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## Wada Test

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### Definition

The Wada test is named after a neurologist Juhn A. Wada and is also known as the “intracarotid sodium amobarbital procedure” (ISAP) (Wada 1949). This test evaluates the effects of successive unilateral hemianesthesia of the first cerebral hemisphere and then the second, the purpose of which is to determine which of the cerebral hemispheres is predominately responsible for essential cognitive, memory, and language processes. Wada was developed and established by Dr. Wada as a standard test for the presurgical evaluation of patients with intractable epilepsy prior to surgical ablation. The main purpose of this test is to ensure that the nonoperative cerebral hemisphere can sustain adequate language and memory function following elective ablative surgery on the contralateral hemisphere.

ISAP includes the injection of an anesthetic, usually 75–200 mg of sodium amobarbital, into the right or left internal carotid artery supplying either of the cerebral hemispheres. Within seconds of injection, the language and memory functions of the affected cerebral hemisphere are shut down,

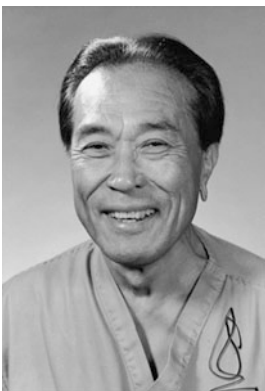
and a reversible hemiparesis occurs. During this time, the patient undergoes language and neuropsychological assessments to isolate and evaluate the essential abilities subserved by the unaffected cerebral hemisphere. Since its introduction in 1949, the ISAP test has evolved from an experimental procedure to a widely used clinical assessment tool for speech and memory lateralization. Advances in noninvasive neuroimaging techniques, particularly magnetic resonance imaging (MRI) and functional magnetic resonance imaging (fMRI) have now offer safer alternatives to the Wada test in the preoperative evaluation and prediction of outcome in patients undergoing surgery for epilepsy. However the Wada test continues to be considered as the “gold standard” and continues to contribute in the presurgical assessment of patients with intractable seizures particularly when safer and less invasive neuroimaging techniques are inadequate or equivocal.

### Historical Background

Dr. Juhn Wada was born on March 24, 1924, in Tokyo, Japan (Van Emde Boas and Juhn 1999). He studied medicine at the Hokkaido Imperial University and received his medical license in 1947 and a PhD in medicine in 1951. In 1949, he first introduced the Wada test as a method to allow unilateral electroconvulsive shock therapy for psychosis (Van Emde Boas and Juhn 1999).

Dr. Wada was appointed as a lecturer at Hokkaido Imperial University in 1951 and as an assistant professor in 1953; between 1953 and 1957, he succeeded in developing a brain surgery program and established the department of Neurosurgery at Hokkaido Imperial University (Van Emde Boas and Juhn 1999). During these years, he also spent some time in Canada and the United States. In 1955–1956, he worked at the Wilder Penfield in the Montreal Institute, Quebec, and obtained his Canadian medical degree and citizenship. Since 1960, Dr. Wada had held appointments at the University of British Columbia in Vancouver, and, in 1970, he was appointed as the Full Professor and Director of the Departments of Clinical Neurophysiology, Seizure Investigation Unit, Experimental Epileptology and of the Epilepsy Surgery Program, all of which he had initiated (Van Emde Boas and Juhn 1999).

Dr. Wada has a long and distinguished career in the study of neurobiological mechanisms of epilepsy and the asymmetry of human brain functions. He has published extensively on kindling, the induction of epilepsy in animals, secondary epileptogenesis and pathophysiological processes, and treatment of seizure disorders (Van Emde Boas and Juhn 1999). With the introduction of the ISAP, Dr. Wada has contributed extensively to the understanding of language localization and its relation with handedness (Van Emde Boas and Juhn 1999) (Fig. 1).



**Wada Test, Fig. 1** Juhn A. Wada

## Current Knowledge

Baxendale (2009) reported a significant shift and decline in the use and role of the Wada test in the presurgical evaluation of epilepsy in light of research on the reliability and validity of noninvasive alternatives such as functional magnetic resonance imaging (fMRI) and/or magnetoencephalography. In 2009, these authors reported that the majority of epilepsy centers no longer conducted a Wada test on every surgical candidate and that fMRI was being used in some centers to lateralize and localize language function in epilepsy surgery candidates. These authors also emphasized that in view of the risks associated with the Wada test, the clinical indications for a Wada test should be determined on a case-by-case basis, and consideration should be given to available noninvasive alternatives, to ensure that the risk-benefit ratio is appropriate for every patient (Baxendale 2009).

Current literature review supports the decline in use of the Wada test with the development of advances in the use of less invasive neuroimaging techniques, especially fMRI, along with multiple clinical, demographic, and multivariate models of cerebral function for the preoperative assessment of patients and risk of memory or language decline following surgical procedure. Since 2009 fMRI as well as advances in other noninvasive neuroimaging techniques have continued to offer safer alternatives to the Wada test in the preoperative evaluation and prediction of outcome in patients undergoing surgery for epilepsy.

In a review article in 2015, Dupont (2015) reported that numerous studies have demonstrated that the Wada test could reliably be replaced by fMRI for presurgical evaluation of patients with medial temporal lobe epilepsy (MTLE); however this author noted that there was considerable individual variation in the extent, nature, and direction of postoperative memory change in these patients and that patients who were surgical candidates needed precise information about the potential cognitive after effects, particularly postoperative memory changes following temporal lobe epilepsy surgery. Clinical and neuropsychological data in

addition to fMRI added useful information preoperatively to predict the postoperative memory outcome; however Dupont noted that this information was not always sufficient to replace the Wada test, which is considered “the gold standard” in predicting postoperative decline following surgery for epilepsy. Furthermore Dupont reported that fMRI studies seemed to suggest that it is the functional adequacy of the resected hippocampus rather than the functional reserve of the contralateral hippocampus that was important in the determination of the extent of postoperative memory decline and emphasized that new functional neuroimaging procedures that explore more widespread network disruptions commonly found in MTLE such as diffusion tensor imaging (DTI) or connectivity studies could in the future constitute a reliable approach, combined with fMRI activation studies, to significantly improve the prediction of postsurgical memory impairment following surgical resection for epilepsy.

Suarez et al. (2014) designed a study to evaluate passive language fMRI protocols for clinical application in pediatric epilepsy to validate usefulness in very young patients where overt language participation may not be possible. These authors compared two fMRI language mapping paradigms, one active in nature (requiring participation from the patient) and the other passive in nature (requiring no participation from the patient), and concluded that language activation maps could reliably be achieved using the passive language fMRI protocols even in very young (average 7.5 years old) or sedated pediatric epilepsy patients.

Although fMRI techniques can usually accurately lateralize language, the Wada test as considered by many as the “gold standard” for preoperative lateralization is occasionally still required if there are nondiagnostic findings on fMRI. Chiu et al. (2015) reported that apparently amobarbital, the agent of choice for the Wada test, has become increasingly difficult to obtain and requires regulatory approval and suggested that propofol may be an acceptable alternative. These authors performed a literature review and outlined a case study of a patient who underwent a Wada test

using propofol and suggested a protocol for the use of propofol for a Wada test when necessary.

In conclusion, evolving noninvasive or less invasive neuroimaging techniques, particularly fMRI, along with clinical, neuropsychological, and multimodal assessments continue to advance in scope and reliability to offer less invasive alternative techniques to the Wada test in the preoperative evaluation of a patients with intractable epilepsy. However the Wada test remains the “gold standard test” and continues to be utilized especially when less invasive, multimodal preoperative assessments are inconclusive or equivocal.

## Future Directions

Continued advances in safer and less invasive functional neuroimaging and multimodal assessments are anticipated to gradually replace the Wada test in its current adjunctive role in the preoperative assessment of patients requiring surgery for intractable epilepsy.

## Cross-References

- ▶ [Epilepsy](#)
- ▶ [Intracarotid Sodium Amobarbital Test](#)
- ▶ [Localization](#)

## References

- Baxendale, S. A. B. (2009). The Wada test. *Current Opinion in Neurology*, *22*(2), 185–189.
- Chiu, A. H., Bynevelt, M., Lawn, N., Lee, G., & Singh, T. P. (2015). Propofol as a substitute for Amobarbital in Wada testing. *Journal of Clinical Neuroscience*, *22*(11), 1830–1832.
- Dupont, S. (2015). Imaging memory and predicting postoperative memory decline in temporal lobe epilepsy: Insights from functional imaging. *Revue Neurologique*, *171*(3), 307–314.
- Suarez, R. O., Taimouri, V., Boyer, K., Vega, C., Rotenberg, A., Madsen, J. R., Loddenkemper, T., Duffy, F. H., Prabhu, S. P., & Warfield, S. K. (2014). Passive fMRI mapping of language function for paediatric epilepsy surgical planning: Validation using Wada, ECS, and FMAER. *Epilepsy Research*, *108*(10), 1874–1888.

Van Emde Boas, W., & Juhn, A. (1999). Wada and the sodium amytal test in the first (and last?) 50 years. *Journal of the History of the Neurosciences*, 8(3), 286–292.

Wada, J. (1949). *A new method for the determination of the side of cerebral speech dominance*. A preliminary report of the intra-carotid injection of sodium amytal in man (Vol. 14, pp. 221–222). Tokyo: Igaku to Seibutsugaki.

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## Waisman Center, University of Wisconsin-Madison

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The Waisman Center at the University of Wisconsin-Madison is dedicated to the advancement of knowledge about human development, developmental disabilities, and neurodegenerative diseases. One of only 14 centers of its kind in the United States, the Waisman Center encompasses laboratories for biomedical and behavioral research, a brain imaging center, and a clinical biomanufacturing facility for the production of pharmaceuticals for early-stage human clinical trials.

In addition to its research efforts, the Waisman Center provides highly specialized clinical care and support to children and families affected by a broad range of developmental disabilities that include autism, Down syndrome, cerebral palsy, and genetic disorders through 11 specialty clinics and 3 autism treatment service programs.

The center also has an integrated preschool, statewide capacity-building initiatives to improve the quality of life of individuals with disabilities and their families and trains scientists and clinicians who will serve our nation in the future.

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## Wallenberg's Syndrome

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### Synonyms

Lateral medullary syndrome

### Short Description or Definition

Described in 1895 by Wallenberg and later outlined by Fisher et al. (1961), this syndrome is produced by a lesion in a wedge of lateral medulla lying posterior to the inferior olivary nucleus. It is frequently the result of an infarction, and however, although much rarer, may also be due to a small well-localized hemorrhage or a tumor. The complete syndrome involves the vestibular nuclei, spinothalamic tract, descending sympathetic tract, fascicles of the ninth and tenth nerves, otolith nucleus, olivocerebellar and/or spinocerebellar fibers, and restiform body. The symptoms most frequently associated with this syndrome are ataxia (84%), numbness (78%), dysphagia (69%), vertigo (60%), nausea (58%), dysarthria (49%), headache (44%), as well as hoarseness, facial pain, hiccups, and others. Long-term disability affecting multiple domains can result from this strategically localized infarction.

### Symptoms

See Table 1.

### Etiology

The most common etiology of a classical Wallenberg's syndrome remains ischemic. Although traditionally described as arising from the posterior inferior cerebellar arterial (PICA) territory, it has been more accurately localizable to territory of

**Wallenberg's Syndrome, Table 1** Cerebral localizations and their associated symptoms commonly seen in Wallenberg syndrome

Structures involved	Common associated symptoms
Descending sympathetic tract	Ipsilateral Horner's syndrome (miosis, ptosis, anhidrosis)
Anterior spinocerebellar tract	Ataxia and muscle hypotonia
Inferior cerebellar peduncle	Limb ataxia and asynergia
Anterior spinothalamic tract	Loss of sensation of pain and temperature in the contralateral body and extremities
Spinal trigeminal nucleus (CN V)	Loss of sensation of pain and temperature on the face; reduced corneal reflex; occasionally, pain and burning of the ipsilateral face (Dejerine Roussy syndrome)
Nucleus ambiguus (CN IX)	Dysarthria and dysphagia (paralysis of palatal elevation pharyngeal constrictors, and vocal cord)
Dorsal nucleus of the vagus nerve (CN X)	Tachycardia and dyspnea, ipsilateral paralysis of the palate and vocal cords
Inferior vestibular nucleus (CN VIII)	Nystagmus, oscillopsia, skew deviation, vertigo, nausea, vomiting
Cochlear nucleus (CN VIII)	Hyperacusis
Nucleus tractus solitarii	Ageusia (loss of taste)
Restiform body	Ipsilateral ataxia, falling to the ipsilateral side, ipsiversive lateropulsion

thrombotically or embolically occluded medullary branches off the ipsilateral vertebral artery that course through the lateral medullary fossa and supply the lateral medulla. A local vessel atherosclerotic plaque formation and/or an ostial branch occlusion from a ruptured plaque may result in thrombotic etiology. Alternatively, a distant embolus from the parent vertebral artery, either at the origin or from a cardiac or aortic arch, can also be the source of embolic material.

Atherosclerosis is by far the most common cause of vertebrobasilar ischemia, making it most common among patients with cardiovascular risk factors such as age, hypertension, diabetes mellitus, smoking, and

dyslipidemias. In general, vertebrobasilar ischemia may result from any disease process that has an impact on the arterial supply to the posterior fossa, including the following: fibromuscular dysplasia, rotational occlusion (Bow hunter's stroke), mechanical occlusion or stenosis of the vertebral artery at the C1-C2 level caused by lateral flexion, vertebral artery dissection, vertebrobasilar aneurysms, or severe dolichoectasia of the vessels of the posterior circulation.

Hemorrhage (primary or secondary to a tumor) or a malignancy is a rare alternative to stroke in patients with isolated lateral medullary syndrome and can usually be easily ruled in or out by imaging characteristics.

## Evaluation

Fragmentary syndromes that do not involve the complete syndrome frequently occur at the onset of the syndrome. Thus, vertigo and ptosis, ataxia and vertical diplopia, or hoarseness and disequilibrium may be the initial presenting features. Hiccups, frequently seen with the classical "Wallenberg syndrome," are actually a syndrome of the posterior medullary region which can be affected, likely, through local edema. Hiccups are usually self-limited within a few days except in isolated cases where they may last longer and are refractory to therapies. Importantly, isolated vertigo is almost never a concern for lateral medullary syndrome. Adams and Victor et al. report the smallest lateral medullary syndrome that they have encountered which comprised symptoms of lateropulsion and mild ipsilateral ataxia. Internuclear ophthalmoplegia or a skew deviation may be present (with the affected side's globe usually in the hypertrophic position).

Neurological examination should focus on assessing the degree of swallowing and gait disability brought on by the full or incomplete lateral medullary syndrome, as well as on formulating a rehabilitation plan with the help of physiatrists and physical, occupational, and speech therapists.

In terms of etiology, computerized tomography (CT) of the head may help rule out CNS hemorrhage or mass effect secondary to cerebellar

infarction. However, CT is generally not a good modality for detecting posterior fossa, including brain stem, pathologies because of bony interference. Hence, magnetic resonance imaging (MRI) is far superior to CT for brain stem and posterior fossa imaging and is more sensitive to small ischemic areas due to branch occlusion off the large arteries of the vertebrobasilar circulation. Magnetic resonance angiography (MRA) may be as good as cerebral angiography for detecting occlusions and stenoses of the vertebrobasilar circulation, but it may not be as good for quantifying degree of stenosis. CT angiography with iodinated contrast may be utilized to look at flow characteristics through the large extra- and intracranial vessels of cerebral circulation. Large vessel anatomy may be definitively assessed via conventional angiography. Transcranial and cervical Doppler ultrasonography may complement MRA and provide important hemodynamic data on degree of extracranial vertebral as well as vertebrobasilar stenosis disease. If cardioembolic source is suspected, a transthoracic or transesophageal echocardiogram may be performed to rule out aortic arch or intracardiac clot presence. Telemetry monitoring can rule out cardiac arrhythmias. In most individuals their lipid profile, diabetes screen, and routine blood work are performed. Some patients also undergo testing for hypercoagulable or inflammatory etiologies.

