and age related disorders, leading to disruption of lifestyle and loss of independence. One obstacle for progressive research in this area of cognition is a paucity of valid and reliable tasks that specifically tap domains of EF. The EXAMINER project is working to create a battery of domain specific executive function tasks that will make it easier for clinical researchers to measure these constructs in standardized ways. Cross-cultural issues are also important; the resulting tool will be available in Spanish.

Although there is a general consensus that executive abilities are a central component of human cognition, there is little agreement about what these executive abilities are, how they are organized, what their relationships are with frontal brain regions, or how they should be measured. Presently, three separate approaches are integrated to conceptualize and measure executive functioning. The first approach is to parse executive ability into more discrete and measurable constructs such as working memory, set shifting, fluency, and inhibition. The second approach is to utilize tasks that may be cognitively more complex, but more strongly resemble tasks that subjects encounter in daily life (e.g., insight, decision making, social cognition). The third approach is to apply informant-based rating scales. Measures reflecting these three approaches are described below.

Discrete Constructs

EF has been subdivided into more discrete subunits that are only modestly correlated with one another. These subunits include working memory, set shifting, fluency, and inhibition.

Working Memory

Spatial working memory: Spatial working memory is assessed using the *n*-back paradigm. The *n*-back is composed of two subtests: the 1-back and the 2-back. In each subtest, the participant views a series of squares that vary in location. In the 1-back, the participant indicates via a keyboard press whether each square is in the same location as the immediately preceding square. This is a test of spatial attention. In the 2-back, the participant indicates whether each square is in the same location as the square 2 before. This is a test of spatial working memory and relies on flexible updating capabilities.

Verbal working memory: Verbal working memory is assessed with a dot counting task. Subjects count the number of blue circles on a computer screen filled with blue and green circles and squares. These screens are presented in series that range in length from two to six screens. At the end of each series of screens, subjects state the number of blue circles on each screen in that series. The length of the series ranges from two to six.

Set Shifting

General and specific shift costs: This task is computer-administered. In task-homogeneous blocks, participants perform either Task A (e.g., classifying shapes) or Task B (e.g., classifying colors). In task-heterogeneous blocks, participants alternate between the two tasks pseudo-randomly. The combination of task-homogeneous and task-heterogeneous blocks allows measurement of general switch costs (latency differences between heterogeneous and homogeneous blocks) and specific switch costs (latency differences between switch and non-switch trials within heterogeneous block).

Fluency

Verbal fluency: Phonetic verbal fluency (words generated in 60-s beginning with the letters F and L); semantic fluency (animals and vegetables).

Inhibition

Flanker test: In this computer-administered task, the target stimulus is a centrally presented arrow facing either to the left or right. Subjects are instructed to identify the direction of this central arrow by pressing one key for the left direction and a different key for the right direction. The targets can be flanked on either side by two arrows that are either facing in the same direction (congruent condition) or in the opposite direction (incongruent condition). The capacity for inhibition is reflected in the latency differences between the congruent and incongruent conditions.

Continuous performance/Go-No Go: In this computer-administered task, there are four blocks of trials consisting of 48 successively presented stimuli. The ratio of targets to non-targets varies across the four blocks of trials, ranging from 5:1 to 1:5.

Anti-saccade: There are two blocks of trials in which subjects look at a fixation point in the center of a

computer screen and move their eyes upon presentation of a laterally presented stimulus. In the first block, subjects are instructed to move their eyes in the direction of the presented stimulus. In the second block (anti-saccade), subjects are instructed to move their eyes in the opposite direction of the presented stimulus.

Random number generation: Subjects are asked to generate a set of 100 numbers randomly, avoiding any obvious or predictable patterns.

Cognitively Complex Tasks

Insight: Subjects are asked to rate themselves on their performance immediately after completing the verbal fluency tasks.

Complex task of decision-making: This is an unstructured task modeled after the 6-elements test. Subjects are presented with three booklets, each containing five pages of simple puzzles (six per page). The puzzles were designed to be cognitively quite simple (e.g., connect the dots; trace the design) but require on average between 4 and 80 s to complete. Each puzzle has a designated dollar value. Subjects are given 6 min to earn as many dollars as possible. The cost-benefit ratio of the puzzles varies systematically, requiring subjects to plan ahead and to avoid items that have a large reward value but are strategically poor choices. In addition, the average cost-benefit ratio of the items increases as a subject progresses through a book, requiring further strategic decisions about when to switch booklets.

Social Cognition

The Awareness of Social Inference Test (TASIT): This modified version of the TASIT contains items from the first subtest, called the Emotion Evaluation subtest. This is designed to assess subjects' emotion comprehension with dynamic, ecologically valid stimuli. Subjects must interpret naturalistic emotional displays that include multiple matched modalities of emotional expression, including facial movement, voice prosody, and upper body posture and gestures. Subjects watch brief (20-s) vignettes of professional actors depicting one of five basic emotions (surprised, sad, fearful, angry, and disgusted) with a semantically neutral script. The facial expressions in these videos have also been FACS-coded to ensure reliability and validity of emotional expression. After watching each video, subjects are asked to select the correct emotion from a screen with the five emotions written on it. This subtest includes 20 vignettes, four for each of the five emotions.

Social norms questionnaire (SNQ): The SNQ measures subjects' crystallized knowledge of social norms in a linguistically and cognitively simple manner. This 20-item yes-no questionnaire is designed to determine the degree to which subjects actually understand and can accurately identify implicit but widely accepted social boundaries in the dominant US culture. This social norms questionnaire includes both inappropriate behaviors (e.g., "Cut in line if you are in a hurry," "Pick your nose in public," and "Wear the same shirt every day") and generally acceptable behaviors (e.g., "Tell a coworker your age," "Blow your nose in public," and "Eat ribs with your fingers,"). The subject must decide whether the behavior is socially appropriate or not. This measure also has an alternate form for test-retest purposes.

Revised Self-Monitoring Scale (RSMS): In order to measure subjects' awareness of their own social behavior, their own self-reported degree of self-concern and self-focus will be obtained using the Lennox and Wolfe version of the Revised Self-Monitoring Scale informant-based reports. The RSMS is a 13-item measure of the subjects' sensitivity to the expressive behavior of others, and their ability to monitor their self-presentation. The subjects' responses will be compared to those of their informants (see below) as an indirect measure of how accurate their self-assessment is.

Informant-Based Reports

The Frontal Systems Behavior Scale (FrSBe): The FrSBe is a 46-item behavior rating scale that is intended to measure behavior associated with damage to frontal systems. It consists of two rating forms: a self-rating form to be completed by the patient and a family rating form to be completed by an informant who has regular contact with the patient. Each FrSBe form yields a total score and scores for subscales measuring apathy, disinhibition, and executive dysfunction. Scores are obtained on each scale for baseline behavior and current behavior.

The Behavior Rating Inventory of Executive Functions (BRIEF): The BRIEF is an inventory designed to measure executive dysfunction in children aged 5–18. It produces two indexes composed of several clinical subscales. The Behavioral Regulation Index is made up of the Inhibit, Shift, and Emotional Control subscales. The Metacognition Index is made up of the Initiate, Working Memory, Plan/Organize, Organization of Materials, and Monitor subscales. There is also a Global Executive Composite score incorporating all eight clinical scales.

The Interpersonal Reactivity Index (IRI): The IRI is a questionnaire measure of both the cognitive and the affective components of empathy. Its 28 items include two 7-item subscales measuring cognitive empathy, perspective taking (PT: the tendency to spontaneously imagine the cognitive perspective of another person)

and fantasy (FS: the tendency to project oneself into the place of fictional characters in books and movies), as well as 27-item subscales measuring emotional empathy, empathic concern (EC: the other-centered emotional response resulting from the perception of another's emotional state) and personal distress (PD: the self-centered emotional response involving fear or distress that results from witnessing another's stressful circumstances or negative emotional state). Theoretically, the PD subscale reflects a primitive form of empathy that actually interferes with an effective empathic response; thus it tends to drop as the other scales rise (particularly in relation to the PT scale), and is negatively related to measures of overall social functioning. Higher scores on the PT, FS, and EC scales are associated with a more highly developed capacity for empathy.

Revised Self-Monitoring Scale (RSMS): In order to measure subjects' awareness of their own social behavior, informant-based reports will be obtained on the Lennox and Wolfe version of the Revised Self-Monitoring Scale, 13-item measure of the patient's degree of self-concern and self-focus. This questionnaire measures the subject's sensitivity to the expressive behavior of others, and their ability to monitor their self-presentation. This questionnaire will also be filled out by the subjects about themselves (see above), and their responses will be compared to those of their informant as another, indirect measure of how accurate their self-assessment is.

Historical Background

Executive function is of increasing interest and importance in cognitive neuroscience and clinical assessment. The frontal lobes represent over 30% of the cortical surface and play a major role in the organization of behavior and cognition. The functions represented in this vast cortical expanse are complex and multi-faceted, often categorized under the category of "executive control." Simple theories regarding a unitary function of the frontal lobes have been supplanted by sophisticated cognitive and neuroscience research that has helped to fractionate the broad term "executive control" into multiple sub-components bound together to help with the organization, initiation and control of cognition and behavior. Additionally, it is now evident that executive control is not anatomically restricted to the frontal lobes but also depends upon the intactness of subcortical white matter and the basal ganglia, and neural networks that rely on input from posterior structures (Ravizza & Ciranni, 2002).

Despite these advances, clinical investigators are faced with several challenges. For example, while the importance of preserved executive abilities for daily living is widely recognized, most neuropsychological test batteries for clinical trials do not include measures of executive ability, ignore social cognitive aspects of executive control, and do not link executive function to changes in daily living. In addition, the sheer number of available tasks reputed to measure executive function is overwhelming. It has also become clear that simple paper and pencil tasks will not always capture the real-life social and executive deficits in patients with ventromedial prefrontal injury. Tasks that capture deficits in executive control in the areas of social-cognition and activities of daily living are needed. The psychometric properties of executive tasks pose yet another challenge to clinical investigators interested in measuring executive functioning. Construct validity refers to how well an instrument measures what it purports to measure. Clinical neuropsychological instruments have been criticized for being multifactorial, drawing on several non-executive component skills. In fact, several of the top 20 "executive tasks" listed by Rabin, Barr and Burton (2005) in their neuropsychologists' survey were a memory task (CVLT) and visuospatial tasks such as clock drawing, Rey–Osterrieth Complex Figure, and block design. Another psychometric issue is test-retest reliability. This has particular importance for clinical trials where researchers must be able to attribute change in cognition to the intervention and not poor reliability or practice effects (Beglinger et al., 2005; Bowden, Benedikt, & Ritter, 1992).