### **Natural History, Prognostic Factors, and Outcomes**

The onset of symptoms is sudden in only 40% of cases. More often it is progressive over 24–48 h. A quarter of the patients report transient attacks preceding the fixed deficit. Even alert patients with good extremity strength may have compromised swallowing mechanisms. Recovery is slow, but many patients go on to regain ability to walk independently within 3 months and do not require long-term alternative feeding, that is, percutaneous endoscopic gastrostomy (PEG) tube. When feeding the elderly, precautions should be taken, as aspiration pneumonitis remains the leading cause of morbidity and mortality in patients

with vertebrobasilar strokes. Overall, the outcomes are not always favorable. Poor outcome may be related to the potential mass effect from the coexisting cerebral infarcts, cardiovascular and respiratory dysfunctions, clot propagation/embolization, and bilateral vertebral artery disease. Cerebellar infarctions causing brainstem compression, acute hydrocephalus, or herniation may also occur though are rare.

### **Treatment**

Treatment and management options are targeted at specific areas of dysfunction as well as at secondary prevention. More severe cases may have significant dysphagia, ataxia, and vertigo each of which needs to be managed individually. Physical, occupational, and speech therapy should be utilized for restoration of gait and balance, manual dexterity, and swallowing technique. In cases of ischemic disease, secondary stroke prevention measures will need to be undertaken as per the American Heart Association guidelines and depending on the stroke etiology. In cases of likely atherosclerotic disease of the vertebral artery or its branches, the use of an antiplatelet agent (currently, aspirin, clopidogrel, or extended-release aspirin/dipyridamole) is warranted unless medically contraindicated. Anticoagulation is limited to select cases of extracranial acute vertebral dissection and is definitively recommended for most cases of atrial fibrillation in older adults unless medically contraindicated. A risk-benefit ratio needs to be evaluated in patients with severe truncal ataxia or gait disturbance as frequent falls may pose significant risk in those patients on anticoagulation. In addition, acute use of anticoagulation in large ischemic strokes (i.e., those with strokes in addition to the small lateral medullary infarction) should be used with caution as risk of hemorrhagic conversion may outweigh the benefit of anticoagulation and should be discussed on a case-by-case basis. Control of modifiable risk factors of stroke, including smoking, blood pressure, diabetes, and cholesterol are the mainstay of any secondary stroke prevention.



## References and Readings

- Bogousslavsky, J., & Caplan, L. R. (2001). *Stroke syndromes*. New York: Cambridge University Press.
- Caplan, L. (2000). Posterior circulation ischemia: Then, now, and tomorrow. The Thomas Willis Lecture. *Stroke*, 31(8), 2011.
- Caplan, L. R., Pessin, M. S., Scott, R. M., & Yarnell, P. (1986). Poor outcome after lateral medullary infarcts. *Neurology*, 36, 1510–1513.
- Mohr, J. P., James, C. G., & Dennis, W. C. (Eds.). (2004). *Stroke: Pathophysiology, diagnosis and management*. Philadelphia (not Elsevier): Elsevier Health Sciences/Churchill Livingstone.
- Ropper, A. H., Brown, R. H., Adams, R. D., & Victor, M. (2005). *Principles of neurology*. New York: McGraw-Hill Medical Publishers.
- Shoja, M. M., Tubbs, R. S., Loukas, M., & Ardalan, M. R. (2008). Adolf Wallenberg (1862–1949): Physician and neuroanatomist. *Childs Nervous System*, 24(9), 979. [Epub ahead of print].

## Warfarin (Coumadin)

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## Synonyms

Blood Thinner; Coumadin<sup>®</sup>

## Definition

Warfarin (trade name Coumadin<sup>®</sup>) is an oral anti-coagulant that is most frequently used to prevent or control thromboembolic disorders such as stroke, myocardial infarction, peripheral artery disease, venous thrombosis, pulmonary embolism, and others.

## Current Knowledge

By inactivating the production of Vitamin K-dependent coagulation factors II, VII, IX, and X that help to form blood clots, warfarin makes it

more difficult for the system to create a clot. However, as a result of its anticoagulant effect, it also can cause a number of hemorrhagic complications, including gastrointestinal bleeding and cerebral hemorrhage. In order to insure appropriate anticoagulation effect while minimizing the risk of adverse effects, a simple blood test, the International Normalized Ratio (INR), can be used to measure, monitor, and adjust the anticoagulation level. The ideal target INR level depends on the specific clinical situation, but generally the target range is between 2 and 3. Administration of Vitamin K reverses the anticoagulation effect. Patients treated with warfarin require close monitoring to avoid bleeding, but the drug has been shown to prevent 20 strokes for every bleeding episode that it causes (Horton and Bushwick 1999).

## Cross-References

- ▶ [Anticoagulation](#)
- ▶ [Heparin](#)
- ▶ [Thrombosis](#)

## References and Readings

- Holbrook, A. M., Pereira, J. A., Labiris, R., et al. (2005). Systematic overview of warfarin and its drug and food interactions. *Archives of Internal Medicine*, 165, 1095–1106.
- Horton, J. D., & Bushwick, B. M. (1999). Warfarin therapy: Evolving strategies in anticoagulation. *American Family Physician*, 59, 635–646.

## Warrington Recognition Memory Test

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## Description

The Recognition Memory Task by Warrington consists of two recognition tasks. One is

recognition of words and the other is recognition of faces. It takes approximately 15 min to administer.

## Historical Background

The Recognition Memory Test was designed to quickly assess material-specific memory deficits. Both verbal and nonverbal memory are tested in order to allow clinicians to make judgments about lateralization of brain damage, right hemisphere versus left hemisphere. It has been criticized for poor reliability and poor specificity (see Sweet et al. 2000).

## Psychometric Data

Normative data is included on 300 individuals aged 18–70 years in the test package provided by Western Psychological Services. Test-retest reliability for the faces subtest was reported to be 0.81, and validity was reported to be moderate (Soukup et al. 1999). The subtests have adequate internal consistency (Malina et al. 1998), but there is evidence of a significant ceiling effect on the words subtest (Naugle et al. 1994). There are no parallel forms.

As regards validity, despite the goals of the test, there is limited evidence that it is useful in diagnosing laterality of brain damage (Sweet et al. 2000), and it has not been shown to be related to performance on the Wechsler Memory Scale (Compton et al. 1992).

## Clinical Uses

It is a brief screening test of verbal and nonverbal memory that is designed for use in a wide variety of populations, particularly, in those who have had cerebral vascular accident or other acquired brain injury. It has been used with individuals with

epilepsy and in children with autism. More recently, it has also been used as a test of response bias by looking at the probability of obtaining particular scores on the test (Kim et al. 2010; Millis 2002).

## Cross-References

- ▶ [Auditory Verbal Learning](#)
- ▶ [Benton Visual Retention Test](#)
- ▶ [California Verbal Learning Test \(California Verbal Learning Test-II\)](#)
- ▶ [Hopkins Verbal Learning Test](#)
- ▶ [Recognition Memory Test](#)
- ▶ [Rey Auditory Verbal Learning Test, Rey AVLT](#)
- ▶ [Wechsler Memory Scale All Versions](#)

## References and Readings

- Compton, J., Sherer, M., & Adams, R. (1992). Factor analysis of the Wechsler memory scale and the Warrington recognition memory test. *Archives of Clinical Neuropsychology*, *7*, 165–173.
- Kim, M., Boone, K., Victor, T., Marion, S., Amano, S., Cottingham, M., et al. (2010). The Warrington recognition memory test for words as a measure of response bias: Total score and response time cutoffs developed on “real world” credible and noncredible subjects. *Archives of Clinical Neuropsychology*, *25*, 60–70.
- Malina, A., Bowers, D., Millis, S., & Uekert, S. (1998). Internal consistency of the Warrington recognition memory test. *Perceptual and Motor Skills*, *86*, 1320–1322.
- Millis, S. (2002). Warrington’s recognition memory test in the detection of response bias. *Journal of Forensic Neuropsychology*, *2*, 147–166.
- Naugle, R., Chelune, G., Schuster, J., Luders, H., & Comair, Y. (1994). Recognition memory for words and faces before and after temporal lobectomy. *Assessment*, *1*, 373–381.
- Soukup, V., Bimbela, A., & Schiess, M. (1999). Recognition memory for faces: Reliability and validity of the Warrington recognition memory test in a neurological sample. *Journal of Clinical Psychology in Medical Settings*, *6*, 287–293.
- Sweet, J., Demakis, G., Ricker, J., & Millis, S. (2000). Diagnostic efficiency and material specificity of the



Warrington recognition memory test: A collaborative multisite investigation. *Archives of Clinical Neuropsychology*, 15, 301–309.

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## Warrington, Elizabeth

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### Major Appointments

- Research Assistant, Experimental Psychology, Institute of Neurology, Queen Square, London (1954–1955)
- Research Fellow, Experimental Psychology, Institute of Neurology, Queen Square, London (1956–1959)
- Clinical Psychologist, Department of Psychological Medicine, National Hospital, Queen Square, London (1960–1982)
- Head of the Department of Clinical Neuropsychology, National Hospital for Neurology and Neurosurgery, Queen Square, London (1982–1996)
- Professor of Neuropsychology, National Hospital for Neurology and Neurosurgery, Queen Square, London (1982–1996)
- Kenneth Craik Fellowship, St. John's College, Cambridge (1981–1982)
- Board of Directors, Royal Hospital for Neurodisability, London (2000)
- President-Elect, International Neuropsychological Society (2003)
- Fellow of University College (1994–present)
- Emeritus Professor in London University (1996–present)
- Honorary Consultant Neuropsychologist, The National Hospital for Neurology and Neurosurgery (1996–present)

### Major Honors and Awards

- Fellow, The Royal Society (1986)
- Laurea ad honorem in psicologia, Bologna University, Italy, (Honorary Doctorate) (1998)
- Honorary Doctorate, York University, England (1999)
- Docteur Honoris Causa, Universite Louis Pasteur, Alsace, France (Honorary Doctorate) (2006)

### Landmark Clinical, Scientific, and Professional Contributions

- Dr. Elizabeth Warrington has been a central figure in establishing and advancing neuropsychology in Britain and internationally through clinical practice and research. Her research has focused on cognitive sequelae of brain injury and using that information to make distinctions about the organization and processes of neural networks within the brain. Her combination of research and clinical practice enabled her to broaden the understanding and diagnosis of brain dysfunction.
- Warrington's practice as a clinical neuropsychologist at the National Hospital for Neurology and Neurosurgery in London brought to her attention interesting single cases, careful study of which allowed her to develop theories from patterns observed in patients with similar symptoms. Excited by the incongruity of several single cases, and as a strong proponent of single case methodology, Warrington devised tests to diagnose the different symptoms she was observing, and has designed and expanded numerous diagnostic tests across a variety of cognitive domains including memory, language, and visual-perceptual disorders including blind sight. For example, she developed the well-known Warrington Recognition Memory Test (RMT) and the Unusual Views Test.
- In the early stages of her career (1961–1966), Warrington worked with Oliver Zangwill and Marcel Kinsbourne, among others, publishing

findings on topics such as finger agnosia, simultagnosia, alexia, aphasia, and disorders of short-term visual memory. Her collaborations with Lawrence Weiskrantz led to the identification a form of memory now formally defined as implicit memory. Warrington was one of the first neuropsychologists to distinguish between short-term and long-term memory as distinct processes. Her study with Tim Shallice of a patient with a disorder in auditory short-term memory led to a publication on the theory of memory as a multifaceted and not entirely homogenous process, contrary to the orthodox view that considered long-term memory merely on a continuum with short-term memory. Her collaboration with Shallice played a major role in the development of cognitive neuropsychology. As Warrington continued to research short-term memory, she further differentiated it into auditory and visual stores, collaborating with Alan Baddeley, whose well-known model of working memory is based on auditory and visual differences in memory. Together they reported their findings on the distinctive influences of auditory and visual aspects of memory in amnesia.

- In 1975, Warrington was the first to describe the condition now known as semantic dementia, identifying differences in the storage of episodic memory versus semantic memory. Warrington later collaborated with Rosaleen McCarthy to study impairments in specific domains of cognitive functioning such as reading, naming and comprehension, and the independent organization of seemingly similar things: verbs versus nouns, abstract versus concrete, etc. They assessed the differences in these independent processes, which were observed, for instance, in patients who could identify animate but not inanimate objects, and vice versa, as a result of lesions in different loci.
- Dr. Elizabeth Warrington's research has led to over 200 publications in peer-reviewed journals on a range of topics including visual recognition disorders, semantic memory disorders, and various forms of acquired dyslexia and dysphasia. Her dedication to the field of

neuropsychology, both in research and clinical practice, has greatly influenced and changed the theories and tests associated with cognitive functioning and brain damage.

### Short Biography

Warrington was born as Elizabeth Kerr Butler to John Alfred and Margaret Lois Butler. She graduated with a Bachelor of Science from the University College London in 1954. She received her Ph.D. from the London University in 1960; her doctoral research focused on the "visual completion" effect, in which hemianopic patients report seeing a whole shape despite part of it being presented within the "blind" hemifield. At University College London, Warrington continued her work as a clinical neuropsychologist at the Institute of Neurology and the National Hospital for Neurology and Neurosurgery in Queen Square, where she would spend her entire career.

In 1970, Warrington established an independent department of Clinical Neuropsychology within the National Hospital. In 1975, Warrington earned her Doctorate of Science from the University of London. In 1982, she became the head of the department and remained so until her retirement in 1996. In addition to her clinical practice and research, Warrington was Professor of Clinical Neuropsychology at the National Hospital of Neurology and Neurosurgery from 1982 to 1996.

In 1986, Dr. Elizabeth Warrington was elected by her peers as a Fellow of the Royal Society, a prestigious award recognizing her dedication and intellectual curiosity in the field of neuropsychology. Since her retirement, Warrington has remained fully engaged in research as an emeritus professor within the Dementia Research Group that is jointly based at the Institute of Neurology and the National Hospital for Neurology and Neurosurgery. In 2002, the British Neurological Society honored her achievements by creating the Elizabeth Warrington Prize, which is given annually to an individual who has contributed significantly to the field of neuropsychology,

particularly in the early stages of their career. Warrington served as President of the International Neuropsychological Society in 2003.

Warrington has one daughter, Jane Margaret Decca Warrington.

**Principal Publications**

Warrington is the author of over 200 peer-reviewed journal articles in neuropsychology. The table lists some landmark publications.

1962	The completion of visual forms across hemianopic field defects. <i>Journal of Neurology, Neurosurgery and Psychiatry</i> , 25, 208–217.
1970	Independent functioning of verbal memory stores: A neuropsychological study. <i>Quarterly Journal of Experimental Psychology</i> , 22, 261–273 (with T. Shallice).
1973	Contribution of the right parietal lobe to object recognition. <i>Cortex</i> , 9, 152–164 (with A. M. Taylor).
1974	Amnesia and memory for visual location. <i>Neuropsychologia</i> , 12, 257–263 (with A. Baddeley).

(continued)

1975	The selective impairment of semantic memory. <i>Quarterly Journal of Experimental Psychology</i> , 27, 635–657.
1988	Visual apperceptive agnosia; A clinicoanatomical study of three cases. <i>Cortex</i> , 24, 13–32 (with M. James).
1990	The dissolution of semantics. <i>Nature</i> , 343, 599–599, (with R.A. McCarthy).
1991	Failure of object recognition due to breakdown of figure-ground discrimination in a patient with normal acuity. <i>Neuropsychologia</i> , 29, 969–980 (with L. D. Kartsounis).

**Author’s Note**

We are especially grateful to Dr. Warrington for her graciousness in provision of biographical information and photos.

Figure 1 is a photo taken on the steps of the Psychology Department in Cambridge at a meeting of the EPS (Experimental Psychology Society) in 1973. Elizabeth Warrington is standing between Donald Broadbent, author of Perception and Communication as well as the Filter Theory,



**Warrington, Elizabeth, Fig. 1** Photo Courtesy of Elizabeth Warrington



**Warrington, Elizabeth, Fig. 2** Photo Courtesy of Elizabeth Warrington

and Bob Audley, former Head of the Psychology Department at University College London. (Photo courtesy of Elizabeth Warrington).

Figure 2 is a recent photo of Elizabeth Warrington.

### Cross-References

- ▶ [International Neuropsychological Society](#)
- ▶ [Kinsbourne, Marcel \(1931–\)](#)
- ▶ [Warrington Recognition Memory Test](#)
- ▶ [Zangwill, Oliver \(1913–1987\)](#)

### References and Readings

- Constitution-BNS. (2003). Retrieved 1 July 2008, from <http://www.psychology.nottingham.ac.uk/bns/Constitution.htm>
- Grusser, O. J., & Landis, T. (1991). *Vision and visual dysfunction, vol. 12: Visual agnosias and other disturbances of visual perception and cognition*. Amsterdam: Macmillan.
- Kolb, B., & Wishaw, I. Q. (1990). *Fundamentals of human neuropsychology*. New York: W.H. Freeman and Company.

Shallice, T. (1988). *From neuropsychology to mental structure*. Cambridge: Cambridge University Press.

The Royal Society. (n.d.). *Professor Elizabeth Warrington FRS-Cognition and behaviour*. Retrieved 1 July 2008, from <http://excellenceinneuroscience.org/page.asp?id=1565>

The Vivian Smith Advanced Studies Institute of the International neuropsychological Society. (2003). Retrieved 3 July 2008, from <http://www.uth.tmc.edu/clinicalneuro/institute/2003/Faculty2.html#Warrington>

Today's Neuroscience, Tomorrow's History. (n.d.). Retrieved 3 July 2008, from <http://www.ucl.ac.uk/histmed/audio/neuroscience/warrington>

### Weber Test

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### Definition

A hearing test in which a vibrating tuning fork is applied to specific points on the midline of the skull and face. Patients are asked to report the location of the sound. Those with normal hearing typically report that the sound appears to be coming from the middle of their head (i.e., they hear equally well in both ears). In unilateral sensorineural hearing loss (nerve deafness) patients report that the sound is heard in the intact ear, while those with conductive loss report the sound as emanating from the affected side. The former can result from the damage to the inner ear (e.g., cochlea) or the vestibulocochlear nerve, while the latter is typically related to problems involving the external auditory canal (such as excess cerumen), the tympanic membrane or the ossicles of the middle ear.

### Cross-References

- ▶ [Auditory Cortex](#)
- ▶ [Auditory System](#)
- ▶ [Cochlea](#)
- ▶ [Neurologic Examination](#)

## References and Readings

Weber, P. C., & Klein, A. J. (1999). Hearing loss. *Medical Clinics of North America*, 83(1), 125–137.

## Wechsler Individual Achievement Test

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## Synonyms

WIAT-III

## Description

The Wechsler Individual Achievement Test-Third Edition (WIAT-III) is an individually administered clinical instrument developed by Pearson and designed to assess academic achievement. Academic achievement is defined as the ability to apply cognitive skills and learned knowledge to grade-level expectations. The results obtained from the administration of the WIAT-III can be utilized to identify academic achievement strengths and weaknesses, inform educational decisions, diagnose a learning disability, and design interventions.

According to the technical manual, the WIAT-III is designed to be administered to individuals aged 4–19 years (or prekindergarten through grade 12).

The WIAT-III consists of 16 subtests designed to evaluate reading, writing, mathematic, listening, and speaking skills. Specific subtests related to reading include early reading skills, word reading, pseudoword decoding, reading comprehension, and oral reading fluency. Specific subtests related to writing include alphabetic writing fluency, spelling, sentence composition, and essay composition. Specific subtests related to mathematics include math problem solving; numerical operations;

math fluency, addition; math fluency, subtraction; and math fluency, multiplication. Lastly, specific subtests related to listening and speaking include listening comprehension and oral expression. The WIAT-III yields the following composite scores: oral language, total reading, basic reading, reading comprehension and fluency, written expression, mathematics, and math fluency.

The administration of the WIAT-III is flexible, allowing examiners to administer the test in test in its entirety or individual subtests dependent upon the student and purpose. However, standardized administration is recommended. Administration time varies between 30 and 145 min and is dependent upon grade level, academic achievement, testing style, and behavior. Administration time is also dependent upon the number of subtests administered, examiners familiarity and style, and establishment of rapport with student. Administration materials include stimulus booklets, response booklet, record form, reading booklet, word cards, and scoring workbook.

The WIAT-III may be scored via the scoring workbook or WIAT-III Scoring Assistant. The WIAT-III Scoring Assistant includes an interactive scoring guide for the essay composition subtest, performs all basic scoring conversions, provides a clinician score report, performs in-depth analysis of skills, provides a parent report, provides a pattern of strengths and weaknesses (PSW) when utilized in conjunction with a cognitive abilities assessment, and reports an ability-achievement discrepancy when administered in conjunction with a cognitive abilities assessment.

Raw scores for the subtests are converted to age- and grade-based standard scores, which then can be combined into composite scores for each of the broad domains. Scoring information may also include percentile ranks, age- and grade-based equivalents, normal curve equivalents, stanines, and cumulative percentages.

## Historical Background

The first version of the WIAT was initially developed in 1992 as an assessment of academic achievement of individuals in kindergarten



through 12th grade. It was distinctive from other tests in its coverage of the areas required for a learning disability diagnosis. Only 4 years after the publication of the WIAT, the revision process began. Focus groups defined the new constructs, and then the constructs were compared to national and state standards, curricula, and academic research. The most current form of the WIAT, the WIAT-III, was introduced in 2009.

### Psychometric Properties

The WIAT-III is nationally standardized in the United States on 2,775 students from kindergarten to 12th grade.

With regard to reliability, internal-consistency reliabilities were calculated using a split-half reliability for subtests with individual items. All subtest reliability coefficients range from 83 to 97 with the exception of the alphabetic writing fluency subtest. All composite score reliability coefficients ranged from 90 to 98. Test-retest reliability was computed using Pearson's product-moment correlation using the US sample only. All subtest test-retest reliability coefficients range from 82 to 94 with the exception of the listening comprehension and sentence composition subtests. Inter-rater reliability is reported to be 98–99% for objective subtests and 91–99% for subjective subtests.

With regard to validity, final items utilized for the assessment closely align with the theoretical framework. Intercorrelational studies were conducted to assess the correlation between subtests. Intercorrelations range from 41 to 93, among the oral language, total reading, basic reading, reading comprehension and fluency, written expression, mathematics, and math fluency composites. When compared with other constructs on other Wechsler assessments, correlations were acceptable. All WIAT-III subtest correlations ranged from 41 to 76 with the full-scale IQ score on the WISC-IV.

### Clinical Uses

The WIAT-III is developed for use within the clinical and educational setting to identify an

individual's academic strengths and weaknesses, inform educational and placement decisions, support the development of interventions, and measure all eight areas of academic achievement related to learning disabilities as specified by IDEA legislation. The WIAT-III is appropriate for clinical, education, and/or research settings and may include administration in schools, clinics, private practice, and residential facilities.

The WIAT-III is classified as a level B measure. As such, individuals with training and familiarity in the administration of individual assessment instruments within the clinical and educational settings are qualified to administer the WIAT-III.

### References

- McCrimmon, A. W., & Climie, E. A. (2011). Test review. *Canadian Journal of School Psychology, 26*, 148–156.
- McCrimmon, A. W., & Smith, A. D. (2013). Test review. *Journal of Psychoeducational Assessment, 31*, 337–341.
- Wechsler, D. (2012). *Wechsler individual achievement test* (3rd ed.). San Antonio: NCS Pearson.

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### Wechsler Intelligence Scale for Children

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### Synonyms

WISC; WISC-V



## Description

The WISC-V (Wechsler 2014) is an individually administered measure of cognitive abilities developed for use with children ages 6 to 16 years. It represents the most recent revision of the Wechsler Intelligence Scale for Children and includes normative updates and factor restructuring from the WISC-IV (Wechsler 2003). The WISC-V was normed on 2,200 children ages 6 to 16 years, stratified by age, race/ethnicity, parent education, and geographic region, according to 2012 US census data. A total of 100 boys and 100 girls were included in each of 11 age levels of the standardization sample.

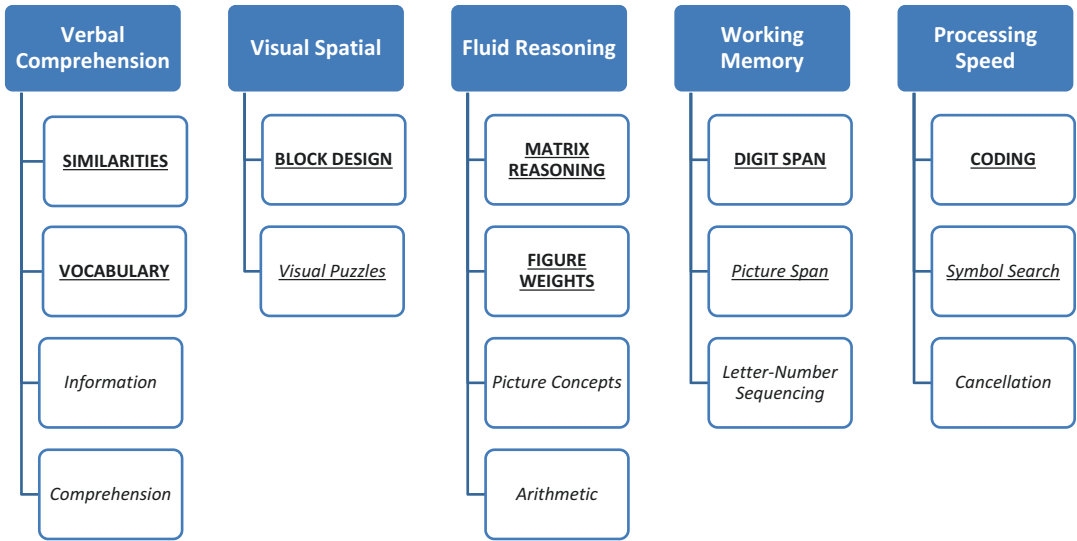
The WISC-V consists of ten primary, six secondary, and five complementary subtests, yielding five Primary Index, five Ancillary Index, and three Complementary Index scales, along with a Full-Scale IQ (FSIQ). Notably, the terms “core” and “supplemental” subtests are not used due to changes that only permit subtest substitution for the FSIQ. The WISC-V incorporates several important revisions. First, the number of subtests increased from 15 to 21. Thirteen subtests were retained from the WISC-IV, and eight new subtests were added. Word Reasoning and Picture Completion were omitted; three subtests were adapted from other measures (e.g., Visual Puzzles and Figure Weights from the WAIS-IV; Picture Span from the WPPSI-IV); and five complementary subtests were newly developed.

Second, the proposed factor structure of the WISC changed significantly. The five Primary Index Scales (i.e., Verbal Comprehension (VCI), Visual Spatial (VSI), Fluid Reasoning (FRI), Working Memory (WMI), and Processing Speed (PSI)) are each comprised of two, rather than three, subtests. The WISC-IV Perceptual Reasoning Index is replaced by VSI and FRI to delineate nonverbal skills into fluid reasoning and visual spatial abilities. Only seven of the primary subtests are included in the FSIQ (e.g., two subtests from both VCI and FRI; one subtest each from VSI, WMI, and PSI). The Ancillary Index Scales (i.e., Quantitative Reasoning

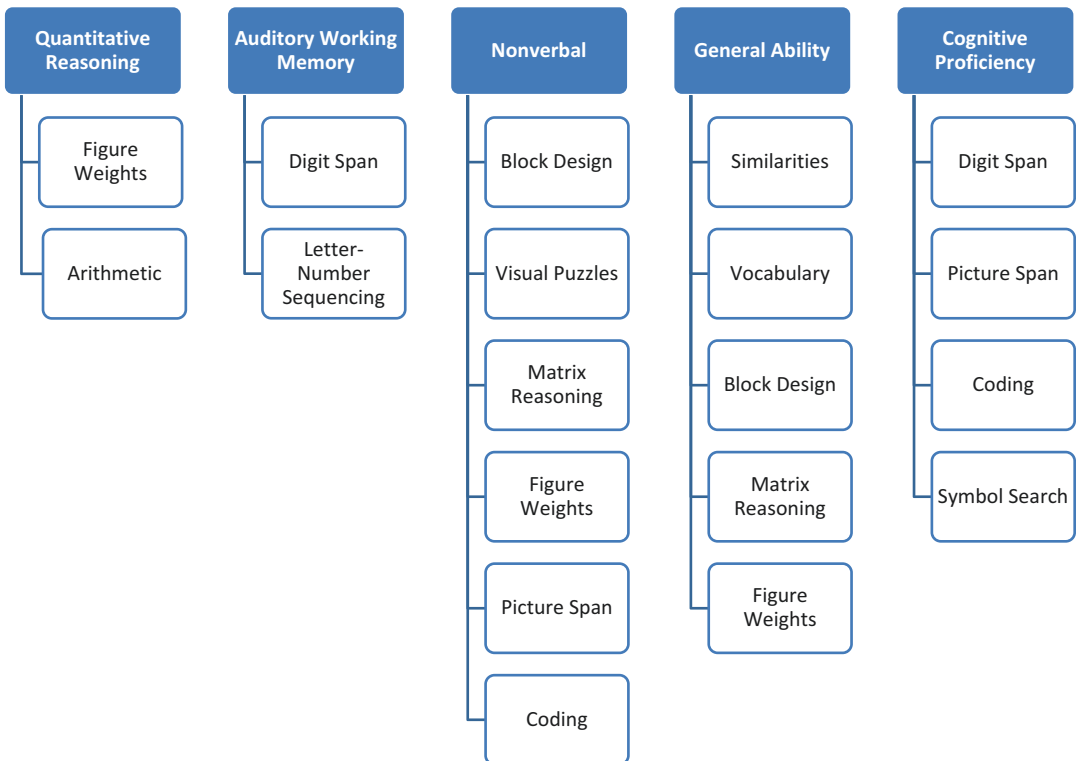
(QRI), Auditory Working Memory (AMWI), Nonverbal (NVI), General Ability (GAI), and Cognitive Reasoning (CRI)) are comprised of two to six primary or secondary subtests and were designed to provide additional details regarding cognitive abilities. The five complementary subtests provide two Complementary Index Scales (i.e., Naming Speed (NSI) and Symbol Translation (STI)), which together comprise a third scale (i.e., Storage and Retrieval (SRI)). A complete breakdown of the subtests comprising each Index and the FSIQ are included in Figs. 1, 2, and 3, adapted from information derived from the WISC-V manual (Wechsler 2014).

Third, administration and scoring procedures were revised to increase “developmental appropriateness” and “user-friendliness” (Wechsler 2014, p. 22). In general, the number of demonstration, sample, and teaching items was increased, and efforts were made to ensure that directions were concise and consistent with children’s receptive vocabulary. Furthermore, scoring criteria “for those subtests that require more elaborate responses” were changed to focus more on “response meaning rather than precise verbatim content” (Wechsler 2014, p. 29). Lastly, the emphasis on time (time bonus points) was reduced on Block Design, as it was not intended to be a measure of speed. Test developers also simplified the measure by reducing the number of subtest items, shortening discontinuous rules, reducing the number of subtests contributing to the FSIQ, and clarifying instructions.