Most current measures of executive function also have limited cross-cultural application. Often the stimuli require reasonable mastery of English (e.g., Similarities; Stroop; D-KEFS Card Sorting) or are culturally based (e.g., proverb interpretation). Executive tasks also tend to be highly correlated with education, and education levels vary across ethnic groups. Even when tasks appear to be readily translatable, assessment of differential item functioning has revealed item bias (Marshall, Mungas, Weldon, Reed, & Haan, 1997). The shortage of validated clinical measures that are applicable across ethnic and language groups poses a major obstacle to clinical research.

In sum, despite the wealth of available instruments, there are continued concerns about psychometric properties, validity, generalizability to settings and populations other than the ones they were developed for, suitability for all ages and non-English speaking subjects, and adaptability for clinical trials. There is also no consensus on what the primary components of executive functioning are or how terms are defined. There remains a compelling

need to have a battery of tests that can be routinely integrated into neurobehavioral research that will reliably and validly measure constructs that clinical investigators agree are important.

Psychometric Data

Psychometric properties of the EXAMINER are to be determined.

The EXAMINER project is currently in year three of a 5 year contract ending December 2010. Data collection is ongoing at ten academic and clinical sites. To date, there have been approximately 540 participants in the EXAMINER project. Roughly half of the participants are normal controls and half have been clinically diagnosed with conditions included in our study design. Diagnoses include Attention-Deficit Hyperactivity Disorder, Alzheimer's disease, mild cognitive impairment, frontotemporal dementia, Huntington's disease, Parkinson's disease, multiple sclerosis, progressive supranuclear palsy, sickle cell anemia, traumatic brain injury, focal lesions, and very low birth weight children. Currently, 22% of participants are Hispanic, 12% are African-American, 49% are Caucasian, and 17% are of other ethnicities.

Clinical Uses

The EXAMINER will reliably and validly assess executive function for clinical investigations and clinical trials. The test battery will be adaptable across a wide range of ages and disorders.

Cross References

► Behavioral Assessment of Dysexecutive Syndrome (BADS)
► Cambridge Neuropsychological Test Automated Battery (CANTAB)
► Delis–Kaplan Executive Function System (D-KEFS)
► NEPSY-II

References and Readings

Beglinger, L. J., Gaydos, B., Tangphao-Daniels, O., Duff, K., Kareken, D. A., Crawford, J., et al. (2005). Practice effects and the use of alternate forms in serial neuropsychological testing. *Archives of Clinical Neuropsychology, 20*(4), 517–529.

Bowden, S. C., Benedikt, R., & Ritter, A. J. (1992). Delayed matching to sample and concurrent learning in nonamnesic humans with alcohol dependence. *Neuropsychologia, 30*(5), 427–435.

Marshall, S. C., Mungas, D., Weldon, M., Reed, B., & Haan, M. (1997). Differential item functioning in the mini-mental state examination in English- and Spanish-speaking older adults. *Psychology of Aging, 12*(4), 718–725.

Miller, B. L., & Cummings, J. L. (2007). *The human frontal lobes, second edition: Functions and disorders*. New York: The Guildford Press.

Rabin, L. A., Barr, W. B., & Burton, L. A. (2005). Assessment practices of clinical neuropsychologists in the United States and Canada: A survey of INS, NAN, and APA Division 40 members. *Archives of Clinical Neuropsychology, 20*(1), 33–65.

Ravizza, S. M., & Ciranni, M. A. (2002). Contributions of the prefrontal cortex and basal ganglia to set shifting. *Journal of Cognitive Neuroscience, 14*(3), 472–483.

Executive Functioning

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Synonyms

Executive processes

Definition

Executive functioning refers to higher-level abilities in the following areas: planning, problem-solving, attention, mental flexibility, initiation, judgment, inhibition, and abstract reasoning. This set of abilities is most commonly associated with the frontal lobe region of the brain. It should be noted that executive functioning is not exclusive to the frontal lobes and is thought to extend to other regions of the brain.

Current Knowledge

Most of the current knowledge surrounding executive functioning has resulted from studying individuals with frontal lobe lesions. Executive functioning process may be disrupted by traumatic brain injury, other cerebral lesions, substance abuse, and maternal substance abuse during pregnancy. These disruptions may be manifested in personality changes or adaptive functioning. For

example, a person who experiences brain damage that impacts his or her executive processes may instantly, or over a period of time, begin to display symptomology that deviates from the ways he or she may have previously been characterized (e.g., cognitions, behaviors, emotions). The developmental trajectory spans through early adulthood.

References and Readings

Dise-Lewis, J. E., Calvery, M. L., & Lews, H. C. (2002). BrainStars

D'Amato, R. C., Fletcher-Janzen, E., & Reynolds, C. R. (2005). *Handbook of school neuropsychology.* Hoboken, New Jersey: Wiley.

Jurado, M., & Rosselli, M. (2007). The elusive nature of executive functions: A review of our current understanding. *Neuropsychological Review, 17*, 213–233.

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

Executive Interview

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Synonyms

EXIT; EXIT15; EXIT-25; TEXAS

Description

The Executive Interview (EXIT25) (Royall, Mahurin, & Gray, 1992) provides a brief, clinical-based, bedside measure of executive control functions (ECF). EXIT25 scores have been used to identify the presence and severity of executive impairments and to predict problems in self-care, functional status, decision-making capacity, and problem behavior. The EXIT25 has been validated mainly in elderly persons but has also been used in a wide range of other populations, including medical patients, psychiatric patients, community samples, and as an outcome measure in clinical trials. It is available in many languages including English and Spanish, but published validation studies exist only for Dutch, Brazilian Portuguese, and Cantonese Chinese translations.

The EXIT25 is a compilation of 25 items that target motor, behavioral, and cognitive features associated with frontal systems dysfunction. Items include verbal and design fluency, an aural Trail Making task, repetition of anomalous sentences (e.g., "Mary fed a little lamb"), a stroop-like interference task, a go/no-go task, Luria hand sequences, etc. Additionally, neurological procedures are incorporated (e.g., to elicit paratonia, grasp reflex, snout reflex, and echopraxia). Thus, the EXIT25 provides an opportunity for the examiner to document a wide range of frontal-type clinical features, including perseveration, disinhibition, motor impersistence, and intrusions. Moreover, the "interview" is choreographed to facilitate the examiner–testee interaction, and to support test–retest and inter-rater reliability. Shortened versions (e.g., the EXIT15 and the Telephone Executive Assessment Scale [TEXAS]) have been developed for specific applications but are less well studied.

The EXIT25 takes 10–15 min to administer. The items are presented "in rapid succession and with minimal instruction, which allows little time for reflection and therefore may enhance any tendency of disinhibition or inappropriate responses" (Stokholm, Vogel, Gade, & Waldemar, 2005, p. 1578). Each item uses a three-point response scale: 0 (intact performance), 1 (a specific partial error or equivocal response), and 2 (specific incorrect response or failure to perform). Total score ranges from 0 to 50, with higher scores indicating greater impairment. A cut-off score of 15/16 is recommended.

In a brief, practical bedside format, the EXIT25 provides a reliable, sensitive, discriminating, and valid measure of ECF. The EXIT25 has particular utility for the assessment of functional status and potential as a measure of decision-making capacity. A notable feature of the EXIT25 is that it can demonstrate executive impairments across a broad spectrum of disorders and across a broad range of severity within those conditions. Traditional neuropsychological examinations, however, may be required to assess impairments in specific executive domains. The EXIT25 is not well suited for the discrimination between dementing disorders, as executive impairments are likely to be present in any dementing illness that has evolved to a clinically disabling point in its natural history. However, when used in combination with nonexecutive measures, accurate discriminations are possible on the basis of the pattern of test scores that emerges (Royall, 2002). The EXIT25's ability to detect comparably severe executive deficits in "non-demented" medical patients without significant memory or global cognitive impairment poses a challenge to dementia case finding.

A general description of items comprising the EXIT25 is contained in Royall et al. (1992). The test itself is available from Dr. Royall. It was recently reviewed by Tate (2010).

Historical Background

Development of the EXIT25 started with a set of 50 items, generated from review of the literature and the author's clinical experience. Items were selected for acceptability to the examinee, suitability for bedside testing, and unambiguous scoring. During a process of pilot testing, item content was refined by excluding those with low clinical utility and poor intercorrelation. The final set of 25 items has a fixed order of presentation and standardized administration procedures.

Psychometric Data

Royall et al. (1992) standardized the EXIT25 on a sample of 40 elderly retirees (descriptive data below), who were recruited from 537 residents of a single comprehensive care retirement community (CCRC) in San Antonio, Texas, USA. The community had four levels of care: (1) retirement apartments without services, (2) domestic services provided, (3) intermediate care nursing units, and (4) "Alzheimer's Care Units". Levels 1 and 2 were designated by the authors as "noninstitutionalized"; Levels 3 and 4 as "institutionalized." Ten subjects were selected from each level and interviewed blind to their level of care. Validation instruments included the Mini-Mental State Examination (MMSE), TMT, Wisconsin Card Sorting Test (WCST), Serial Attention Test (SAT), and Nursing Home Behavior Problem Scale (NHBPS). Cognitive testing was performed blind to EXIT25 scores. No significant correlation coefficients were found between EXIT25 scores and age, sex, or education. Inter-rater reliability was examined in an independent sample ($n = 30$) from a range of accommodation levels who were tested independently by two physicians.

Cut-off scores were explored in the standardization sample, and were provisionally set at 15/16, below which no abnormal score on the NHBPS was found. In a subsequent publication, Royall, Rauch, Román, Cordes, and Polk (2001) cite data from a sample of 200 people, in which a receiver operating characteristic (ROC) analysis yielded an area under the curve of 0.93 (sensitivity 93%; specificity 83%) for the discrimination of institutionalized and noninstitutionalized subjects. In a study involving the Chinese translation, Chan, Chiu and Lam (2006) confirm an ROC of $c = 0.986$ for the discrimination between all-cause dementia and controls. The best discrimination was again obtained at the 15/16 cutoff. Stokholm et al. (2005) have confirmed significant associations between the EXIT25s and traditional executive measures, including the Stroop Test and verbal fluency. Chan et al. (2006) report significant correlations between the EXIT25 and "Category Number," "Failure to Maintain Set," "Total Errors," "Perseverative errors," and "Non-Perseverative Errors" from a Chinese translation of the WCST.

Discriminant validity was assessed by Royall, Cabello and Polk (1998). They tested the EXIT25's ability to discriminate among residents at different levels of care among 107 older retirees (mean age = 83.7 ± 7.2 years), including 17 community-dwelling, well, older controls and 90 CCRC residents. Sixty-one subjects resided at a noninstitutionalized level of care and 46 were institutionalized. The EXIT25 was significantly associated with the level of care, independently of medication usage, depression ratings, and problem behavior. Together, these three variables accounted for 69% of the total variance in the level of care ($R^2 = 0.69$; F [df 7.99] = 32.1, $p < .001$). In a subsequent study (Royall, Chiodo, & Polk, 2005) in a larger sample ($n = 193$) from a second retirement community, EXIT25 scores contributed significantly to an algorithm for the detection of residents in need of an "assisted living" vs. independent living levels of care.

Royall et al. (2001) have examined the association between magnetic resonance imaging (MRI) and EXIT25 scores in a convenience sample of 52 older people recruited from a dementia assessment clinic. EXIT25 scores were associated with ratings of lesion severity performed by a neuroradiologist (blinded to EXIT25 scores). EXIT25 scores were significantly associated with left frontal ($p < .002$), left medial ($p < .03$), right frontal ($p < .02$), and right medial ($p < .02$) cortical lesions by MRI, independently of age and MMSE scores. The EXIT25's associations with right hemisphere lesions did not persist after adjusting for left frontal lesions. Left posterior lesions did not significantly affect the EXIT25. Similarly, left frontal circuit pathology worsened EXIT25 scores ($p < .05$). Pathology in left anterior subcortical structures showed a trend ($p = .052$). EXIT25 scores were neither affected by right subcortical pathology nor by pathology in either hippocampus.

Kochunov et al. (2009) have explored the relationship between structural neuroimaging-based indices of cerebral integrity and EXIT25 scores across the life span. In aging adults, higher EXIT25 scores were associated with atrophic changes in cerebral white matter (WM) (sulcal and intergyral span) primarily in the superior frontal and anterior cingulate regions. Sixty-two percent of the variance in EXIT25 scores could be explained by variability in the structural indices from those two regions.

Evidence for the ecological validity of the EXIT25 is provided by the authors and independent research groups. The EXIT25 is significantly associated with problem behaviors as measured by the Frontal Behavioral Inventory (FBI) (Stokholm et al., 2005) and strongly associated with resistiveness in demented elderly ($r = 0.73$) (Stewart, Gonzalez-Perez, Zhu, & Robinson, 1999) as well as with NHBPS scores ($r = 0.79$) in its original standardization sample. Apathy is unrelated to EXIT25 scores in depressed elderly patients (Marin, Butters, Mulsant, Pollock, & Reynolds, 2003), while insight is unrelated to EXIT25 scores in schizophrenia (Hwang, Lee, & Cho, 2009). This suggests that the EXIT25 may be specifically associated with some "frontal" behaviors more than others. Similarly, the EXIT25 loads most strongly on a psychometric factor that is co-labeled by verbal fluency, digit symbol substitution, and an executive clock-drawing task, but neither WCST indices nor TMT, each of which labels orthogonal ECF factors (Royall, Chiodo, & Polk, 2003).

EXIT25 scores are stable over time. The annual rate of change in CCRC residents is estimated at EXIT25: 0.89 points (SE = 0.16) (Royall et al., 2004). There are no practice effects (Royall et al., 2005). O'Shaughnessy et al. (2005) have used the EXIT25 in a placebo-controlled clinical trial of erythropoetin for chemotherapy-related cognitive impairment. After 4 weeks of therapy, the mean change in EXIT25 scores in the placebo group was only 0.3 ± 2.4.

Clinical Uses

The EXIT25 was originally validated in geriatric samples. It has been widely used in elderly patients with dementia, and normative data is available well into the eighth decade. It has also been used in younger patients with a wide variety of conditions (reviewed in Royall et al., 2002), including head injury (Dreer, DeVivo, Novack, Krzywanski, & Marson, 2008; Larson, Leahy, Duff, & Wilde, 2008), HIV dementia, frontotemporal dementia, and mental disorders including schizophrenia (Hwang et al., 2009) and bipolar illness (BPI) (Gildengers et al., 2004; Altshuler et al., 2007). In schizophrenia, EXIT25 scores are significantly associated with the level of care, the medication adherence, and the risk of extrapyramidal movement disorders after neuroleptic exposure. In patients with BPI, the EXIT25 is associated with employment status (Altshuler et al., 2007) and with suicide attempts in depressed elderly patients (Dombrovski et al., 2008). The TEXAS, a five-item telephone version, predicts relapse risk in recently discharged BPI patients (Bauer, McBride, Shea, Gavin, & Fogel, 1994).

Dementia screening: Chan et al. (2006) report and ROC of $c = 0.986$ for the EXIT25's discrimination between all-cause dementia and controls. More difficult discriminations are those between normal controls and "Mild Cognitive Impairment" (MCI), or between MCI and dementia. However, the EXIT25 discriminates MCI patients from both normal controls and demented patients (Periera, Yassuda, Oliveira, & Forlenza, 2008) and among subjects at each dementia stage, as assessed by the Clinical Dementia Rating (CDR) scale (Chan et al., 2006). Outside of Alzheimer's disease (AD), the EXIT25 also distinguishes non-demented patients with Parkinson's disease (PD) from both normal controls and demented PD patients (Martin et al., 2008) and between "prodromal" ischemic vascular disease and vascular dementia (Ramos-Estébanez et al., 2008). The EXIT25 can distinguish different levels of care among non-demented subjects, as well as predict their use of prostheses and Instrumental Activities of Daily Living (IADL) impairment (Periera et al., 2008). In contrast, Larson et al. (2008) report a ceiling effect in mild traumatic brain injury (TBI). However, this study was performed among the subset of referrals previously selected for rehabilitation, which may exert a positive bias on EXIT25 scores.

The EXIT25 has also been used successfully in patients with HIV and frontotemporal dementia (FTD). Moreover, there is a high prevalence of unrecognized executive impairment among medical patients with normal MMSE scores (Schillerstrom, Deuter, Wyatt, Stern, & Royall, 2003;2005 Schillerstrom et al., ; Fuller et al., 2008). Such cases are demonstrably impaired in their functional status and decision-making abilities and are not merely "preclinical" MCI cases.

This is particularly true in the presence of vascular risk factors (DeCoteau et al., 2006; Thabit et al., 2009). Because their memory performance is generally normal, such cases are not currently diagnosable with "dementia," at least according to the Diagnostic and Statistical Manual (DSM). However, they are not adequately described as MCI either, because of the strong and specific association between executive function, as measured by the EXIT25 and functional status (Royall, 2006).

Royall has suggested that this issue can be reconciled by the recognition of a second dementia syndrome, dominated by executive impairment (Royall & Polk, 1998; Royall, 2000). This proposal would effectively make executive impairment the "essential" feature of dementia, while impairments in memory and other specific cognitive domains would be relegated to the status of optional

features (Royall, 2006). This approach is supported by the failure of several authors to distinguish between dementing illnesses on the basis of their EXIT25 scores. When disorders are matched to functional status, EXIT25 scores are effectively matched as well (Royall et al., 1993).

Functional status assessment: In its review of the cognitive correlates of functional status, the Research Committee of the American Neuropsychiatric Association identified 12 published multivariate regression models, comprising 813 unique subjects, in which the EXIT25 was used as a predictor of a functional outcome measure. The mean partial R^2 reported in these models was $R = 0.24$ (Royall et al., 2007). The EXIT25 is a significant predictor of IADL in both cross-sectional and longitudinal. It is strongly associated with performance-based functional assessments, e.g., the Cognitive Competency Test (CCT) (partial $R = 0.40$) (DeCoteau et al., 2006) and the Direct Assessment of Functional Status (DAFS-R) ($r = -0.872$, $p < 0.001$). The latter effect is independent of age, gender, education, and Cambridge Cognitive Test (CAMCOG) performance (Pereira et al., 2008). At its recommended cut-point is of 15/50, the EXIT25 perfectly predicts the capacity of older patients with chronic obstructive pulmonary disease (COPD) to learn to use an inhaler correctly (Allen, Jain, Ragab, & Malik, 2003). The EXIT25 is less strongly associated with Basic Activities of Daily Living (BADL).

The EXIT25 is significantly associated with level of care in elderly populations, adherence to medications and safe sexual practices in human immunodeficiency virus (HIV) infected patients. EXIT25 scores are inversely correlated with self-care activities in diabetic patients (Thabit et al., 2009) and are significantly associated with employment status in younger adults with BPI, independently of lifetime psychiatric hospitalizations and number of psychotropic medications (Altshuler et al., 2007).

Capacity assessment: The EXIT25's associations with capacity assessments have been reviewed elsewhere (Royall et al., 2007). EXIT25 scores predict financial capacity. As measured by the Capacity to Consent to Treatment Instrument (CCTI) ($r = 0.53$–0.75). EXIT25 was the strongest predictor of the "understanding treatment" standard ($R^2 = 0.56$) and the only significant predictor of a "rational reasons" standard ($R^2 = 0.45$). In a follow-up study of patients with traumatic brain injury, the baseline EXIT25 was significantly associated with recovery of capacity after six months (Dreer et al., 2008).