Subtests have age-specific start points as well as reversal rules for administration of items before the start point, if needed. Raw scores are converted to age-normed subtest scaled scores ( $M = 10$ ,  $SD = 3$ ) with subtest scaled scores within each Index summed and converted to Index standard scores ( $M = 100$ ,  $SD = 15$ ) (with the exception of the Storage and Retrieval Index, which is comprised of the Naming Speed Index and Symbol Translation Index scores, not subtest scaled scores). The FSIQ score represents performance across seven primary subtests (Table 1).



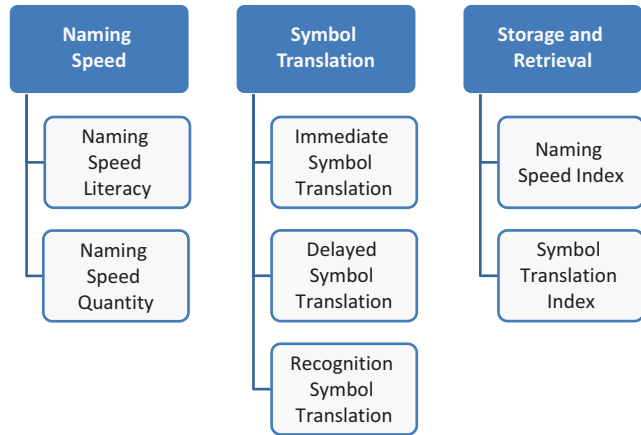
**Wechsler Intelligence Scale for Children, Fig. 1** WISC-V FSIQ – Subtests and Index Structure (bold capitalized indicates an FSIQ subtest; underlined indicates a subtest required for index calculations; and italics indicate a possible FSIQ substitution)



**Wechsler Intelligence Scale for Children, Fig. 2** WISC-V Ancillary Index Scales – Subtests and Index Structure

**Wechsler Intelligence Scale for Children,**

**Fig. 3** WISC-V  
Complementary Index  
Scales – Subtests and Index  
Structure



**Wechsler Intelligence Scale for Children,**

**Table 1** Abbreviations used for cognitive test measures

Test abbreviation	Definition
WISC-III	Wechsler Intelligence Scale for Children, Third Edition
WISC-IV	Wechsler Intelligence Scale for Children, Fourth Edition
WISC-V	Wechsler Intelligence Scale for Children, Fifth Edition
WB-I	Wechsler Bellevue (original)
WB-II	Wechsler Bellevue, Second Edition
WAIS	Wechsler Adult Intelligence Scale (original)
WAIS-IV	Wechsler Adult Intelligence Scale, Fourth Edition
WISC-R	Wechsler Intelligence Scale for Children, Revised
WPPSI-III	Wechsler Preschool and Primary Scale of Intelligence, Third Edition
WPPSI-IV	Wechsler Preschool and Primary Scale of Intelligence, Fourth Edition
DAS-II	Differential Ability Scales, Second Edition
KABC-II	Kaufman Assessment Battery for Children, Second Edition

**Historical Background**

Cognitive or “intelligence” testing began with efforts around the turn of the last century to identify children likely to have difficulty benefiting from typical classroom settings (those with some degree of intellectual disability; Binet and Simon 1905). Terman revised Binet’s test in 1916 as the

Stanford-Binet, and it became used by clinicians worldwide. With America’s involvement in World War I, Yerkes and others borrowed from Terman’s work to create measures for group testing of individual Army recruits’ cognitive ability, producing the Army Alpha (primarily verbal) and Beta (primarily nonverbal) tests (Matarazzo 1972). Group cognitive testing yielded much clinical data, which generated debate regarding the nature of intelligence, whether unitary (e.g., Spearman’s “g,” or one general intelligence factor) or fractionated (e.g., Thorndike’s multiple specific factors of intelligence).

Since Spearman and Terman, the intelligence debate has continued. The Cattell-Horn-Carroll (CHC) theory of cognitive abilities conceptualizes general intelligence (“g”) as a hierarchical structure comprising nine to ten broad ability factors, which in turn encompass about 70 narrow factors (Carroll 1998; Horn 1991), including broad elements such as crystallized knowledge and fluid reasoning, as well as more specific abilities such as reaction time or processing speed. The Luria model (Luria 1973) similarly conceptualizes discrete cognitive processes, including simultaneous and sequential processing, planning, and learning abilities, as more important for determining pattern of performance than overall “g.” Other theorists conceptualize additional components, with Gardner (1983) popularizing the view of “multiple intelligences” across more functional domains (e.g., logical-mathematical, musical).



Based upon clinical work assessing cognition for the Army and Bellevue Psychiatric Hospital, David Wechsler created the Wechsler-Bellevue Intelligence Scale (WB-I) in 1939. This measure represented significant changes to intellectual assessment, both psychometrically and clinically. Wechsler defined intelligence as one's global capacity to act purposefully, think rationally, and deal effectively with the environment (Matarazzo 1972) and acknowledged that such capacity must be malleable. As such, his measure comprised a number of different tests designed to provide an estimate of individual cognitive capacity based upon a broad sampling of domains, with an overall score of cognitive functioning ("g") as well as separate scores of verbal and performance, or nonverbal, abilities. For the first time, scores provided an estimate of an individual's relative standing as compared with same-aged peers, or a *deviation IQ* score, rather than a "mental age" estimate. This deviation score relied upon application of Gaussian statistical classification methods (e.g., "normal curve") to assess individuals' cognitive functioning.

Early intelligence measures (e.g., Binet/Simon; Terman's Stanford-Binet) provided ratio-based scores, representing the ratio of "mental age," or raw score performance, to chronological age. This ratio IQ score is problematic both psychometrically and clinically. Psychometrically, ratio-based scores do not remain constant for individuals whose skills are significantly above or below the mean. Clinically, such scores have limited meaning for defining functioning beyond the point at which "mental age" scores no longer increase with chronological age (Matarazzo 1972). The deviation-based IQ score corrects these problems, determining one's score as relative standing in comparison to same-aged peers. The deviation IQ score compares an individual's score to a predetermined mean and standard deviation and describes it in terms of the degree of deviation from the mean score for individuals of a given age. Thus, although one's raw score changes over time, relative standing is less likely to change, resulting in a constant "IQ." Wechsler introduced the deviation IQ score in the 1939 version of the Wechsler-Bellevue and continued

it into later revisions. It was then adopted by Terman in his 1960 Stanford-Binet revision and is used in most major test batteries today.

Wechsler's original Wechsler-Bellevue and its revisions (e.g., WB-II, WAIS) were subsequently modified for use with school-aged children (e.g., WISC; Wechsler 1949). The original WISC adapted 11 Wechsler-Bellevue subtests (Information, Arithmetic, Similarities, Vocabulary, Digit Span, Comprehension, Picture Completion, Picture Arrangement, Block Design, Object Assembly, and Coding) and added one subtest (Mazes) designed especially for children (Wechsler 2003). The WISC retained the verbal, performance, and Full-Scale scores from the WAIS. The first revision, the WISC-R (Wechsler 1974), expanded the age range of the WISC to the current 6 to 16 years. With the third revision of the scale (WISC-III; Wechsler 1991), an additional measure of processing speed was added (Symbol Search), and four optional Index scores were introduced. The WISC-IV dropped the Verbal IQ and Perceptual IQ but retained the four Indices (i.e., VCI, PRI, WMI, and PSI) and FSIQ. Two Indices were renamed for clarity (i.e., Perceptual Organization Index renamed to PRI and Freedom from Distractibility Index renamed to WMI). The WISC-IV also omitted three WISC-III subtests (i.e., Picture Arrangement, Object Assembly, and Mazes) and added five new subtests (i.e., Word Reasoning, Matrix Reasoning, Picture Concepts, Letter-Number Sequencing, and Cancellation) (Wechsler 2014).

## Psychometric Data

Reliability (internal consistency) of the WISC-V FSIQ and Primary Index scores is generally high, with average coefficients for all ages ranging from 0.84 (PSI) to 0.97 (FSIQ). Reliability for the Ancillary Index scores is also high, ranging from 0.91 (AVMI) to 0.97 (GAI). The composites and subtests show maintained and sometimes improved average reliability coefficients from the WISC-IV. Average reliability (i.e., split-half; test-retest for speeded tests) across individual subtests is generally good to excellent across ages, ranging from 0.81 to 0.94, but reliability is adequate, if not questionable for

individual decision-making, for some subtests within certain age groups (e.g., 0.67 for Symbol Search at ages 12 to 13).

Reliability was also calculated for the 13 special groups included in the standardization sample, including individuals who were intellectually gifted, or who had mild to moderate intellectual disability, borderline intellectual functioning, specific learning disorders (i.e., reading, mathematics, or written expression), attention-deficit/hyperactivity disorder (ADHD), disruptive behavior, traumatic brain injury (TBI), autism spectrum disorder (with and without language impairment), or who were English language learners. Average subtest reliability coefficients of the primary and secondary subtests for the groups ranged from 0.89 (Block Design) to 0.97 (Figure Weights). As with reliability in the normative sample, reliability coefficients for some subtests within certain groups were as low as 0.67 (e.g., Matrix Reasoning in the reading disorder group). As test-retest coefficients are more appropriate for measuring reliability of timed subtests (e.g., Coding, Symbol Search, Cancellation) than split-half, reliability coefficients were not reported for these subtests as test-retest data were not collected for special groups. Results suggest that the WISC-V is a generally reliable instrument for assessing both typically developing children and those with specific diagnoses.

Stability of the WISC-V subtests and composites is also generally good. Test-retest reliability was examined in a sample of 218 children over a mean interval of 26 days (range, 9 to 82 days) and suggested that scores are generally stable over time (Wechsler 2014). Mean test-retest coefficients for subtests range from 0.71 (Picture Concepts) to 0.90 (Vocabulary), with the PSI showing the largest practice gains and the VCI and FSIQ the greatest stability (0.92) over the test-retest interval.

The WISC-V can be scored reliably, even for those subtests requiring a greater degree of clinical judgment. For those such subtests (e.g., VCI subtests), inter-rater reliability coefficients (intraclass correlations) ranged from 0.97 (Similarities, Comprehension) to 0.99 (Information). Inter-scorer agreement for other subtests ranged from 0.98 to 0.99.

Evidence for validity of the WISC-V comes from its strong correlations with other, similar measures of cognitive ability (e.g., construct validity) as well as factor analytic studies of the standardization sample and various clinical groups and referred samples. Given the significant changes to subtests and composition of the indices from WISC-IV to WISC-V, the existing base of evidence for the WISC-IV is not directly transferable to the WISC-V. Although the overall FSIQ correlates highly across the two measures (0.86), using a subset of the standardization sample (242 children), subtest correlations range from 0.57 (Symbol Search) to 0.82 (Vocabulary). The corrected correlations for indices are lowest between the WISC-IV PRI and WISC-V FRI (0.63) and VSI (0.66) and between the WISC-IV and WISC-V WMI (0.65). Correlations are highest for VCI (0.85) and FSIQ (0.86).

Correlations between the WISC-V and other measures of cognitive abilities are generally high. The WISC-V FSIQ correlates 0.83 with the WPPSI-IV FSIQ. At age 16, the WISC-V FSIQ correlates 0.89 with the WAIS-IV FSIQ. The WISC-V FSIQ correlates less well (in comparison to the WISC-IV) with composite scores from the KABC-II, with correlations of 0.81 and 0.77 with the Fluid Crystallized (FCI) and Mental Processing Indices (MPI), respectively.

Test authors suggest that confirmatory factor analytic studies, examining all 16 subtests loading onto the FSIQ in the standardization sample, provide evidence supporting five factors (Wechsler 2014, p. 82). Subtest loadings across factors vary, with Arithmetic factorially complex in their preferred model. However, initial external work using standardization data (Canivez et al. 2015) found better support for a four-factor model similar to that of the WISC-IV (VCI, WMI, PSI and Perceptual Reasoning [Block Design, Visual Puzzles, Matrix Reasoning, Figure Weights]) explaining 53% of common variance in scores; a five-factor solution yielded an inadequate fifth factor. Of note, these analyses require administration of 16 subtests, which may not be consistent with anticipated patterns of general clinical usage. Taken together, however, these data suggest interpretation should focus on the primary (higher

order) factor or “g.” Although the WISC-IV factor structure was found to be robust among clinical or referred samples, there is as of yet no published data examining the factor structure of the WISC-V in these groups. Given the changes to the subtests and theoretical factor structure, data from the WISC-IV cannot be applied to the new version of the measure.

An upward drift in normative performances on intelligence tests, known as the “Flynn effect,” has been documented across various measures (Flynn 1987). Score increases in the United States have historically amounted to approximately 3 IQ points per decade. Correspondingly, over time, normative samples used to develop new measures of intelligence can represent a higher functioning group than those individuals in the normative sample for previous versions of the same test. As a result, when an IQ test is renormed, the mean is reset to 100, and children may have to become “smarter” or otherwise show improvement in performance in order to maintain a constant performance level (Kanaya et al. 2003).

Consistent with these trends, children tend to score lower on all the WISC-V index scores compared to the WISC-IV, except the PSI. Research indicates, however, the WISC-V “current gain is approximately half,” which “seems to indicate that the [Flynn effect] is slowing down” (Grégoire et al. 2016, p. 204). Furthermore, researchers caution performance comparisons between WISC-IV and WISC-V given the extensive structural changes in the updated version (Grégoire et al. 2016). Nevertheless, it is important to recognize that at the lower end of the distribution, the “Flynn effect” can significantly affect children’s eligibility and access to services. Kanaya et al. (2003) found that in examining trends over time, students with IQ scores in the borderline range “lost” an average of 5 points when retested on renormed tests and thus were more likely to be classified with intellectual disability compared with children retested on the same test. Similarly, when IQ scores decline following test revision, the likelihood of identifying a discrepancy-based learning disability is reduced, potentially resulting in delays in provision of interventions (Truscott and Frank 2001).

## Clinical Uses

Although intelligence tests were originally developed to predict children’s ability to benefit from instruction, they are commonly used to assess cognitive dysfunction among individuals with central nervous system disorders, including neurodevelopmental disabilities (Groth-Marnat et al. 2000). Some prominent neuropsychologists have advocated abandoning intelligence tests for the purpose of understanding patterns of cognitive dysfunction (e.g., Lezak 1988, 2004), yet current cognitive measures, such as the WISC-V, go beyond providing an estimate of “g” to render detailed information regarding specific cognitive skills and processing abilities and continue to yield important information about patterns of cognitive performance. The WISC-V also expands upon previous process score analyses and provides ancillary subtests and index scores. Although more empirically supported research regarding these scores is needed, the updated structure and decreased emphasis on speeded performance may provide more detailed characterization of functioning within a wide variety of clinical diagnoses across cognitive domains.

Assessing children with intellectual disability is particularly challenging; however, since diagnosis of intellectual disability relies on both estimates of intelligence and adaptive functioning, formal intellectual assessment using a standardized IQ test should be conducted whenever possible. Obtaining an accurate assessment of overall intellectual ability to classify an individual as having an intellectual disability is important when predicting supports required throughout the lifetime (Sattler 2001). Notably, classification of intellectual disability severity is no longer based upon IQ score ranges but, instead, allows for more clinical judgment in determining the nature and extent of an individual’s intellectual and adaptive difficulties (American Psychiatric Association 2013). As such, with the new factor structure, additional subtests, and optional index score calculations, the WISC-V can be a useful measure in combination with other data (e.g., adaptive functioning ratings), in identifying intellectual disability.