The EXIT25 is significantly associated with medical decision-making and the capacity to manage one's affairs. The EXIT25 correlates significantly with each MacArthur Competency Assessment Tool-Treatment (MacCAT-T) decision-making capacity domain, i.e., "Understanding," "Appreciation," and "Reasoning" (Schillerstrom, Rickenbacker, Joshi, & Royall, 2007). It is significantly correlated with the Hopkins Competency Assessment Test (HCAT), independently of MMSE scores, age, education, or number of prescribed medications. The best EXIT25 score for the discrimination of subjects who have lost their HCAT rated capacity to make an advance directive was 24/50 ($c = 0.95$). However, the EXIT25 out-performs the HCAT as a screen for the capacity to consent to inpatient psychiatric treatment. Among Adult Protective Services (APS) referrals, the EXIT25 is strongly related to autonomy, as measured by the Kohlman Evaluation of Living Skills (KELS) ($r = .705$) (Burnett, Dyer, & Naik, 2009).

Epidemiological applications: Royall et al. have published extensively on the EXIT25 among CCRC residents. In addition, the EXIT25 has been used to demonstrate a high prevalence of executive impairment among large community samples of patients with schizophrenia and bipolar disorder. However, the EXIT25 may be too long for more widespread acceptance in an epidemiological context.

On the other hand, a 15-item version of the EXIT (EXIT15) has been employed in the Health, Aging and Body Composition (Health ABC) study, a large community survey ($n = 2{,}349$ older subjects). In that study, the EXIT15 was significantly associated with longitudinal changes in gait speed, after adjusting for baseline speed (Atkinson et al., 2007). The TEXAS may be suitable for telephone screening of community samples. It correlates $r = 0.88$ with the EXIT25. At a threshold of 3/15, the TEXAS has sensitivity = 1, specificity = 0.88 vs. EXIT25 scores >10/50 (Bauer et al., 1994).

Clinical trials: Because of its brevity, test–retest reliability, sensitivity to change, and strong association with functional status, the EXIT25 has been used in several clinical trials (Auchus et al., 2007; Dichgans et al., 2008; O'Shaughnessy et al., 2008; Boxer et al., 2009). Cholinesterase inhibitors appear to have modest effects on EXIT25 scores in subcortical infarcts and leukoencephalopathy (CADASIL) (Dichgans et al., 2008) but not in vascular dementia (Auchus et al., 2007; Román, Salloway, Black, Royall, & DeCarli, in press). In contrast, sertraline may improve EXIT25 scores in vascular cases (Royall et al., 2009).

Cross References

► Executive Functioning

References and Readings

Allen, S. C., Jain, M., Ragab, S., & Malik, N. (2003). Acquisition and short-term retention of inhaler techniques require intact executive function in elderly subjects. *Age Ageing, 32*, 299–302.

Altshuler, L., Tekell, J., Biswas, K., Kilbourne, A. M., Evans, D., Tang, D., et al. (2007). Executive function and employment status among veterans with bipolar disorder. *Psychiatric Services, 58*, 1441–1447.

Atkinson, H. H., Rosano, C., Simonsick, E. M., Williamson, J. D., Davis, C., Ambrosius, W. T., et al. (2007). Cognitive function, gait speed decline, and comorbidities: The health, aging and body composition study. *Journals of Gerontology. Series A, Biological Sciences and Medical Sciences, 62*, 844–8450.

Auchus, A. P., Brashear, H. R., Salloway, S., Korczyn, A. D., De Deyn, P. P., & Gassmann-Mayer, C. (2007). GAL-INT-26 study group Galantamine treatment of vascular dementia: A randomized trial. *Neurology, 69*, 448–458.

Bauer, M., McBride, L., Shea, N., Gavin, C., & Fogel, B. (1994). Screening for executive cognitive dysfunction in bipolar disorder. *Biological Psychiatry, 35*, 636.

Boxer, A. L., Lipton, A. M., Womack, K., Merrilees, J., Neuhaus, J., Pavlic, D., et al. (2009). An open-label study of memantine treatment in 3 subtypes of frontotemporal lobar degeneration. *Alzheimer Disease and Associated Disorders, 23*, 211–217.

Burnett, J., Dyer, C. B., & Naik, A. D. (2009). Convergent validation of the Kohlman evaluation of living skills as a screening tool of older adults' ability to live safely and independently in the community. *Archives of Physical Medicine and Rehabilitation, 90*, 1948–1952.

Chan, S. M., Chiu, F. K., & Lam, C. W. (2006). Correlational study of the Chinese version of the executive interview (C-EXIT25) to other cognitive measures in a psychogeriatric population in Hong Kong Chinese. *International Journal of Geriatric Psychiatry, 21*, 535–541.

DeCoteau, W. E., Selk, B., Stirling, G., Fentiman, P., Savage, C., & Landeryou, J. (2006). Dementia due to hypertensive microvascular disease of the brain often goes unrecognized because the patient is not tested, or the wrong tests are done. *The Journal of Clinical Hypertension, 8*(Suppl. A No. 5), 36.

Dichgans, M., Markus, H. S., Salloway, S., Verkkoniemi, A., Moline, M., Wang, Q., et al. (2008). Donepezil in patients with subcortical vascular cognitive impairment: A randomised double-blind trial in CADASIL. *Lancet Neurology, 7*, 310–318.

Dombrovski, A. Y., Butters, M. A., Reynolds, C. F. 3rd, Houck, P. R., Clark, L., Mazumdar, S., et al. (2008). Cognitive performance in suicidal depressed elderly: Preliminary report. *American Journal of Geriatric Psychiatry, 16*, 109–115.

Dreer, L. E., DeVivo, M. J., Novack, T. A., Krzywanski, S., & Marson, D. C. (2008). Cognitive predictors of medical decision-making capacity in traumatic brain injury. *Rehabilitation Psychology, 53*, 486–497.

Fuller, C. D., Schillerstrom, J. E., Jones, W. E. 3rd, Boersma, M., Royall, D. R., & Fuss, M. (2008). Prospective evaluation of pretreatment executive cognitive impairment and depression in patients referred for radiotherapy. *Journal of Radiation Oncology, Biology, Physics, 72*, 529–533.

Gildengers, A. G., Butters, M. A., Seligman, K., McShea, M., Miller, M. D., Mulsant, B. H., et al. (2004). Cognitive functioning in late-life bipolar disorder. *American Journal of Psychiatry, 161*, 736–738.

Hwang, S. S., Lee, J. Y., Cho, S. J., et al. (2009). The model of the relationships among the predictors of quality of life in chronic stage of schizophrenia. *Progress in Neuropsychopharmacology & Biological Psychiatry, 33*, 1113–1118.

Kochunov, P., Robin, D. A., Royall, D. R., Coyle, T., Lancaster, J., Kochunov, V., et al. (2009). Can structural MRI indices of cerebral integrity track cognitive trends in executive control function during normal maturation and adulthood? *Human Brain Mapping, 30*, 2581–2594.

Larson, E. B., Leahy, B., Duff, K. M., & Wilde, M. C. (2008). Assessing executive functions in traumatic brain injury: An exploratory study of the executive interview. *Perceptual and Motor Skills, 106*, 725–736.

Marin, R. S., Butters, M. A., Mulsant, B. H., Pollock, B. G., & Reynolds, C. F. 3rd. (2003). Apathy and executive function in depressed elderly. *Journal of Geriatric Psychiatry and Neurology, 16*, 112–116.

Martin, R. C., Okonkwo, O. C., Hill, J., et al. (2008). Medical decision-making capacity in cognitively impaired Parkinson's disease patients without dementia. *Movement Disorders, 23*, 1867–1874.

O'Shaughnessy, J. A., Vukelja, S. J., Holmes, F. A., Savin, M., Jones, M., Royall, D., et al. (2005). Feasibility of quantifying the effects of epoetin alfa therapy on cognitive function in women with breast cancer undergoing adjuvant or neoadjuvant chemotherapy. *Clinical Breast Cancer, 5*, 439–446.

Osorio, R., García de Lózar, B., Ramos, I., & Agüera, L. (2009). Executive function in patients with late onset depression. *Actas Españolas De Psiquiatría, 37*, 196–199.

Pereira, F. S., Yassuda, M. S., Oliveira, A. M., & Forlenza, O. V. (2008). Executive dysfunction correlates with impaired functional status in older adults with varying degrees of cognitive impairment. *International Psychogeriatrics,* 1–12, *20*, 1104–1115.1–12,

Ramos-Estébanez, C., Moral-Arce, I., Muñoz-Arrondo, R., González-Mandly, A., Matorras-Galán, P., González-Macias, J., et al. (2008). Vascular cognitive impairment: Prodromal stages of ischemic vascular dementia. *Dementia and Geriatric Cognitive Disorders, 25*, 451–460.

Román, G. C., Salloway, S., Black, S. E., Royall, D. R., & DeCarli, C. (2010). A randomized, placebo-controlled clinical trial of donepezil in vascular dementia: Differential effects by hippocampal size. *Stroke*, (in press).

Royall, D. R. (2000). Executive cognitive impairment: A novel perspective on dementia. *Neuroepidemiology, 19*, 293–299.

Royall, D. R. (2002). Bedside assessment of vascular dementia. In S. Gauthier & T. Erkinjuntti (Eds.), *Vascular cognitive impairment* (Chap. 18., pp. 307–322) London: Martin Dunitz Ltd.

Royall, D. R. (2006). Mild cognitive impairment and functional status. *Journal of the American Geriatrics Society, 54*, 163–165.

Royall, D. R., Cabello, M., & Polk, M. J. (1998). Executive dyscontrol: An important factor affecting the level of care received by older retirees. *Journal of the American Geriatrics Society, 46*, 1519–1524.

Royall, D. R., Chiodo, L. K., & Polk, M. (2003). Executive dyscontrol in normal aging: Normative data, factor structure, and clinical correlates. In J. C. M. Brust & S. Fahn (Eds.), *Current neurology and neuroscience reports* (Vol. 3(6), pp. 487–493). Philadelphia: Current Science Inc.

Royall, D. R., Chiodo, L. K. & Polk, M. J. (2005). An empiric approach to level of care determinations: the importance of executive measures. *Journals of Gerontology, 60A*, 1059–1064.

Royall, D. R., Lauterbach, E. C., Cummings, J. L., Reeve, A., Rummans, T. A., Kaufer, D. I., et al. (2002). Executive control function: A review of its promise and challenges to clinical research. *Journal of Neuropsychiatry and Clinical Neurosciences, 14*, 377–405.

Royall, D. R., Lauterbach, E. C., Kaufer, D. I., Malloy, P., Coburn, K. L., & Black, K. J. (2007). The cognitive correlates of functional status: A

review from the committee on research of the American Neuropsychiatric Association. *Journal of Neuropsychiatry and Clinical Neurosciences, 19*, 249–265.
Royall, D. R., Mahurin, R. K., & Gray, K. F. (1992). Beside assessment of the executive cognitive impairment: The executive interview. *Journal of the American Geriatrics Society, 40*, 1221–1226.
Royall, D. R., Mahurin, R. K., True, J., Anderson, B., Brock, P. I., Freeburger, L., et al. (1993). Executive impairment among the functionally dependent: Comparisons between schizophrenic and elderly subjects. *American Journal of Psychiatry, 150*, 1813–1819.
Royall, D. R., & Polk, M. J. (1998). Dementias that present with and without posterior cortical features: An important clinical distinction. *Journal of the American Geriatrics Society, 46*, 98–105.
Royall, D. R., Rauch, R., Román, G. C., Cordes, J. A., & Polk, M. J. (2001). Frontal MRI findings associated with impairment on the executive interview (EXIT25). *Experimental Aging Research, 27*, 293–308.
Royall, D. R., Román, G. C., Cordes, J. A., Velez, A., Edwards, A., & Schillerstrom, J. S. (2009). Sertraline improves executive function in patients with vascular cognitive impairment. *Journal of Neuropsychiatry and Clinical Neurosciences, 21*, 445–454.
Schillerstrom, J. E., Deuter, M. S., Wyatt, R., Stern, S. L., & Royall, D. R. (2003). Prevalence of executive impairment in patients seen by a psychiatry consultation service. *Psychosomatics, 44*, 290–297.
Schillerstrom, J. E., Horton, M. S., Schillerstrom, T. L., Joshi. K. G., Earthman, B. S., Velez, A. M., et al. (2005). Prevalence, course and risk factors for executive impairment in patients hospitalised on general medicine service. *Psychosomatics, 46*, 411–417.
Schillerstrom, J. E., Rickenbacker, D., Joshi, K. G., & Royall. D. R. (2007). Executive function and capacity to consent to a noninvasive research protocol. *American Journal of Geriatric Psychiatry, 15*, 159–162.
Stewart, J. T., Gonzalez-Perez, E., Zhu. Y., & Robinson, B. E. (1999). Cognitive predictors of resistiveness in dementia patients. *American Journal of Geriatric Psychiatry, 7*, 259–263.
Stokholm, J., Vogel, A., Gade, A., & Waldemar, G. (2005). The executive interview as a screening test for executive dysfunction in patients with mild dementia. *American Journal of Geriatric Psychiatry, 53*, 1577–1581.
Tate, R. L. (2010). *A compendium of tests, scales, and questionnaires: The practitioner's guide to measuring outcomes after acquired brain impairment*. Hove, UK: Psychology Press.
Thabit, H., Kennelly, S. M., Bhagarva, A., Ogunlewe, M., McCormack, P. M., McDermott, J. H., et al. (2009). Utilization of frontal assessment battery and executive interview 25 in assessing for dysexecutive syndrome and its association with diabetes self-care in elderly patients with type 2 diabetes mellitus. *Diabetes Research and Clinical Practice, 86*, 208–212.

Executive Processes

► Executive Functioning

Exencephaly

► Anencephaly

Exhaustion

► Fatigue

EXIT

► Executive Interview

EXIT15

► Executive Interview

EXIT-25

► Executive Interview

Expanded Disability Status Scale

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Synonyms

EDSS; Kurtzke expanded disability status scale

Description

The Expanded Disability Status Scale (EDSS) is based upon the original Kurtzke Disability Status Scale (DSS) and Functional Systems (FS) (Kurtzke, 1961). There are eight FSs: pyramidal, cerebellar, brainstem, sensory, visual, bowel and bladder, cerebral, and other. Following neurological examination, each FS is rated on a scale of 0–5 (cerebellar and brainstem), 0–1 (other), and 0–6 (all others). These ratings assist in assigning an EDSS score

which ranges from 0 to 10 in 0.5 increments, where 0 indicates a normal neurological examination (but cerebral grade 1 allowed) and 10 corresponds to death due to multiple sclerosis (MS) (see Kurtzke, 1983 for a full description of the rating scale). In general, EDSS scores ranging from 1.0 to 4.5 quantify the disability/impairment experienced by individuals with MS who are ambulatory, while EDSS scores from 5.0 to 9.5 correspond to disability/impairment of individuals who have some impairment of ambulation. For example, an EDSS of 3.5 describes an individual who is fully ambulatory but has moderate disability in one FS and more than minimal disability in several other FSs, while an EDSS of 8.5 corresponds to an individual who is restricted to bed for most of the day but has some use of the arms and is able to perform some self-care functions.

Historical Background

The development of the DSS and FS, upon which the EDSS is based, occurred in the early 1960s, when the neurological examinations of 762 men who had been diagnosed with MS while in the army were systematically reviewed (Kurtske, 1961). The examinations spanned approximately the first 20 years of the course of the disease. A team of neurologists agreed as to the primary diagnosis of MS for 527 of the sample, while 146 were classified as "definitely not" MS (Nagler et al., 1966). These two groups were examined over time for the development of MS-specific signs and symptoms which served to validate the DSS and FS categories (Beebe, Kurtzke, Kurland, Auth, & Nagler, 1967).

The EDSS is one of the most widely used impairment measures for clinical trials in multiple sclerosis (MS). The EDSS has been criticized in that it is not a linear, ordinal assessment tool nor is it an equal-interval or ratio scale; thus parametric statistics and stepwise comparisons are not appropriate. In addition, the average time that an individual spends at each level of the scale is highly variable and tends to be longer at the two extremes of the scale (Weinshecker et al., 1989).

Some recent trials have used a measure of treatment failure (TF) and appropriate nonparametric statistics, examining the proportion of patients presented TF and/or time to TF. Since there is nonequivalence of intervals, it has been suggested that the TF should be taken as a worsening of 1.0 point for individuals with a baseline EDSS of 5.5 or less, while at a baseline EDSS of 5.5 or greater, TF should be considered a worsening of 0.5 point (Amato and Ponziani, 1999).

Psychometric Data

Reliability: Inter- and intra-rater reliabilities are dependent upon the definition of "agreement" that is used. When agreement between raters or after an interval is defined as a difference no greater than 1.0 point, inter-rater agreement was 96% (Sharrack, Hughes, Soudain, & Dunn, 1999); however it fell to 89% if the difference was defined as no greater than 0.5 point.

Validity: The EDSS has high correlations with the FIM, Scripps Neurological Rating Scale, and patient's self-assessment of disability (Sharrack et al., 1999). It has also shown high correlations with the Barthel Index but not the physical and mental component scores of the SF-36, the London Handicap Scale, and the General Health Questionnaire (Hobart, Freeman, & Thompson, 2000); this indicates that it can discriminate overall disability from related health constructs.

Clinical Uses

The EDSS was not sensitive to clinical change over a 9-month follow-up period in 25 individuals with MS who reported changes in symptom and function (Sharrack et al., 1999). It was also unresponsive in a cohort of 64 individuals with moderate to severe disability (Hobart, Lamping, Freeman, & Thompson, 1996) and a group of 137 individuals with clinically definite MS (Hobart et al., 2000).

In addition, the EDSS had limited variability in scores in a sample of 137 individuals when compared to the FIM and Barthel Index (Hobart et al., 2000).

It is the contention of multiple authors that the EDSS is based on sound clinical intuition, but does not measure up to psychometric evaluation (Hobart et al., 2000; Sharrack et al., 1999; Thompson and Hobart, 1996).

Cross References

- ▶ Barthel Index
- ▶ Functional Assessment Measure
- ▶ SF-36/SF-12

References and Readings

Amato, M. P., & Ponziani, G. (1999). Quantification of impairment in MS: discussion of the scales in use. *Multiple Sclerosis, 5*(4), 216–219.

Beebe, G. W., Kurtzke, J. F., Kurland, L. T., Auth, T. L., & Nagler, B. (1967). Studies on the natural history of multiple sclerosis. 3. Epidemiologic analysis of the Army experience in World War II. *Neurology, 17*, 1–7.

Hobart, J. C., Lamping, D. L., Freeman, J. A., & Thompson, A. J. (1996). Reliability, validity and responsiveness of the Kurtzke expanded disability status scale (EDSS) in multiple sclerosis (MS) patients. *European Journal of Neurology, 3*(Suppl 4), 13.

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

Kurtske, J. F. (1961). On the evaluation of disability in multiple sclerosis. *Neurology, 11*, 686–694.

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

Nagler, B., Beebe, G. W., Kurtzke, J. F., Kurland, L. T., Auth, T. L., & Nefzger, M. D. (1966). Studies on the natural history of multiple sclerosis. 1. Design and diagnosis. *Acta Neurol Scand 42*(Suppl 19), 141–156.

Sharrack, B., Hughes, R. A. C., Soudain, S., & Dunn, G. (1999). The psychometric properties of clinical rating scales used in multiple sclerosis. *Brain, 122*, 141–159.