Within other referred samples (e.g., traumatic brain injury, ADHD), research on WISC-III and WISC-IV indicated that specific index scores often discriminated between referred children and controls (Calhoun and Mayes, 2005; Donders and Janke 2008). Previous research also indicated that in children with ADHD, autism, bipolar disorder, and learning disabilities, processing speed and working memory deficits were common (e.g., Calhoun and Mayes 2005), with these children scoring lower on PSI and Freedom from Distractibility (FDI, now WMI) indices. Data from WISC-V indicated that, although children in the ADHD special group obtained average mean primary index scores, their lowest scores were on the PSI. Similarly, children with TBI also scored lowest on the PSI. These findings are consistent with previous research indicating the *sensitivity* of the PSI to neurological disorders. However, with the updated factor structure and fewer subtests contributing to each primary index and overall FSIQ, additional research is needed to examine overall WISC-V performance of special groups in comparison to WISC-IV.

For children with specific academic difficulties (e.g., learning disabilities), cognitive testing provides an important component, especially for those who do not respond to more intensive instruction. Academic difficulties can represent a final common pathway of a combination of neurobiologic, behavioral, environmental, and cognitive factors, with an understanding of the core cognitive processes involved helpful in clarifying specific mechanisms and better understanding the disorder (Fletcher 2009). Although more research regarding the WISC-V is needed, with more distinct primary factors and additional ancillary subtests and factors, this measure may help clarify our understanding of the underlying cognitive mechanisms involved in learning disorders and better target our interventions.

## See Also

- ▶ [Intelligence](#)
- ▶ [Normal Curve](#)
- ▶ [Test Reliability](#)

## References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association. <https://doi.org/10.1037/t19590-000>.
- Binet, A. & Simon, S. (1905). Méthodes nouvelles pour le diagnostic du niveau intellectuel des anormaux. *L'Année psychologique*, *11*, 191–336.
- Calhoun, S. L., & Mayes, S. D. (2005). Processing speed in children with clinical disorders. *Psychology in the Schools*, *42*(4), 333–343. <https://doi.org/10.1002/pits.20067>.
- Canivez, G. L., Watkins, M. W., & Dombrowski, S. C. (2015). Factor structure of the Wechsler intelligence scale for children-fifth edition: Exploratory factor analyses with the 16 primary and secondary subtests. *Psychological Assessment*. <https://doi.org/10.1037/pas0000238>.
- Carroll, J. B. (1998). Human cognitive abilities: A critique. In J. J. McArdle & R. W. Woodcock (Eds.), *Human cognitive abilities in theory and practice* (pp. 5–24). Mahwah: Lawrence Erlbaum Associates.
- Donders, J., & Janke, K. (2008). Criterion validity of the Wechsler intelligence scale for children-fourth edition after pediatric traumatic brain injury. *Journal of the International Neuropsychological Society*, *14*(4), 651–655. <https://doi.org/10.1017/S1355617708080752>.
- Fletcher, J. (2009). Neuropsychology of learning disabilities: An interdisciplinary perspective. Presidential address at the meeting of the International Neuropsychological Society, Atlanta.
- Flynn, J. R. (1987). Massive IQ gains in 14 nations: What IQ tests really measure. *Psychological Bulletin*, *101*, 171–191. <https://doi.org/10.1037/0033-2909.101.2.171>.
- Gardner, H. (1983). *Frames of mind: The theory of multiple intelligences*. New York: Basic Books.
- Grégoire, J., Daniel, M., Llorente, A.M., Weiss, L. C. (2016). The Flynn effect and its clinical implications. In L. G. Weiss, D. H. Saklofske, J.A. Holdnack, A. Prifitera, L.G. Weiss, D. H. Saklofske, . . . A. Prifitera (Eds.), *WISC-V assessment and interpretation: Scientist-practitioner perspectives* (pp. 187–212). San Diego: Elsevier Academic Press. <https://doi.org/10.1016/B978-0-12-404697-9.00006-6>.
- Groth-Marnat, G., Gallagher, R. E., Hale, J. B., & Kaplan, E. (2000). The Wechsler intelligence scales. In G. Groth-Marnat (Ed.), *Neuropsychological assessment in clinical practice: A guide to test interpretation and integration* (pp. 129–194). New York: Wiley.
- Horn, J. L. (1991). Measurement of intellectual capabilities: A review of theory. In K. McGrew, J. K. Werder, & R. W. Woodcock (Eds.), *WJ-R technical manual* (pp. 197–232). Itasca: Riverside Publishing.
- Kanaya, T., Scullin, M. H., & Ceci, S. J. (2003). The Flynn effect and U.S. policies: The impact of rising IQ scores on American society via mental retardation diagnoses. *American Psychologist*, *58*(10), 778–790. <https://doi.org/10.1037/0003-066X.58.10.778>.

- Lezak, M. D. (1988). IQ: R.I.P. *Journal of Clinical and Experimental Neuropsychology*, 10(3), 351–361. <https://doi.org/10.1080/01688638808400871>.
- Lezak, M. D. (2004). Theory and practice of neuropsychological assessment: Basic concepts. In M.D. Lezak, D. B. Howieson, & D. W. Loring (Eds.), *Neuropsychological assessment* (4th ed., pp. 15–38). New York: Oxford University Press.
- Luria, A. (1973). *The working brain: An introduction to neuropsychology*. Harmondsworth: Penguin.
- Matarazzo, J. D. (1972). *Wechsler's measurement and appraisal of adult intelligence*. New York: Oxford University Press.
- Sattler, J. M. (Ed.). (2001). *Assessment of children: Cognitive applications* (4th ed.). San Diego: J.M. Sattler.
- Truscott, S. D., & Frank, A. J. (2001). Does the Flynn effect affect IQ scores of students classified as LD? *Journal of School Psychology*, 39(4), 319–334. [https://doi.org/10.1016/S0022-4405\(01\)00071-1](https://doi.org/10.1016/S0022-4405(01)00071-1).
- Wechsler, D. (1949). *Wechsler intelligence scale for children*. New York: The Psychological Corporation.
- Wechsler, D. (1991). *Wechsler intelligence scale for children* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2003). *Wechsler intelligence scale for children* (4th ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2014). *Wechsler intelligence scale for children* (5th ed.). Bloomington: PsychCorp.
- Wechsler, D. (1974). *WISC-R manual*. New York: The Psychological Corporation.

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## Wechsler Intelligence Tests for Adults and Children

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### Synonyms

WAIS; WAIS-III; WAIS-IV; WAIS-R; WB; WISC-IV; WISC-V; WPPSI-III; WPPSI-IV

### Description

The Wechsler scales for assessing the intelligence of adults (Wechsler Adult Intelligence Scale (WAIS)), school-age children (Wechsler Intelligence Scale for Children (WISC)), and preschool and young children (Wechsler Preschool and Primary Scale of Intelligence (WPPSI)) have a long and distinguished history. Though there have been significant revisions in the last few decades, the Wechsler scales are the most often used tests of intelligence and have been adapted, translated, and standardized for use in many countries. The most recent revision has been to the WISC, which is now in the fifth edition (WISC-V, 2014). The very first test published by Dr. David Wechsler in 1939 was the Wechsler-Bellevue Intelligence Scale (WB). This introduced several major changes to the field of intellectual assessment that had been dominated by the well-established Stanford-Binet test, first published at the turn of the twentieth century in France. The most significant change was the splitting of the Full-Scale Intelligence Quotient (FSIQ) to yield a Verbal (VIQ) and Performance (PIQ) intelligence quotient. Progressing from the original WB are the now well-known Wechsler intelligence scales noted above as well as other tests that carry the Wechsler name, for example, the Wechsler Individual Achievement Test, the Wechsler Nonverbal Scale of Ability, the Wechsler Abbreviated Scale of Intelligence, and the Wechsler Memory Scales, now in its fourth edition. Although Dr. Wechsler passed away in 1981, his legacy continues in the many intelligence, memory, and achievement tests that carry his name.

### Historical Background

#### Early Definition of Intelligence Reflected in the Wechsler Tests

All the current Wechsler intelligence tests have their foundations in the WB, which was intended to assess the intelligence of persons aged 7–69 years. Wechsler's original view of intelligence holds to this day:

Intelligence is the aggregate or global capacity of the individual to act purposefully, to think rationally, and to deal effectively with his environment. (Wechsler 1944, p. 3)

Following the decision to concentrate the assessment of adult intelligence into a separate test, the WAIS (1955), WAIS-R (1981), and WAIS-III (1997) were published by the Psychological Corporation (now Pearson Assessment) and quickly embraced by both research and practicing psychologists as reliable and clinically useful assessment instruments. The foundational model and primary interpretive scores for the WAIS and WAIS-R were the FSIQ, VIQ, and PIQ scores. The VIQ was considered a good measure of crystallized intelligence (Gc) (see also “► [Verbal IQ](#)” and “► [Intelligence](#)” headings in this volume). Thus, VIQ represents a summary of acquired knowledge, language use, and verbal concept formation acquired from the family, school, and other learning environments. PIQ was intended more as a measure of fluid intelligence (Gf), assessed by subtests tapping nonverbal reasoning, spatial perception, and abstract problem solving. VIQ and PIQ share variance and have been combined in the Wechsler intelligence tests to produce a Full-Scale IQ (FSIQ) score. The FSIQ serves as a measure of general mental ability, or “g,” first described by Charles Spearman and later by Cyril Burt and Philip E. Vernon (see also “► [Intelligence Quotient](#)” and “► [Intelligence](#)” in this volume).

### **The Changing Purposes of Assessing Intelligence**

While IQ scores obtained from the earlier Wechsler tests mainly aided classification and placement decisions, the VIQ and PIQ score discrepancies were also deemed useful for some diagnostic purposes of the day (e.g., diagnosing borderline to mild mental retardation based on a FSIQ of 74 when, for example, VIQ was 74 and PIQ was 71, in comparison with low measured intelligence due to limited educational opportunities or a hearing impairment based also on an FSIQ of 74 but comprising a VIQ of 61 and a PIQ of 92).

In contrast to these earlier limited uses of intelligence tests, psychologists have increasingly emphasized the importance of clinical assessment to provide a more comprehensive description of individual cognitive differences and to apply this information to improve diagnoses and treatment planning. Thus, interpretation has moved beyond reporting and evaluating differences between VIQ and PIQ or using the FSIQ for placement decisions. Psychologists have increasingly looked to glean more information from the WAIS tests, such as examining subtest scores to better understand the cognitive strengths and weaknesses of individuals. This encouraged an exploration of both interindividual and intraindividual comparisons of test scores. Concurrent with the increased clinical use of intelligence tests have been advances in intelligence theory and research and a growing connection between cognitive psychology and neuroscience. In addition, there have been psychometric advances, allowing for improved measurement development.

These important advances in our understanding of the structure, causes, and correlates of intelligence and how this contributes to a better understanding of cognitive abilities and individual differences are reflected in the current Wechsler scales and their use in clinical assessment contexts. The most recent generation of the Wechsler tests has moved away from a reliance on FSIQ and the VIQ-PIQ description to better reflect the composition of “g” and also assess major cognitive abilities such as working memory and processing speed.

## **Wechsler Adult Intelligence Scales**

### **WAIS-IV**

#### **Overview**

The WAIS-IV (Wechsler 2008) was published in the latter part of 2008 and was joined by the Wechsler Memory Scale – fourth edition (WMS-IV) in 2009. The goals for revision of the WAIS-III included updating the Wechsler Adult Intelligence Scales to (a) integrate findings from the ongoing research of intelligence theory, cognitive

neuroscience, and aging, (b) improve the developmental appropriateness of test and test items, (c) increase the ease of use of the test, (d) improve clinical utility, and (e) further improve the psychometric properties of the test (Tulsky et al. 2003).

The VIQ-PIQ level of analysis was dropped and the focus of interpretation now rests with the four-factor index scores. Other key changes in the WAIS-IV include the introduction of several new subtests and renaming the POI as the Perceptual Reasoning Index (PRI). The renaming of POI to PRI was warranted with the addition of a new subtest, called visual puzzles, which increased the load of fluid intelligence and perceptual reasoning. The test can be used with examinees in the 16–90 year age range. The FSIQ range of the WAIS-IV has been extended from the WAIS-III and now ranges from 40 to 160. As in previous versions, the mean IQ and index score is 100 with a standard deviation of 15. Subtest scores have a mean of 10 and a standard deviation of 3. Extending work from the WAIS-III, other combinations of Wechsler subtest scores are included as part of the manual. These additional indexes include the General Ability Index (GAI) comprising the VCI and PRI that serves as a measure of general intelligence, like FSIQ, but where the variance in measured intelligence attributable to processing speed and psychomotor ability is minimized.

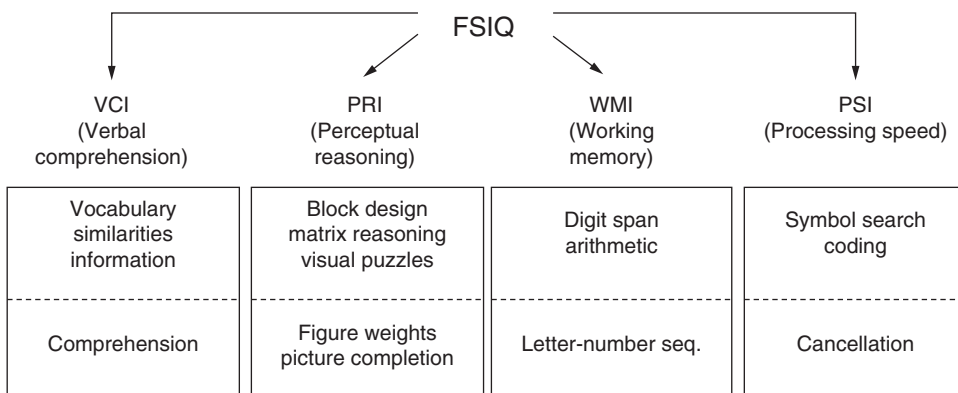
The structure and composition of the WAIS-IV is shown in Fig. 1. The ten subtests in the upper part of the boxes are required to obtain the four

index scores and FSIQ; those in the lower part are supplemental subtests that can serve as substitutes if needed or be administered to gather additional information about an individual’s cognitive functioning.

**Psychometric Data**

The WAIS-IV provides contemporary norms based on US population statistics (the Canadian standardization version was also published in the 2008). Clinical appeal has also been improved in a variety of ways ranging from shorter administration times, changes in test items, and broader basal and ceiling items. Evidence for clinical utility is provided in the test manual describing the performance of special group studies, including clinical groups diagnosed with intellectual disability, traumatic brain injury, Asperger’s disorder, dementia, and depression.

Initial evaluation confirms that the WAIS-IV has again improved on the assessment of intelligence with high reliability and improved measurement of four factors and FSIQ. All index reliability coefficients are in the 0.9 range with FSIQ at 0.98. These reliabilities hold for the total sample and are essentially found for all age groups. While slightly lower, reliabilities continue to be very acceptable for all the clinical groups ranging from the gifted and intellectual disability sample to the TBI and Alzheimer’s clinical samples. Stability coefficients vary from good to excellent with test-retest score changes reported in various tables. Confirmatory factor analysis



**Wechsler Intelligence Tests for Adults and Children, Fig. 1** The WAIS-IV model

provides compelling support for the four index scores, and second-order FSIQ correlations with other relevant tests from intelligence (WAIS-III) and executive functioning (D-KEFS) to memory (CMS, WMS-III) and achievement (WIAT-II) argue for the criterion validity of the WAIS-IV. Clinical studies comparing special groups with matched controls as well as base rate data that further permit the comparisons of score discrepancies add to the clinical usefulness of the WAIS-IV for both diagnosis and intervention planning.

### Clinical Uses

It is important to note the WAIS-IV should not be construed to measure all domains of intelligence described in, for example, CHC theory (Cattell-Horn-Carroll theory of cognitive abilities) or the Das-Naglieri PASS model (planning, attention, simultaneous and sequential processing). Wechsler argued that cognitive functioning comprises a portion of intelligence, but other attributes such as planning, goal awareness, impulsivity, anxiety, and persistence also account for the variability observed in human behavior. Thus, the WAIS-IV provides a reliable and valid measure of cognitive functioning, but no index score or the FSIQ should be viewed as a complete and exhaustive assessment of a person's "intelligence." Rather, the WAIS-IV scores reflect the quantitative measurement of a subset of cognitive abilities, which account for some (but not all) variance in functions comprising an individual's intelligence (see also headings "► [Intelligence Quotient](#)" and "► [Intelligence](#)" in this volume).