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

Expert v. Treater Role

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Definition

Often, psychologists may begin a case as a treater in a clinical setting and then they are asked to serve in a forensic role. A particular conflict arises when this occurs and the psychologist must be careful not to occupy dual roles. Indeed, there is a clear distinction between the role of a treater and an expert. In clinical settings, a psychologist embodies the treater role and is an *advocate for the patient*. Conversely, the expert role requires full objectivity at all times and the expert is an *advocate of the facts* which could be potentially damaging to the plaintiff's (e.g., their patient) case. The APA Ethical Principles (2002) strongly advise against performing "multiple and potentially conflicting roles in forensic matters." In order to protect against serving multiple roles, forensic psychologists should demand to only fulfill one of three possible roles: treater, trial consultant, or testifying expert. Greenberg and Shuman (1997) provide an extensive overview of the problems associated with treaters who are asked to also serve as an expert witness. Most notably, if a psychologist accepts a role as an expert witness, then his/her patient is no longer the client; instead, it is the attorney who becomes the client. Shuman, Greenberg, Heilbrun, and Foote (1998) argue that treaters should be prohibited from testifying as expert witnesses and that attorneys for both the plaintiff and the defense should be required to obtain their own independent examiners.

Cross References

► Expert Witness
► Independent Neuropsychological Examination

References and Readings

American Psychological Association (2002). Ethical principles of psychologists and code of conduct. *American Psychologist, 57*, 1060–1073.

Committee on Ethical Guidelines for Forensic Psychologists (1991). Specialty guidelines for forensic psychologists. *Law and Human Behavior, 15*, 655–665.

Greenberg, S. A., & Shuman, D. W. (1997). Irreconcilable conflict between therapeutic and forensic roles. *Professional Psychology: Research and Practice, 28*, 50–57.

Greiffenstein, M. F., & Cohen, L. (2005). Neuropsychology and the law: Principles of productive attorney Neuropsychologist relations. In G. Larrabee (Ed.), *Forensic neuropsychology: A scientific approach.* New York: Oxford University Press.

Shuman, D. W., Greenberg, S., Heilbrun, K., & Foote, W. E. (1998). An immodest proposal: Should treating mental health professionals be barred from testifying about their patients? *Behavioral Sciences and the Law, 16*, 509–523.

Expert Witness

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Definition

First and foremost, one must distinguish between a "fact witness" and an "expert witness." A fact witness only provides testimony regarding direct knowledge of the

facts related to the issue at hand (e.g., an "eye witness" account of things). In contrast, an expert witness can provide facts, but can also offer opinions and report hearsay. Since the decision rendered in *Jenkins v. United States* (1962), psychologists have been allowed to offer expert witness testimony in civil and criminal proceedings. Still, the admissibility of expert witness testimony is a critical issue. This determination is guided by a series of rules from the Federal Rules of Evidence Article (FRE) VII ("Opinions & Expert Testimony"). Specifically, FRE rule 702 states, "If scientific, technical or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise." In the case of *Frye v. United States* (1923), the *Frye* standard was established, which stated that: only scientific methods and concepts with "general acceptance" within a particular field are admissible. In the more recent case of *Daubert v. Merrell* Dow (1993), it was determined that scientific testimony has to abide by two criteria: the testimony must be (a) scientifically valid and (b) relevant to the case at hand. Once an expert witness has been identified, he/she is subject to discovery as well as direct and cross-examinations. During the first phase of the direct examination, the opposing attorney challenges the expert witness' credentials and requests that the court *voir dire* him/her. This involves asking additional questions to assess the expert's competency. After this phase, a trial judge decides whether the testimony of the expert witness is to be permitted. If allowed, direct examination by the retaining attorney reconvenes and cross-examination by the opposing attorney follows.

Cross References

- ▶ Cross-Examination
- ▶ *Daubert v. Merrell Dow*
- ▶ Direct Examination
- ▶ Discovery
- ▶ *Expert v. Treater Role*
- ▶ *Federal Rules of Evidence*
- ▶ Testifying Expert Versus Fact Witness

References and Readings

Daubert v. Merrell Dow, 509 U.S. 579 (1993).
Federal Rules of Evidence (1975).
Frye v. U.S. (D.C. Cir. 1923). 293 F. 1013.
Goldstein, A. M. (2003). Overview of forensic psychology. In A. Goldstein (Ed.), *Handbook of Psychology (Vol 11). Forensic Psychology.* New Jersey: John Wiley & Sons.
Greiffenstein, M. F., & Cohen, L. (2005). Neuropsychology and the law: Principles of productive attorney neuropsychologist relations. In G. Larrabee (Ed.), *Forensic neuropsychology: A scientific approach.* New York: Oxford University Press.
Jenkins v. U.S., 307 F. 2d 637 (1962).

Explicit Memory

- ▶ Declarative Memory
- ▶ Episodic Memory

Expressive Aphasia

- ▶ Broca's Aphasia
- ▶ Nonfluent Aphasia

Expressive One-Word Picture Vocabulary Test

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Synonyms

EOWPVT

Description

The Expressive One-Word Picture Vocabulary Test (EOWPVT) is an individually administered instrument designed to evaluate a person's knowledge of English-speaking vocabulary. The test is norm-referenced and designed to be used with individuals between the ages of 2 years, 0 months to 18 years, 11 months. During administration, the examiner shows the examinee a series of pictures depicting actions, objects, and concepts. The examinee is asked to name the illustration presented

and then the answer is recorded in the EOWPVT protocol. The test kit consists of 170 test plates in a spiral-bound easel. According to the manual, administration and scoring time takes between 15 to 20 min, depending on the individual. Raw scores are obtained which are then translated to standard scores, percentiles, and age equivalents.

Historical Background

Measures of vocabulary have long held an important place in psychoeducational assessment. Research consistently demonstrates the effect that vocabulary has on academic achievement (Baker, Simmons, & Kameenui, 1998). The historical significance of vocabulary assessment has been noted as early as 1916 by Terman, who demonstrated that vocabulary is an excellent predictor of cognitive ability. Furthermore, identifying young children with language difficulties and monitoring speech rehabilitation are just some of the worthy roles that vocabulary tests, like the EOWPVT, have taken in clinical settings.

The current edition of the EOWPVT (2000) is an update from the original publication in 1979 and its subsequent revision in 1990. In the newest edition, norms are improved, outdated items removed, clearer illustrations are in full color, and slight administration changes were made to reduce examinee confusion. It is often used in conjunction with its "sister" test, the Receptive One-Word Picture Vocabulary Test (ROWPVT), a measure of receptive vocabulary. Use of both measures results in a comprehensive assessment of receptive and expressive vocabulary.

Psychometric Data

Internal consistency, test–retest reliability, and interrater reliability have all been evaluated for the EOWPVT. The manual reports acceptable coefficient alphas ranging from 0.93 to 0.98 with a median of 0.96. Split-half coefficients (corrected for the full length) are reported to have a median of 0.98. Two hundred and twenty-six examinees were retested to investigate test–retest reliability. A coefficient of 0.90 was found for the entire sample, providing evidence that the tool is suitably stable over time. Interrater reliability was investigated in three different ways and all of them demonstrated strong consistency among multiple examiners. The authors established content validity by using only those words that could obviously be illustrated. Early items for the test were collected through parent questionnaires that asked parents to indicate the first words spoken by their children while some obscure words were used to tap into superior vocabulary ability at the higher levels. The rest of the words were selected with reference to their frequency in written material and the grade level at which the words appear in curriculum materials. Criterion-related validity for the EOWPVT has been amply established between it and a host of other vocabulary tests. Finally, the authors have verified construct validity between their instrument and a variety of sources including cognitive ability, academic ability, previous editions of the test, and expressive and receptive vocabulary. Correlations range from .64 to .89 with these measures.

The measure has been newly standardized in 1999 when it was administered to 3,661 people. From this pool, 2,327 were selected, at random, to meet demographic criteria for establishing norms. Overall, the sample closely mirrored the U.S. population in terms of region of residence, race/ethnicity, gender, disability status, residence (urban vs. rural), and parent educational attainment.

Clinical Uses

Although the EOWPVT can be used for research purposes, its primary use is clinical in nature. Results from EOWPVT testing can help identify language impairment in preschool children, monitor growth over a period of time and/or treatment, or as a way to evaluate a person learning English as a second language. Other uses are for diagnosing aphasia, diagnosing reading difficulty, or as a secondary measure of cognitive ability. Versatile in its clinical use, the test is used in educational, hospital, clinical, early childhood, and rehabilitation settings.

Some limitations must, of course, be noted. For one, the tool only assesses a fraction of one's English vocabulary. As with all tests, results from the EOWPVT should not be used in isolation. A single test cannot confirm or deny the presence of a clinical diagnosis. Rather, it must be used in conjunction with historical information, other test results, and observations that coalesce to a diagnostic impression. Examiners should always be aware of individual factors like hearing, visual, or attentional problems that may lead to an invalid administration. Finally, the EOWPVT can only be used with native English speakers or those learning the English language.

Cross References

- ▶ Language
- ▶ Speech
- ▶ Speech/Communication Disabilities

- ▶ Speech-Language Pathology
- ▶ Speech-Language Therapy
- ▶ Vocabulary

References and Readings

Baker, S., Simmons, D., & Kameenui, E. (1998). Vocabulary acquisition: Research bases. In D. C. Simmones & E. J. Kameenui (Eds.), *What reading tells us about children with diverse learning needs.* Mahwah, NJ: Lawrence Erlbaum Associates.

Brownell, R. (2000). *Expressive one-word picture vocabulary test manual.* Novato, CA: Academic Therapy Publications.

Extended Glasgow Outcome Scale

- ▶ Glasgow Outcome Scale – Extended

Extensor Posturing

- ▶ Decerebrate Posturing

External Aids

- ▶ Cognitive Correctors

External Memory Aids

- ▶ Prosthetic Memory Aids

Extinction

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Synonyms

Sensory extinction; Extinction to double simultaneous stimulation (DSS)

Short Description or Definition

Extinction refers to a failure to detect one of two simultaneously presented stimuli, even though when presented individually each stimulus can be detected.

Categorization

Extinction during DSS can occur in the somatasensory, auditory, and visual modalities.

Epidemiology

Extinction most often occurs with damage to the right hemisphere usually seen after stroke or neoplasm. However, under rare circumstances, extinction has been shown to occur in normal individuals and in patients with left hemisphere lesions. Extinction is usually considered as a manifestation of the neglect syndrome but often persists after other symptoms of neglect resolve. Neglect in humans can occur with lesions in the inferior parietal lobe, dorsal frontal lobe, cingulated gyrus, basal ganglia, and thalamus, but is most frequently observed after temporoparietal lesions.

Natural History, Prognostic Factors, Outcomes

In a study of behavioral recovery from abnormalities after right hemisphere stroke (Hier et al., 1993), 26 of 41 patients initially presented with extinction. Whereas all patients recovered from neglect, 20% continued to show extinction after 1 year. Patients with hemorrhage recovered more quickly than those with infarcts. Recovery is less complete in patients with prestroke cortical atrophy, suggesting that the integrity of the left hemisphere is essential.

Neuropsychology of Extinction

From a neuropsychological perspective, extinction provides a glance into how the brain mediates spatial attention. Walter Poppelreuter and Gordon Holmes provided some of the earliest descriptions of visual extinction early in the twentieth century. Because visual acuity could be completely normal in the affected hemi-field when one stimulus was presented, Poppelreuter (1990) called the phenomenon a "hemianoptic weakness of attention." Their recognition that this was an impairment of attention provided much of the groundwork for later theories about how the brain

mediates spatial attention (see Halligan & Marshall, 1993). Additional evidence that extinction is an attentional disorder comes from studies showing that extinction can be modified by experimental manipulations. When a left-sided stimulus is presented less than a second before the right-sided stimulus, extinction decreases dramatically. The probability that extinction will occur in the right hemisphere damaged patients can also be modified by expectation (Kaplan, Verfaellie, DeWitt, & Caplan, 1990). By manipulating the unilateral stimuli prior to DSS, these investigators showed that they could increase or reduce the probability of extinction. To what extent learning to anticipate left-sided stimulation is part of the recovery process is unclear, but it may be of compensatory strategy.

Theories of neglect are based on the premise that there is asymmetry between the cerebral hemispheres in mediating attention in space. The normal right hemisphere directs attention to both sides of space, whereas the left hemisphere mediates attention to contralateral space. Consequently, after right hemisphere damage, the patient will show a strong attentional bias to the left side of space, and neglect, including extinction, for the right side of space. Moreover, because the normal right hemisphere can direct attention to both sides of space, neglect after left hemisphere damage is relatively rare.

Evaluation

Testing for extinction to DSS is typically done at the bedside or office. To elicit visual extinction, the patient faces the examiner, while the examiner presents a finger to the left, right, or both visual fields. After each trial, the patient indicates whether he or she saw a finger on the left, right, or both sides. During testing for auditory extinction, the examiner stands behind the patient, and rubs his or her fingers together next to one of both ears using a method similar to that described above. For somatasensory extinction testing, the patient is blindfolded or asked to close his/her eyes and the examiner touches either the back of the patient's hand or face. A standardized procedure for testing sensory extinction is part of the Rivermead Assessment of Somatasensory Performance battery (Strauss, Sherman, & Preen, 2006).

Treatment

There is no treatment for extinction separate from treatments for neglect, as extinction is considered part of the neglect syndrome. Most treatments involve manipulations designed to make the patient scan the neglected hemi-field. Unfortunately, most techniques have proved to be short-lived and do not translate to activities of daily living. That being the case, patients should be instructed to avoid activities, i.e., driving, that could cause injuries to themselves or others.

Cross References

- ▶ Hemiinattention
- ▶ Neglect Syndrome

References and Readings

Halligan, P. W., & Marshall, J. C. (1993). The history and clinical presentation of neglect. In I. H. Robertson & J. C. Marshall (Eds.), *Unilateral neglect: Clinical and experimental studies* (pp. 3–19). Hillsdale, NJ: Lawrence Erlbaum.

Hier, D, B., Mondlock, J., & Caplan. L. R. (1983). Recovery of behavioral abnormalities after right hemisphere stroke. Neurology, 33, 345–350.

Kaplan, R. F., Verfaellie, M., DeWitt, L. D., & Caplan, L. R. (1990). Effects of changes in stimulus contingency on visual extinction. *Neurology, 40*, 1299–1301.

Poppelreuter, W. (1990). *Disturbances of lower and higher visual capacities by occipital damage.* (J. Zihl, Trans.). Oxford: Clarendon Press.

Strauss, E., Sherman, E., & Preen, O. (2006). *A compendium of neuropsychological tests* (3rd ed.). New York: Oxford.

Extinction to Double Simultaneous Stimulation (DSS)

- ▶ Extinction

Extraneous Variable

- ▶ Confounding Variables

Extrapyramidal Adverse Reactions

- ▶ Extrapyramidal Symptoms

Extrapyramidal Disease

► Extrapyramidal Symptoms

Extrapyramidal Side Effects

► Extrapyramidal Symptoms

Extrapyramidal Symptoms

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Synonyms

EPS; Extrapyramidal adverse reactions; Extrapyramidal disease; Extrapyramidal side effects; Parkinsonian movements

Indications

"Extrapyramidal" refers to the nerve fibers outside the pyramidal tracts of the central nervous system. The term extrapyramidal symptoms refers to the neurological adverse effects from antipsychotic medications that mimic the characteristics of extrapyramidal disease. Pharmacological agents that decrease dopamine neurotransmission other than neuroleptics, can produce extrapyramidal symptoms as well, though neuroleptics, particularly those that block D_2 receptors, are most often responsible for causing extrapyramidal adverse reactions. Standard practice when administering conventional antipsychotic medication is augmentation with anti-cholinergic agents such as benztropine (Cogentin) that block striatal cholinergic receptors can help regulate cholinergic and dopaminergic neurotransmission reducing the severity of extrapyramidal side effects (Extrapyramidal symptoms, 2009a, b; Kaufman, 2007a; Myers, 2006; Venes, Thomas, Egan, & Houska, 2001).

Mechanisms of Action

The extrapyramidal system comprises interconnections among brain stem nuclei and basal ganglia, relaying signals between the efferent fibers of the thalamus, cerebral cortex, the brainstem and cerebellum, and the five nuclei of the basal nuclei, including the corpus striatum, red nucleus, subthalamic nucleus, substantia nigra, and the reticular formation. The extrapyramidal pathways are the undecussated or uncrossed functional motor nerve nuclei from the brain to the spinal cord's anterior horns. Essential to coordinating locomotion and supporting static and postural reflexes through its efferent fibers, the extrapyramidal tracts are the relay mechanism responsible for sequential and simultaneous movements. The efferent fibers of the extrapyramidal system do not act directly on the lower motor neurons of the spinal cord, but are confined to the brain and modulate the pyramidal or corticospinal tracts (Afifi and Bergman, 1998; Stahl, 2000).

Specific Compounds and Properties

Extrapyramidal diseases encompass the unrelated group of conditions that compromise neuronal functioning within the extrapyramidal tracts such as tardive dyskinesia, chorea, athetosis, extrapyramidal cerebral palsy, Shy–Drager syndrome, Parkinson's or Alzheimer's diseases, and other degenerative neurological disorders. Physical extrapyramidal symptoms include abnormal posture, involuntary movement such as tremor and shuffling gate, muscle dystonia including muscle rigidity and contractility. Other non-movement symptoms of extrapyramidal disease can include anxiety, akathisia, slurred speech, and bradykinesia (Extrapyramidal symptoms, 2009a; Kaufman, 2007b; Myers, 2006; Venes et al., 2001).

Clinical Use

Conventional neuroleptic drugs are used to treat psychotic disorders such as schizophrenia. Clinical observation of early treatments for schizophrenia provided invaluable data about the mechanisms underlying the neurobiological basis of psychosis and ultimately of the antipsychotic medications that would be developed to remediate the positive symptoms of formal thought disorders such as hallucinations. In fact, the term "neuroleptic" derives from "neurolepsis," meaning slow or absent motor movement. The psychomotor retardation

and diminished affect characteristic of the effects of first-generation antipsychotic drugs reflect dopamine antagonism, the mechanism by which these agents decrease psychotic symptoms. It is believed that blocking the D_2 receptors specifically, has a significant effect on the reduction of positive symptoms. Unfortunately, selective D_2 antagonism has not yet been achieved, and this type of dopaminergic antagonism, particularly in the post-synaptic mesolimbic area is also largely responsible for neurolepsis or extrapyramidal symptoms, the slow, dysrhythmic, uncontrolled and involuntary behavior seen among individuals on high doses of conventional/typical, and to a lesser extent atypical or second-generation antipsychotic medications. Blockade of the D_2 nigrostriatal region produces marked vulnerability to tardive dyskinesia, perhaps the most severe of the extrapyramidal symptoms due to its propensity for permanence. Tardive dyskinesia afflicts approximately 20% annually of those taking conventional antipsychotic medications within 4 years (Kaufman, 2007b; Stahl, 2000).

Medications within this class with considerable D_2 antagonism and risk of extrapyramidal symptoms include acetophenazine (Tindal), chlorpromazine (Thorazine), chlorprothixene (Taractan), clozpine (Clozaril), fluphenazine (Prolixin), haloperidol (Haldol), loxapine (Loxitane), mesoridazine (Serentil), molindone (Moban), perphenazine (Trilafon), pimozide (Orap), prochlorperazine (Compazine), thioridazine (Mellaril), thiothixene (Navane), trifluoperazine (Stelazine), and triflupromazine (Vesprin) (Afifi and Bergman, 1998; Kaufman, 2007b; Stahl, 2000).

Cross References

- ▶ Alzheimer's Disease
- ▶ Antipsychotic Medications
- ▶ Chorea
- ▶ Dopamine Antagonists
- ▶ Extrapyramidal Cerebral Palsy
- ▶ Extrapyramidal Syndrome
- ▶ Extrapyramidal System
- ▶ Parkinson's Disease
- ▶ Psychotropic Medications
- ▶ Pyramidal Tract
- ▶ Shy–Drager Syndrome

References and Readings

Afifi, A. K., & Bergman, R. A. (1998). Basal ganglia. In A. K. Afifi & R. A. Bergman (Eds.), *Functional neuroanatomy: Text & atlas* (pp. 276–295). New York: McGraw-Hill.