An additional caveat is that WAIS-IV should not be interpreted in isolation, but is most meaningful in conjunction with other data, including a detailed history of the individual and clinical observations. Additional quantitative and/or qualitative measures are routinely used in conjunction with the WAIS-IV, including data from achievement tests and/or other psychological and neuropsychological measures. The choice of additional data to be used with the WAIS-IV will vary depending upon the referral question(s).

With these caveats in mind, WAIS-IV performance can be interpreted in a number of ways. An examinee's scores can be compared with those

obtained by similar persons (standardized normative data) reflecting interindividual profile analysis, as well as from an intraindividual profile analysis. In addition to profile analysis evaluated from a statistical standpoint, profile analysis can be made using base rate score information, providing an indication of how common or frequent particular score patterns are observed in the general population. A process level analysis is also possible with the WAIS-IV based on calculating various process scores that are provided in the WAIS-IV manual. Using standard score units, a person's performance on any of the index scores and FSIQ can then be seen as falling above, at, or below "average." In addition to standard scores developed for specific age cohorts, demographically adjusted normative data (e.g., adjusted for age, education) is also available for the WAIS-IV.

As noted above, the WAIS-IV allows for analyses of index and subtest score differences based on statistical significant levels and actuarial base rate data from normative samples. Normative comparisons allow an individual's scores to be compared with similar others from the general population (both age cohorts and age and education-matched cohorts) and are derived from the reliability of index and subtest scores. Intraindividual comparisons of differences based on statistical properties permit the determination of individual strengths and weaknesses. For example, an intellectually gifted person who has obtained VCI, PRI, and WMI scores in the 130–140 range but obtains a score of 108 on PSI may be said to have a relative weakness in processing speed even though this score is in the average range. When the difference between two scores is statistically significant (e.g.,  $p < 0.05$ ), this difference can be said to occur by a chance of less than 5 times out of 100 and is a true difference rather than variability due to measurement error and/or chance. Examination of base rates, however, provides an actuarial account of the frequency in which a difference between scores was observed in the standardization sample. Thus, a difference between VCI and PRI scores may be statistically significant ( $p < 0.05$ ), but may be observed in 25–35% of the population, suggesting that this difference is a "normal



variant” of intellectual functioning. Alternatively, a statistically significant difference that also occurs in only 3–4% of the healthy population would suggest that the difference is rare and most likely to have some manifestation in areas related to intellectual functioning and may have relevance to differential diagnosis and/or treatment planning.

The use and interpretation of the WAIS-IV depends upon the referral source/question(s). Administration of the WAIS-IV (and WAIS-III) includes a broad spectrum of psychological and neuropsychological referral questions. Broadly, a Wechsler intelligence test may be administered as part of an assessment to assist in identifying learning/cognitive problems and/or the presence of brain dysfunction.

Central to assessment is the concept of deficit measurement. The identification of a deficit requires a comparison of scores against a measurement standard. The clinical interpretation of the WAIS-IV generally involves comparing the obtained WAIS-IV scores against some comparison standard. Comparison standards may be species-specific (all healthy members of a species can perform this), interindividual comparisons (comparing performance to some healthy normative score), and/or intraindividual comparisons (comparing individual performance against previous performance or expectations). In general, interpretation of Wechsler intelligence tests is based on normative and/or intraindividual level-based comparisons.

#### Psychological/Learning Disability Evaluation

The WAIS-IV is designed to assist in psychological and learning disorder evaluations. A commonly used analysis in deficit measurement for the diagnosis of learning disorders is the ability-achievement discrepancy. The ability-achievement discrepancy analysis compares the obtained intelligence test score against a measure of achievement, such as the Wechsler Individual Achievement Test – third edition (WIAT-III) or other individual achievement test. This difference can be calculated by using either the “simple” difference method or the predicted-difference method. In the “simple” difference method, the

obtained FSIQ is compared with the obtained score on the achievement test ( $IQ > \text{achievement}$  for learning disorder diagnosis), and the difference is evaluated against various traditional or preset cutoff values. Numerous psychometric drawbacks arise with the “simple” difference method, and the predicted-difference method is preferred to evaluate for differences between scores as this method takes into account both the reliability and correlation between test scores.

There are arguments for and against ability-achievement discrepancy models, and an update to the Individuals with Disabilities Education Act (IDEA, 2004) specifies an alternative multi-tier model, termed the response to intervention (RtI) model, as the preferred model. The RtI model utilizes a behavioral observation model with behavioral instruction and progress monitoring being key rather than reliance on data from intelligence tests. A third model has also been proposed for identifying children with learning problems combining RtI with cognitive/neuropsychological assessment. This third model incorporates findings from learning disordered children having brain dysfunction as opposed to previous models suggesting that these children’s academic development was “slow.”

#### Neuropsychological Evaluation

Neuropsychological assessments can (a) diagnose cognitive disorders and identify brain dysfunction, (b) track changes in cognitive function over time, and (c) identify cognitive strengths and weaknesses important for daily functioning. The WAIS-IV can be used as a reliable and valid measure of various cognitive functions. The overall FSIQ score has less interpretive value than the factorially derived index scores. Individual subtest performances can be evaluated to assess cognitive strengths and weaknesses, which, in combination with other assessments, can assist diagnosis and detecting the presence or absence of brain dysfunction. Neuropsychological assessment focuses less on statistical differences among index scores and/or subtests of the WAIS-IV and more on base rate and process level of analyses. Again, the interpretation of the WAIS-IV subtests and/or index scores should not be conducted in



isolation, and comparisons to measures of memory and other neuropsychological domains are standard.

Within this intraindividual comparison perspective, a comparison standard of expected performance must be developed. While various methods have been developed to provide a comparison standard, it is often not possible to obtain an individual's previous performance on a standardized intelligence test (i.e., obtain historical data). Methods to estimate premorbid abilities have been developed that incorporate demographic variables, archival achievement measures, current performance on "hold" tests, abilities thought to be resistant to effects of brain injury and aging, and methods combining demographics and current "hold" tests.

After arriving at a predicted premorbid level of function, the obtained performances on the WAIS-IV (and other neuropsychological measures) may be compared against this reference. Rather than employing a "simple difference" approach, the clinician must incorporate performances inclusive of the individual's history and clinical observations, knowledge of brain-behavior relationships and functional neuroanatomy, as well as psychometric theory and base rate information. Recent data highlight natural variability in neuropsychological test scores among healthy individuals, such that "low" scores (below the 16th percentile) on several cognitive test scores do not, necessarily, suggest brain dysfunction and may be commonly found as "natural variants."

## Summary

In summary, the Wechsler adult intelligence tests have provided reliable and valid measures of cognitive functions comprising intelligence since the mid-1950s. The latest version of the Wechsler Adult Intelligence Scale, the WAIS-IV, continues this tradition. The WAIS-IV has incorporated research advances in intelligence theory, measurement, cognitive neuroscience, and aging with the inclusion of additional subtests and the calculation of factorially derived index scores that more closely assess specific domains of intelligence.

The WAIS-IV has deleted the calculation of the VIQ and PIQ index scores in favor of four-factor index scores termed Verbal Comprehension, Perceptual Reasoning, Working Memory, and the Processing Speed but continues to retain the Full-Scale IQ (FSIQ). More recent studies have suggested that a five-factor structure may also be used in the clinical interpretation of the WAIS-IV (Weiss et al. 2013), and this now defines the recently published WISC-V. The WAIS-IV will continue to evolve with future publications within the next several years describing change scores, corrections for demographic differences, and premorbid prediction algorithms together with links to the WMS-IV and other Wechsler tests.

## Wechsler Intelligence Scale for Children

### WISC-IV

The WISC-IV (Wechsler 2003), an updated and improved version of the WISC-III, measures general intelligence, specifically in the areas of verbal comprehension, perceptual reasoning, working memory, and processing speed for children aged 6–16 years. Changes were implemented into the WISC-IV that caused major restructuring and reconceptualization of previous forms. The WISC-IV consists of ten core subtests and five additional subtests that yield four reliable indexes (the Verbal Comprehension Index (VCI), the Perceptual Reasoning Index (PRI), the Working Memory Index (WMI), and the Processing Speed Index (PSI) and one Full-Scale IQ (FSIQ)). The indices allow for more reliable and accurate assessment of word reasoning and verbal concept formation (VCI), nonverbal and fluid reasoning (PCI), working memory (WMI), and speed of processing information (PSI). The new subtests are based on current neurological models of cognitive functioning and allow for improved clinical utility in comparison to the WISC-III. With an updated norm sample, five new subtests (Word Reasoning, Matrix Reasoning, Picture Concepts, Letter-Number Sequencing, and Cancellation), updated artwork, removal PIQ and VIQ indexes, and increased developmental appropriateness, the WISC-IV has provided extensive insight to

children's cognitive functioning. The WISC-IV has demonstrated exceptional psychometric properties that have been used for over a decade; however, a better understanding of cognitive and intellectual functioning, changing demographics and norms, and advances in neurocognitive processes has led to the evolution of the WISC-IV.

## WISC-V

### Overview

The WISC-V (Wechsler 2014) is the most recent version of the WISC to date. With an evolution of the Wechsler model of intelligence came the emergence of the WISC-V with changes that have expanded the test framework. The benefits of revising the WISC-IV include (a) producing five primary index scores efficiently; (b) increasing test coverage, content, and user-friendliness without increasing testing time; (c) providing measures to support flexible evaluation of learning strengths and disabilities; (d) improving clinical utility and clarity in understanding children's cognitive abilities; and (e) enhancing psychometric properties for improved reliability. While the third and fourth editions of the WISC have a distinguished history and have been well-researched and established for decades, recent research on neurocognitive processes, neuropsychology, and intelligence has paved way for the evolution of the Wechsler scales, which has expanded from a two- to a five-factor theoretical model (Weiss et al. 2016). The WISC-V, which now is available in digital Q-interactive format, has an updated organization and structure that distinguishes it significantly from its predecessors. The FSIQ is an essential component of the WISC as it is one of the most robust and comprehensive measures for determining cognitive ability and intelligence. The FSIQ now comprises seven of the ten primary subtests included in the WISC-V. Other major changes in the WISC-V include the introduction of five primary index scales: Verbal Comprehension Index (VCI), Visual Spatial Index (VSI), Working Memory Index (WMI), Fluid Reasoning Index (FRI), and Processing Speed Index (PSI). Each index score is measured using two primary subtests, totaling to

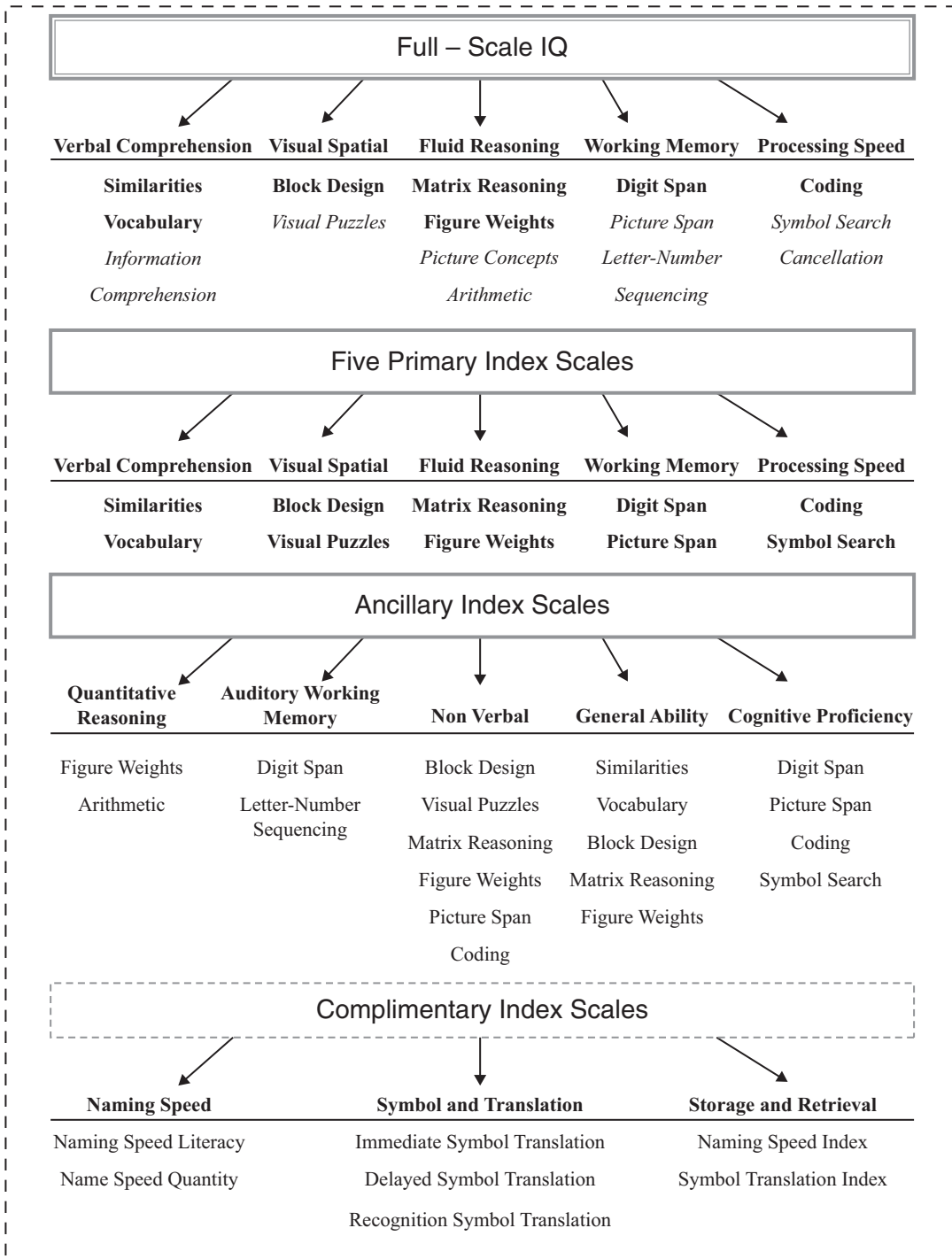
ten primary subtests. The removal of Word Reasoning and Picture Completion subtests coupled with the addition of three new primary subtests (Visual Puzzles, Figure Weights, and Picture Span) extends the content of the WISC-V to better assess children's performance.

In addition to the primary subtests, most domains include secondary clinically improved subtests that provide more information on children's cognitive abilities, strengths, disabilities, and other clinical conditions. The WISC-V is also unique in that it provides five ancillary indexes: Quantitative Reasoning Index (QRI), Auditory Working Memory Index (AWMI), Nonverbal Index (NVI), General Ability Index (GAI), and Cognitive Proficiency Index (CPI). While these subtests are optional, they give the clinician the option to explore a child's WISC-V scores in real-world settings such as the classroom or in daily functions. Finally, complementary indexes and subtests have been added to the WISC-V. While they are not part of any specific domain measure, these tests allow clinicians and test administrators to obtain more comprehensive information for children with learning disorders.

Figure 2 shows a breakdown of the structure and components of the WISC-V. The diagram is divided by FSIQ, five primary index scores, five ancillary index scores, and three complementary index scores.

### Psychometric Properties

The WISC-V provides an updated norm sample standardized on 2,200 children from the ages of 6–16 years, 11 months that is based on current US population census statistics. A Canadian standardization study has also been carried out and is being followed in other developed countries. Clinical efficacy of the test has also been enhanced in several capacities including access to more subtests, expansion of test content, adequate floor and ceiling levels, and shorter administration time. Evidence for clinical utility is provided using special group and validity studies that help identify intellectual and learning disabilities, giftedness, cognitive strengths and weaknesses, and impact of brain injuries. Reliability estimates confirm that the WISC-V has improved reliability on subtests



**Wechsler Intelligence Tests for Adults and Children, Fig. 2** The Wechsler Intelligence Scale for Children – fifth model (WISC-V)

and composite scores that will yield more accurate assessments of intelligence in children. The overall average reliability coefficients for the expanded index scores are excellent (0.95). These reliabilities were higher than those obtained for the subtest and process scores because only two subtests contribute to the primary index scores and four subtests contribute to the expanded index score. All internal consistency estimates ranged from 0.81 to 0.94, while the process scores were between 0.80 and 0.88. Test-retest stability estimates for the WISC-V scores were administered to a sample of 218 children. The WISC-V has moderate to high correlations between similar composite scores of other measures of intelligence including WISC-IV, WPPSI-IV, WAIS-IV, WIAT-III, KTEA-3, Vineland-II, and BASC-2. The highest correlations were observed between the WISC-V FSIQ and the WISC-IV FSIQ, WPPSI-IV FSIQ, WAIS-IV FSIQ, and the Kaufman Assessment Battery for Children (KABC)-II FSIQ. The WISC-V has a number of clinical studies which support the interpretation of the subtests. Studies on the Q-interactive digital format extend the clinical efficiency of the WISC-V, and clinical studies on special groups demonstrate the sensitivity of the WISC-V to cognitive deficits in children. Although there are many developmental and progressive changes accounted for in the WISC-V, more studies will follow that are expected to strengthen its validity for clinical interpretation.

#### Clinical Use and Implications

The WISC-V provides a reliable and valid measure of cognitive functioning for children, but index scores or the FSIQ should not be viewed in isolation when assessing a child's intelligence. For example, the assessment of children suspected of having cognitive impairment should also be accompanied by an assessment of adaptive behavior and developmental history. The WISC-V converts the sums of scaled scores to composite scores that allow for score comparisons at the index and subtest levels.

The WISC-V provides the most meaningful and efficient data when used in conjunction with

other factors such as clinical history, clinical observation, and other test measures. Although the WISC-V does provide additional subtest tests for the clinician to utilize for additional information, data from other neuropsychological measures should not be ruled out. Together with other key tests assessing memory, achievement, and other assessment methods such as observation, development history, and interviews, the WISC-V plays a key role in the assessment of most all clinical conditions where the need for information about intelligence and cognitive functioning is key to diagnosis, prescription, and even prognosis. Two examples follow.

#### (a) *Neuropsychological Evaluation*

A holistic neuropsychological evaluation is recommended for children to determine their patterns of cognitive strengths and weaknesses, to make a diagnosis, and to provide recommendations for effective interventions. Intelligence tests are one of the most well-known and utilized forms of neuropsychological evaluations, though it is necessary to consider other factors before making a diagnosis. The WISC-V is both a reliable and valid measure of children's cognitive abilities, especially when unusual inconsistencies are observed across subtest or index scores. Interpretation of the WISC-V scores should be conducted in comparison to other neuropsychological tests and measures. Rather than identifying discrepancies between individual tests, a clinician must be able to incorporate the child's medical and social history, clinical observations, base rate information, knowledge of theories, brain-behavior relationships, and neuroanatomy before making conclusions. Children's neuropsychological test scores may differ based on external factors such as testing conditions, setting, child's mood, and other factors such as medical/neurological conditions, emotional disorders, or disability.

#### (b) *Learning Disability Evaluation*

The WISC-V has been designed to measure cognitive abilities that can lead to the assessment

and identification of learning disabilities in children. Two chapters on the assessment of learning disabilities and dyslexia are presented in a recently published WISC-V book (Weiss et al. 2016). The WISC-V utilizes two analyses along with the KTEA-3 and WIAT-III to help identify these conditions: the ability-achievement discrepancy analysis (see WAIS-IV section) and the pattern of strength and weakness (PSW) discrepancy analysis. To recap, the ability-achievement discrepancy analysis compares the obtained intelligence test score against a measure of achievement or other individual achievement test. This difference can be calculated by using either the “simple” difference method or the predicted-difference method.

The PSW model identifies learning disabilities by looking at specific cognitive areas, rather than single IQ scores that include memory, auditory processing, processing speed, and phonemic awareness. To determine a child’s weakness, PSW associates academic performance and disabilities with specific cognitive areas. It is critical to rule out processing weaknesses in children as it helps differentiate between a child with a special learning disability and a child who is underachieving for other reasons. The PSW score analysis can be calculated using two score comparisons: the processing strength vs. achievement weakness and the processing strength vs. processing weakness. Both scores must be significantly different to fit the criteria for special learning disabilities. The PSW model is used to promote reliable identification, diagnosis, and intervention planning for children and has been useful in distinguishing each child’s unique pattern of strengths and weaknesses.

The PSW and ability-achievement discrepancy models serve the same purpose of aiding clinicians in generating hypothesis for children’s conditions. These models are to be used in combination with all other information obtained from a child’s history and behavior and by observation.

## Summary

The children’s versions of the Wechsler intelligence tests have provided a psychometrically sound and clinically useful assessment of cognitive functions and intelligence in children since 1949. The test has undergone several major updates and revisions in the last few decades that have reshaped the structure, content, and understanding of intelligence tests. The WISC-V is the most current version of the WISC, and it has incorporated research advances in structural intelligence theories, working memory models, cognitive neuroscience, and neurodevelopmental research with the inclusion of additional subtests and primary index scores that encompass more domains and a wide range of cognitive areas. The most evolutionary change is the update of the WISC from a two- to the current five-factor-based model that now includes Verbal Comprehension, Visual Spatial Index, Fluid Reasoning, Working Memory, and Processing Speed. The FSIQ, which is still an important measure of general cognitive ability, has also coevolved to utilize seven on the ten primary subtests. The WISC will continue to evolve to coincide with updated research studies, new scientific findings, and an ever-changing population. The purpose of the WISC-V is to aid clinicians in the process of generating clinical hypothesis, conducting neuropsychological assessments, and identifying strengths and problem areas in children and adolescents.

## Cross-References

- ▶ [Cognitive Functioning](#)
- ▶ [Cognitive Processing](#)
- ▶ [Individuals with Disabilities Education Act](#)
- ▶ [Intelligence](#)
- ▶ [Intelligence Quotient](#)
- ▶ [Premorbid Intelligence](#)
- ▶ [Processing Speed Index](#)
- ▶ [Wechsler Individual Achievement Test](#)
- ▶ [Wechsler Intelligence Scale for Children](#)
- ▶ [Wechsler Memory Scale All Versions](#)

- ▶ Wechsler Preschool and Primary Scale of Intelligence
- ▶ Working Memory

memory, including short-term, long-term (declarative), and working memory.

## References

- Tulsky, D. S., Saklofske, D. H., Chelune, G. J., Heaton, R. K., Ivnik, R. J., Bornstein, R., Prifitera, A., & Ledbetter, M. F. (2003). *Clinical interpretation of the WAIS-III and WMS-III*. San Diego: Academic.
- Wechsler, D. (1944). *The measurement of adult intelligence* (3rd ed.). Baltimore: Williams and Wilkins.
- Wechsler, D. (2003). *Wechsler intelligence scale for children—fourth edition (WISC-IV)*. San Antonio: The Psychological Corporation.
- Wechsler, D. (2008). *Wechsler adult intelligence scale—fourth edition (WAIS-IV)*. San Antonio: Pearson Assessment.
- Wechsler, D. (2014). *Wechsler intelligence scale for children—fifth edition (WISC-V)*. San Antonio: Pearson Assessment.
- Weiss, L. G., Keith, T. Z., Zhu, J., & Chen, H. (2013). WAIS-IV and clinical validation of the four-and five-factor interpretative approaches. *Journal of Psychoeducational Assessment*, 31(2), 114–131. <https://doi.org/10.1177/0734282913478030>
- Weiss, L. G., Saklofske, D. H., Holdnack, J., & Prifitera, A. (2016). *WISC-V assessment and interpretation: Scientist-practitioner perspectives*. San Diego: Academic.

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## Wechsler Memory Scale All Versions

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## Synonyms

WMS; WMS-III; WMS-IV; WMS-R

## Definition

The Wechsler Memory Scale – IV (WMS-IV) is the latest (2009) revision of a widely used clinical instrument designed to assess domains of

## Historical Background

The WMS (1945) was developed by David Wechsler to serve as a clinical test to detect and evaluate memory disorders. A number of the original subtests were adapted from existing batteries, such as the Binet-Simon Scale and the Wells and Martin battery; however, Wechsler combined the existing methods for evaluating memory with normative data that allowed for meaningful interpretation of test scores.

The original WMS included seven subtests that were combined to compute an overall Memory Quotient (MQ). The first revision occurred in 1987 (WMS-R) and offered an improved normative sample, the inclusion of four additional index scores, and the introduction of additional subtests. The WMS-R de-emphasized the overall MQ score and focused on verbal-visual and immediate-delayed comparisons.

The measure underwent a revision in 1997 (WMS-III). This revision added subtests designed to assess delayed recognition memory, incorporated process scores, increased ecological validity by removing abstract visual designs and replacing them with everyday stimuli, such as pictures of faces (Faces subtest) and pictures of a family engaged in various activities (Family Pictures subtest), and reconceptualized the Attention/Concentration factor as the Working Memory Index. The WMS-III included eight primary index scores: three global composite scores (immediate memory, general memory (delayed recall), and working memory) and five additional index scores assessing auditory and visual immediate and delayed memory and auditory recognition.

The WMS-III consisted of 11 subtests, five of which were optional and did not contribute to any of the index scores. The subtests measured immediate recall, delayed recall and recognition, auditory memory (immediate and delayed), visual memory (immediate and delayed), and working memory. All the subtests are administered in their



entirety, with the exception of the Spatial Span and Letter Number Sequencing subtests, which were discontinued after consecutive failures of two or three trials of an item, respectively. Raw scores were converted to age-adjusted scaled scores, which were summed to obtain index scores and percentile ranks. Administration required 30–35 min for the primary subtests and 15–20 min for the supplemental subtests.

The WMS underwent the latest revision in 2009 (WMS-IV). The revision was designed to improve clinical utility in a variety of ways ranging from shortening administration times, changing test items, and implementing new scoring rules. The WMS-IV includes five index scores: auditory memory, visual memory, visual working memory, immediate memory, and delayed memory, and new contrast scores designed to provide information on the significance of variation in scores across subtests or indexes.

WMS-IV scores are now derived for the Older Adult Battery (age 65–90) and the Adult Battery (ages 16–69). The revision increased difficulty levels and ceilings in younger age groups, increased floors for older subjects, and reduced testing time for older adults by shortening subtests, changing subtest content, and creating an Older Adult Battery, which consists of a reduced number of subtests.

The latest revision produced several changes to the content and structure of the subtests. The WMS-IV has attempted to reduce confounding factors during testing (e.g., eliminating motor requirements when possible and reducing verbal processing on visual memory subtests) and has eliminated construct overlap with the WAIS-IV by removing subtests that appear on both instruments. The WMS-IV dropped several subtests that appeared on the WMS-III; Digit Span and Letter Number Sequencing were removed because those subtests also appear on the WAIS-IV, the Faces subtest was removed due to floor and administration limitations, Family Pictures was removed in order to reduce verbalization requirements on visual memory tasks, Spatial Span and Word List were removed to improve ease and speed of testing, and the Mental Control and Information and Orientation subtests were

incorporated into a new subtest called the Brief Cognitive Status Exam.

The WMS-IV has made slight modifications to the Logical Memory subtest (e.g., slightly changing story content and introducing a new more age-appropriate story for older adults) and significant modifications to the Verbal Paired Associates and Visual Reproduction subtests. Additionally, the WMS-IV introduced new subtests, including the Brief Cognitive Status Exam, Design Memory, Spatial Addition, and Symbol Span. The WMS-IV also attempted to improve the assessment of working memory by focusing on visual working memory through the creation of subtests that require mental manipulation of visual information and minimal verbalizations.

Evidence for clinical utility is provided in the test manual describing the performance of special group studies, including clinical groups diagnosed with intellectual disability, traumatic brain injury (TBI), Asperger's disorder, dementia, and depression.

## Psychometric Data

The WMS-III was normed on a stratified representative sample of 1,250 individuals aged 16–89 years. Both the WMS-III and WMS-IV conformed to the latest revisions of the Wechsler Adult Intelligence Scale (WAIS) to “allow for meaningful comparisons between intellectual ability and memory functioning.” The average reliability coefficients across age groups for WMS-III subtest scores range from 0.74 to 0.93 with a median reliability of 0.81. The average reliability coefficients for the Primary Indexes range from 0.74 to 0.93 with median reliability of 0.87. Interscorer agreement for the subtests was high, all averaging over 0.90. Evidence of concurrent validity of the WMS-III is based on its correlation with other measures, including the WMS-R, Children's Memory Scale (CMS), and the Wechsler Individual Achievement Test (WIAT). (See the WAIS III and WMS-III Technical Manual for full list of correlations between the WMS-III index scores and other measures.)

The WMS-IV has updated norms for ages 16–90 years from a normative sample of 1,400 individuals designed to provide enhanced utility for older adults. Updated psychometric data can be obtained from the WMS-IV manual.

## Current Knowledge

David Wechsler stated that the WMSs were designed to provide a “rapid, simple, and practical memory examination.” The manual suggests that the measure can be used in a variety of contexts to assess the clinically relevant aspects of memory functioning. For example, when used as part of a complete neuropsychological evaluation, the WMS-III can be used to detect and localize cerebral dysfunction and can aid in detection of dementias and neurodegenerative disorders. In educational settings, it can be used to identify the role of memory deficits in educational difficulties and learning disorders. In rehabilitation settings, the WMS-III can be helpful in identifying the areas of weakness to be addressed in interventions and those spared memory functions that can be utilized for compensation. It can also be used to track progress and assess the efficacy of interventions.

The WMS-III has been used in a variety of clinical and research settings. For example, it has been used in batteries to identify neuropsychological profiles of dementia and distinguish between patients with dementia with Lewy bodies and Alzheimer’s disease (Johnson et al. 2005; Oda et al. 2009); it has been used to understand the learning deficits of individuals with schizophrenia (Hawkins 1999; Skelley et al. 2008) and classify the neurocognitive profiles of TBI patients (Ashman et al. 2008).

The WMS-III has also been used successfully to identify malingering; research suggests that, when used as a part of a comprehensive classification system, the primary indices of the WMS-III can accurately identify malingering of neurocognitive dysfunction in mild TBI (Ord et al. 2008). Similarly, low scores on delayed auditory recognition memory task reliably identified 80% of malingerers of TBI (Langeluddecke and Lucas 2003).

The WMS-IV has been demonstrated to be sensitive to memory disorders in individuals with traumatic brain injury (Carlozzi et al. 2013), schizophrenia, and obsessive-compulsive disorder (Cammisuli and Sportiello 2016) and has been used as a clinical measure of effort (e.g., Young et al. 2011).

Although the WMS-IV has demonstrated superior laterality constructs to the WMS-III (Hoelze et al. 2009), Kent (2013) argues that the current version continues to have limitations in its norming, standardization, and theoretical constructs.