Extrapyramidal symptoms. (2009a). In *The free dictionary by Farlex*, Retrieved January 13, 2009, from http://medical-dictionary.thefreedictionary.com

Extrapyramidal symptoms. (2009b). In *MedicineNet*. Retrieved January 13, 2009, from http://www.medterms.com

Kaufman, D. M. (2007a). Central nervous system disorders. In D. M. Kaufman (Ed.), *Clinical neuropsychiatry for psychiatrists* (6th ed., pp. 5–14). Philadelphia: Saunders Elsevier.

Kaufman, D. M. (2007b). Congenital cerebral impairments. In D. M. Kaufman (Ed.), *Clinical neuropsychiatry for psychiatrists* (6th ed., pp. 295–316). Philadelphia: Saunders Elsevier.

Myers, T. (2006). Extrapyramidal symptoms (definition). In T. Myers (Ed.), *Mosby's dictionary of medicine, nursing & health professions* (7th ed., pp. 697). Missouri: Mosby Elsevier.

Shy–Drager syndrome. (2003). In *MedicineNet*. Retrieved February 3, 2009, from http://www.medterms.com.

Stahl, S. M. (2000). Antipsychotic agents. In S. M. Stahl (Ed.), *Essential psychopharmacology: Neuroscientific basis and practical applications* (2nd ed., pp. 401–458). New York: Cambridge University Press.

Venes, D., Thomas, C., Egan, E., & Houska, A. (2001). Extrapyramidal symptoms (definition). In Venes, D., et al. (Eds.), *Taber's medical dictionary* (19th ed., pp. 760). Philadelphia: FA Davis Company.

Extrastriate

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Synonyms

Broadmann areas 18, 19; Heterotypic visual cortex; Secondary visual cortex; V2, V3, V4, V5

Definition

The extrastriate cortex refers to occipital cortical areas found anterior to the striate cortex (i.e., primary visual cortex), corresponding to visual areas 2, 3, 4, and 5 in primates. The extrastriate consists of heterotypic cortical neurons that may respond to different types of inputs relative to the primary visual information arising from the primary visual cortex. Accordingly, it plays an essential role in mid-level visual processing or what neuropsychologists often refer to as visual integration. Extrastriatal neurons tend to respond to visual stimuli within receptive fields, with these responses modulated by other cognitive processes, such as motivational signals, attention, working memory, and response demands.

Cross References

- ▶ Cortical Magnification
- ▶ Enhancement
- ▶ Feature Detection
- ▶ Lateral Inhibition
- ▶ Visual Integration
- ▶ 'What System'
- ▶ 'Where System'

Eye Fields

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Definition

Visual fields refer to the entire region of space observed by both eyes. Visual fields can be subdivided into right and left hemifields. Cardinal positions are the specific eye movements that assess the function of extraocular muscles and the cranial nerves that innervate them.

Current Knowledge

Visual Fields

Visual fields, the entire region of space observed by both eyes, can be divided either into central and peripheral regions or into right and left hemifields. Laterally located retinal cells respond to visual stimuli in central regions, whereas medially located retinal cells respond to stimuli in peripheral portions of the ipsilateral visual field. A lesion to an entire eye or its optic nerve therefore results in the loss of ipsilateral peripheral vision. Central ipsilateral and all of the contralateral visual field regions are still seen by the unaffected, contralateral eye.

As ganglion cells project their fibers into the CNS, visual pathways are reorganized. Medially located ganglion cell fibers cross at the optic chiasm while those from laterally located ganglion cells remain uncrossed. As a result of partial crossing, optic tracts and visual processing centers in the brain (i.e., the lateral geniculate nucleus of the thalamus [LGN] and the primary visual cortex) receive input only from contralateral visual hemifields. Transection of the optic tract or a thalamic lesion therefore results in the loss of an entire contralateral hemifield.

A second reorganization of visual information occurs as LGN neurons relay information to visual cortex. Visual information from the superior quadrant of the contralateral hemifield is sent to inferior regions of visual cortex by thalamic fibers that follow a pathway called Meyer's loop. Conversely, fibers carrying information from the inferior quadrant follow the optic radiations to terminate in superior regions of the visual cortex. Therefore, a lesion to the optic radiations or the superior primary visual cortex results in the loss of vision from the inferior quadrant of the contralateral hemifield. Likewise, a lesion in Meyer's loop or in the inferior primary visual cortex results in the loss of vision from the superior quadrant of the contralateral hemifield.

Eye Movements

Eye movements are controlled by six extraocular muscles: superior, inferior, medial and lateral rectus muscles, and superior and inferior oblique muscles. Three cranial nerves (CNs) originating from the brainstem innervate these muscles: trochlear nerves (CN IV) innervate superior oblique muscles, abducens nerves (CN VI) innervate lateral rectus muscles, and occulomotor nerves (CN III) innervate the rest. Clinicians test extraocular muscle function, and importantly the nerves innervating them, with cardinal positions – sequential movements that determine individual extraocular muscle function. Eyes are first directed to one side, testing the medial rectus of one eye and the lateral rectus of the other. While looking to one side, eyes are shifted upward, testing the superior rectus of the laterally gazing eye and the inferior oblique of the medially gazing eye. While still looking to one side, eyes are then shifted downward, testing the inferior rectus of the laterally gazing eye and the superior oblique of the medially gazing eye. With these six cardinal positions and the knowledge of nerves innervating each extraocular muscle, clinicians can determine potential lesions sites in the brainstem that lead to defects in eye movements.

Cross References

- ▶ Cranial Nerves
- ▶ Frontal Eye Fields
- ▶ Lateral Geniculate Nucleus of Thalamus
- ▶ Visual Cortex

References and Readings

Bear, M. F., Connors, B. W., & Paradiso, M. A. (2001). *Neuroscience: Exploring the brain* (2nd ed.). Baltimore, MD: Lippincott Williams & Wilkins.

Snell, R. S. (1995). *Clinical anatomy for medical students* (5th ed.). Boston, MA: Little, Brown and Company.

Eye Movements

► Conjugate Gaze

Eye Preference

► Lateral Dominance

Eysenck Personality Inventory

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Synonyms

EPI

Definition

The Eysenck Personality Inventory (EPI) is a self-report instrument designed to measure two central dimensions of personality, extraversion and neuroticism. This instrument is comprised of 57 yes/no items and yields total scores for extraversion and neuroticism as well as a validity score (e.g., Lie Scale). Individuals are generally classified as "high" or "low" on the two dimensions. Persons high in extraversion are seen as social, carefree, and optimistic, while low scorers are generally quiet, introspective, and reserved. Individuals classified as high in neuroticism are prone to emotional distress/instability, while those low in this dimension are generally calm and emotionally stable.

Current Knowledge

Test Theory, Development, and Properties

The EPI was developed in 1964 based on a conceptualization of personality that identifies extraversion and neuroticism as the two primary and independent factors comprising the global construct of personality. The specific inventory was constructed from an existing assessment (the Maudsley Personality Instrument; Eysenck & Eysenck, 1964) similarly based on this two-factor model; individual test items for the EPI (and its predecessor) were selected via factor analysis. Subsequent developments included creation of alternate forms (A & B), reported lowering of the reading level (although prior and current reading level are not specified in the manual or associated references), refined item selection to further distinguish the extraversion and neuroticism scales, addition of a 9-item "Lie Scale" to provide an index of validity, and improved test–retest reliability. The EPI Manual notes reliability estimates for the EPI ranging from 0.81 to 0.97 for test–retest reliability and from 0.74 to 0.91 for split-half reliability.

Revisions and Alternate Versions

Since its initial development, the EPI has undergone multiple transformations, including the 1975 expansion to the 90-item Eysenck Personality Questionnaire (EPQ), which added items to assess a third personality dimension (Psychoticism), and revision of the EPQ Psychoticism Scale in 1985 to create the 100-item Eysenck Personality Questionnaire – Revised (EPQR). Each revision was designed to expand the scope of personality characteristics evaluated by the instrument. These revisions also increased the administration time of the instrument, which posed concerns for some and ultimately prompted development of several abbreviated forms (e.g., EPQR-A, EPQR-S, etc.; Francis, Brown, & Philipchalk, 1992).

Suggested Uses and Relevant Research

The EPI Manual recommends use of this measure across research settings as well as in the diagnosis and treatment of individual personality dysfunction. Current use of the EPI for clinical assessment and

diagnosis is arguably surpassed by other personality assessments (e.g., the Minnesota Multiphasic Personality Inventory – 2, Personality Assessment Inventory). However, empirically, the EPI continues to be used to assess the relations between specific personality characteristics and a variety of psychological phenomena. Relevant findings include those suggesting that individuals involved in traumatic accidents leading to disability in motor functioning tend to be higher in extroversion than those with nontraumatic injuries (Malec, 1985); individuals high in extroversion and low in neuroticism have a lower risk for developing dementia than those with other personality profiles (Wang et al., 2009); and individuals higher in neuroticism tend to be at greater risk for acute stress disorder, following mild traumatic brain injury (Bryant & Harvey, 1998). It is noteworthy that while these relations (and others) have been documented in individual studies, replication of findings is generally limited. Furthermore, while discussions of the clinical implications of these correlations exist, empirical investigation of clinical utility is lacking.

Cross References

- ▶ Minnesota Multiphasic Personality Inventory
- ▶ NEO Personality Inventory
- ▶ Neuroticism
- ▶ Personality Assessment Inventory

References and Readings

Bryant, R. A., & Harvey, A. G. (1998). Predictors of acute stress following mild traumatic brain injury. *Brain Injury, 12*(2), 147–154.

Eysenck, H. J., & Eysenck, S. B. G. (1964). *Manual of the eysenck personality inventory.* London: University of London Press.

Francis, L. J., Brown, L. B., & Philipchalk, R. (1992). The development of an abbreviated form of the revised Eysenck Personality Questionnaire (EPQR-A): Its use among students in England, Canada, the U.S.A. and Australia. *Personality and Individual Differences, 13*(4), 443–449.

Malec, J. (1985). Personality factors associated with severe traumatic disability. *Rehabilitation Psychology, 30*(3), 165–172.

Wang, H. X., Karp, A., Herlitz, A., Crowe, M., Kareholt, I., Winblad, B., et al. (2009). Personality and lifestyle in relation to dementia incidence. *Neurology, 72*, 253–259.