## Cross-References

- ▶ [Children’s Memory Scale](#)
- ▶ [Declarative Memory](#)
- ▶ [Episodic Memory](#)
- ▶ [Memory](#)
- ▶ [Memory Impairment](#)
- ▶ [Wechsler Individual Achievement Test](#)
- ▶ [Working Memory](#)

## References and Readings

- Ashman, T., Cantor, J., Gordon, W., Sacks, A., Spielman, L., Egan, M., et al. (2008). A comparison of cognitive functioning in older adults with and without traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 23, 139–148.
- Cammissuli, D., & Sportiello, M. (2016). Cognitive psychopathology in Schizophrenia: Comparing memory performances with Obsessivecompulsive disorder patients and normal subjects on the Wechsler Memory Scale-IV. *Psychiatria Danubina*, 28, 118–126.
- Carlozzi, N., Grech, J., & Tulskey, D. (2013). Memory functioning in individuals with traumatic brain injury: An examination of the Wechsler memory scale-fourth edition. *Journal of Clinical and Experimental Neuropsychology*, 35, 906–914.
- Hawkins, K. (1999). Memory deficits in patients with schizophrenia: Preliminary data from the Wechsler memory scale-third edition support earlier findings. *Journal of Psychiatry & Neuroscience*, 24, 341–347.
- Hoelze, J., Nelson, N., & Smith, C. (2009). Comparisons of Wechsler memory scale-fourth edition and third edition dimensional structures: Improved ability to evaluate auditory and visual constructs. *Journal of Clinical and Experimental Neuropsychology*, 33, 283–291.
- Johnson, D., Morris, J., & Galvin, J. (2005). Verbal and visuospatial deficits in dementia with Lewy bodies. *Neurology*, 65, 1232–1238.

- Kent, P. (2013). The evolution of the Wechsler memory scale: A selective review. *Applied Neuropsychology: Adult*, 20, 4.
- Langeldudecke, P., & Lucas, S. (2003). Quantitative measures of memory malingering on the Wechsler memory scale – Third edition in mild head injury litigants. *Archives of Clinical Neuropsychology*, 18, 181–197.
- Oda, H., Yamamoto, Y., & Maeda, K. (2009). The neuropsychological profile in dementia with Lewy bodies and Alzheimer. *International Journal of Geriatric Psychiatry*, 24(2), 125–131.
- Ord, J., Greve, K., & Bianchini, K. (2008). Using the Wechsler memory scale-III to detect malingering in mild traumatic brain injury. *The Clinical Neuropsychologist*, 22, 689–704.
- Skelly, S., Goldberg, T., Egan, M., Weinberger, D. R., & Gold, J. M. (2008). Verbal and visual memory: Characterizing the clinical and intermediate phenotype in schizophrenia. *Schizophrenia Research*, 105, 78–85.
- The Psychological Corporation. (1997). *WAIS-III-WMS-III technical manual*. San Antonio: The Psychological Corporation.
- Tulsky, D., Chiaravalloti, N., Palmer, B., & Chelune, G. (2003). The Wechsler Memory Scale, Third Edition: A new perspective. In D. S. Tulsky, et al. (Eds.), *Clinical interpretation of the WAIS-III and the WMS-III* (pp. 93–138). San Diego: Academic.
- Uttl, B., & Graf, P. (1999). The Wechsler memory scale-III: Validity and reliability. *Archives of Clinical Neuropsychology*, 14(8), 705–706.
- Wechsler, D. (1945). A standardized memory scale for clinical use. *Journal of Psychology*, 19, 87–95.
- Wechsler, D. (1997). *WMS-III administration and scoring manual*. San Antonio: The Psychological Corporation.
- Young, C., Sawyer, R., Roper, B., & Baughman, B. (2011). Expansion and re-examination of digit span effort indices on the WAIS-IV. *The Clinical Neuropsychologist*, 26, 147–159.

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## Wechsler Preschool and Primary Scale of Intelligence

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### Synonyms

WPPSI-III

### Description

The Wechsler Preschool and Primary Scale of Intelligence-Fourth Edition (WPPSI-IV; Wechsler 2012) is an individually administered intelligence test for children, ages 2 years 6 months (2:6) through 7 years 7 months (7:7). The WPPSI-IV is a substantial revision of the WPPSI-III (Wechsler 2002). The WPPSI-IV provides two age-band subtest batteries for ages 2:6–3:11 and 4:0–7:7. The 2:6–3:11 battery includes a set of 7 subtests that yield a Full Scale IQ (FSIQ) score, three primary index scores (i.e., Verbal Comprehension Index [VCI], Visual Spatial Index [VSI], and Working Memory Index [WMI]), and three ancillary index scores (i.e., Vocabulary Acquisition Index [VAI], Nonverbal Index [NVI], General Ability Index [GAI]). The 4:0–7:7 battery includes a total of 15 subtests (the seven from the 2:6–3:11 and eight additional subtests) that yield the same composite scores as the 2:6–3:11 battery and adds two additional primary index scores (i.e., Fluid Reasoning Index [FRI] and Processing Speed Index [PSI]) and one additional ancillary index score (Cognitive Proficiency Index [CPI]).

For the younger age band, Information and Receptive Vocabulary are used to generate the VCI, Block Design and Object Assembly are used to derive the VSI, and Picture Memory and Zoo Locations contribute to the WMI. The FSIQ is derived from Receptive Vocabulary, Information, Block Design, Object Assembly, and Picture Memory. In some clinical situations, Picture Naming is used with Receptive Vocabulary to generate the VAI. As needed, Block Design, Object Assembly, Picture Memory, and Zoo Locations are used to derive the NVI. Receptive Vocabulary, Information, Block Design, and Object Assembly may be used to obtain the GAI.

For the older age band, Information and Similarities are used to generate the VCI, Block Design and Object Assembly are used to derive the VSI, Matrix Reasoning and Picture Concepts are used to obtain the FRI. Picture Memory and Zoo Locations contribute to the WMI, and Bug Search and Cancellation are used to generate the PSI. The FSIQ is obtained using Information,

Similarities, Vocabulary, Comprehension, Block Design, Matrix Reasoning, Picture Memory, and Bug Search. If needed, Receptive Vocabulary and Picture Naming are used to derive the VAI. Block Design, Matrix Reasoning, Picture Concepts, Picture Memory, and Bug Search can be used to obtain the NVI. Information, Similarities, Block Design, and Matrix Reasoning may be used to obtain the GAI, and the CPI can be derived using Picture Memory, Zoo Locations, Bug Search, and Cancellation. For clinical purposes, supplemental subtests may be used as a substitution for core subtests when deriving any composite score that has at least four subtests. The FSIQ for the younger battery can be obtained in 27 min on average, and the primary index scores can be obtained in approximately 32 min. The older battery's FSIQ can be obtained in approximately 31 min, and the five primary index scores can be obtained in 60 min on average. If additional ancillary index scores are desired or if supplemental subtests are administered, the administration time increases accordingly.

The WPPSI-IV was normed on a stratified sample of 1,700 children, divided into nine age groups, including 200 children in each 6 month interval from ages 2:6 to 5:11, 200 6 year olds, and 100 children ages 7:0–7:7. Sample demographics and geographic locations were based on the US Bureau of the Census for 2010.

## Historical Background

The Wechsler intelligence test series began with the publication of the Wechsler-Bellevue Scale (Wechsler 1939), but the content of the Wechsler-Bellevue has a long history, dating back to the nineteenth century (Boake 2002). The Army intelligence tests, utilized in World War I, including Group Examinations Alpha and Beta, were key sources for subtests incorporated into the Wechsler-Bellevue. As a military psychological examiner, Wechsler experienced the inadequacy of existing intelligence tests. Important innovations in the Wechsler-Bellevue scale included replacing ratio intelligence quotients, calculated as mental-chronological age

ratios, with deviation scores; and utilizing both verbal and performance tests, in part to address bias in verbal subtest content and demands and to assess multiple aspects of intelligence.

The WISC (Wechsler 1949) was developed as a children's version of a revised Wechsler-Bellevue scale. The normative sample for the WISC was Caucasian. In 1967, the WPPSI was published as a preschool version of the WISC, for children aged 4:0–6:6. The initial revision of the WPPSI (WPPSI-R) was published in 1989, with an expanded age range, from 3:0 to 7:3. The WPPSI-R retained previous subtests but added Object Assembly. Age-determined start points for subtests were introduced. Although David Wechsler died in 1981, he continued to be listed as sole author of revised Wechsler tests.

The WPPSI-III further expanded the age range to 2:6–7:3 and addressed long-standing criticisms of the WPPSI-R by reducing testing time, expressive language demands, and confounding effects of speed. In addition, revisions included more age-appropriate test instructions, increased prompts, and simplified administration procedures.

The WPPSI-IV expanded the age range to 2:6–7:7 and updated the theoretical foundations of the test by considering contemporary structural intelligence models, neurodevelopmental and neurocognitive research, and working memory models and research. It increased the developmental appropriateness of the test by changing kit manipulatives (e.g., for the Processing Speed subtests which had weak floors for 4-year-old children, the pencil was replaced with an ink dauber, the abstract stimuli were replaced with child friendly and game-like pictures, and the cognitive demands of the tasks were simplified), simplifying instructions further, and developing more age-appropriate measures of processing speed. It increased the user friendliness of kit components and reduced testing time. It enhanced clinical utility by updating the test structure, reducing the number of initial comparisons in the score differences comparison methodology, adding new ancillary index scores, including new special group studies, and reducing the expressive language requirements.

## Psychometric Data

Internal consistency was examined using split-half methods, with the exception of the Processing Speed subtests. Average subtest reliability coefficients range from 0.75 to 0.91, typically maintaining or improving upon the WPPSI-III values. Composite score reliability coefficients range from 0.86 to 0.96. The PSI average reliability coefficient of 0.86 is slightly lower because it necessarily utilizes test-retest rather than split-half reliabilities. Subtest reliability coefficients from special populations, including children with intellectual giftedness, intellectual disability, cognitive developmental delay, developmental risk factors, pre-literacy concerns, attention-deficit/hyperactivity disorder, disruptive behavior, language disorder with expressive impairment, language disorder with receptive and expressive impairment, English language learners, and autism spectrum disorders with and without language impairment are at a minimum comparable to those from the normative sample. Test-retest stability was demonstrated over a mean interval of 23 days. Average corrected stability coefficients of the subtests are adequate (0.70s) to good (0.80s). Average corrected stability coefficients of the composite scores range from good to excellent, with a mean increase in FSIQ of 4.6 points. Finally, interscorer agreement, examined by double scoring protocols with two independent scorers, was very high (0.98–0.99) for subtests with simple, objective scoring criteria. Interscorer agreement for subtests that require more judgment in scoring (i.e., Information, Similarities, Vocabulary, Comprehension, and Picture Naming), examined among nine independent raters, was also quite high, 0.96–0.99. Generally high reliability is associated with low standard errors of measurement and reasonable confidence intervals that reflect the precision of observed test scores. Tables in the Administration and Scoring Manual provide comprehensive confidence interval data.

Regarding score differences, the WPPSI-IV psychometrics include the statistical significance of primary index score and primary subtest score differences from the mean of the relevant score group (i.e., all primary index scores or all primary subtest scores) within a profile, and of pairwise

discrepancies across pairs of index scores and relevant subtests, as well as base rate frequency of differences in the normative sample for each comparison. Importantly, the clinician can determine the extent to which a statistically significant score difference is relatively common or rare. For example, among children with FSIQ  $\leq 79$ , it is more common to have a VCI that is lower than the mean of the primary index scores than a VSI that shows this result.

Lines of validity evidence examine internal structure and relations with other variables. Evidence of validity based on internal structure includes evidence that all intersubtest correlations are significant, supporting the premise of a general intelligence factor; that is, *g*. In the younger age band, 2:6–3:11, Verbal Comprehension subtests are more highly correlated with other Verbal Comprehension subtests than with Visual Spatial or Working Memory subtests. However, the Visual Spatial and Working Memory subtests do not consistently correlate more highly with each other than with Verbal Comprehension subtests. In the older age band, 4:0–7:7, subtests tend to correlate more highly within each scale (i.e., Verbal Comprehension, Visual Spatial, Fluid Reasoning, Working Memory, Processing Speed) than across scales, apparently reflecting more well-differentiated cognitive abilities with age.

Confirmatory factor analytic studies support a three-factor solution (Verbal Comprehension, Visual Spatial, and Working Memory) for the younger age band, and a five-factor solution (Verbal Comprehension, Visual Spatial, Fluid Reasoning, Working Memory, and Processing Speed) for the older age band. These factor models fit equally well for smaller age groups examined within each age band.

Evidence for validity based on relations with other measures includes evidence that the WPPSI-IV composite scores are not significantly different than Bayley Scales of Infant Development-Third Edition (Bayley-III) cognitive scale scores. The WPPSI-IV FSIQ correlates more strongly with the Bayley-III Cognitive and Language scales than Motor scale. Differential Abilities Scales-Second Edition (DAS-II) and WPPSI-IV composite scores are not significantly different, with the



exception of DAS-II Verbal scores that are slightly higher than WPPSI-IV VCI scores and DAS-II GCA scores that are slightly higher than WPPSI-IV FSIQ scores. The WPPSI-IV FSIQ correlates most strongly with NEPSY-Second Edition (NEPSY-II) Narrative Memory Free Recall, Speeded Naming Combined, and Phonological Processing among all NEPSY-II subtests. Wechsler Individual Achievement Test-III (WIAT-III) linkage studies indicate that the WPPSI-IV VCI is more highly correlated with the WIAT-III reading and written expression subtests than are the WPPSI-IV VSI, FRI, WMI, or PSI. The WPPSI-IV FSIQ correlates 0.75 with WIAT-III Total Achievement.

The initial psychometric data include validity evidence from special groups that are compared with groups of children matched on key demographic variables. These were small samples of convenience, and caution is warranted in generalizing findings to clinical practice. The special group study results were consistent with previous research findings and expectations. Of all primary index scores, the largest effect size of the mean difference between the intellectual giftedness group and matched controls occurred on the VCI. In samples of children previously identified with mild to moderate intellectual disability, WPPSI-IV composite scores generally fell within the expected range, 2–3 standard deviations below the mean for those with mild intellectual disability and 3–4 standard deviations below the mean for those with moderate intellectual disability. In the group with mild intellectual disability, the largest primary index score effect sizes occurred on the VSI and the VCI. In the group with moderate impairment, the lowest primary index score occurred on the VCI and the VSI. For the group of children with cognitive developmental delay, the largest effect sizes were present on the VCI and the FRI. For children with developmental risk factors, the largest effect sizes were present on the WMI, VCI, and VSI. A sample of children exhibiting signs of preliteracy problems had largest effect sizes on the VSI and the VCI. A group of children with ADHD showed the largest effect size on the VCI. The group of children with other disruptive behavior problems

produced the largest effect sizes on the VCI and PSI. Consistent with expectations, the groups with language impairment had the largest effect sizes on the VCI. Also as expected, the group of children who are English language learners showed the largest effect size on the VCI. The NVI effect size was negligible and, interestingly, the English language learner group scored more highly on this index score than did the demographically matched control group. Predictably, the group of children with autism spectrum disorder with language impairment had the largest effect size on the VCI. The group without language impairment, however, showed the largest effect size on the PSI.

## Clinical Uses

The WPPSI was developed to measure intelligence in preschool age children, in part to address the needs of early education and intervention programs. The WPPSI-IV expands on the clinical utility and increases developmental appropriateness relative to its predecessor, the WPPSI-III. Extensive psychometric data support the use of the WPPSI-IV in identifying general levels of intelligence in the 2:6–7:7 age groups.

When compared with other available instruments for younger children, the WPPSI-IV has both advantages and limitations. For example, the WPPSI-IV is primarily designed to measure cognitive development in detail, whereas the Bayley Scales of Infant and Toddler Development, Third Edition, (Bayley-III; Bayley 2005) is designed to assess the development of children across cognitive, language, motor, behavioral, and social-emotional domains without assessing cognitive development in depth. The Bayley-III, however, covers a lower age range and utilizes a much more flexible administration procedure that can be helpful in maintaining a young child's attention. The Kaufman Assessment Battery for Children, Second Edition (KABC-II), does not provide as many separate composite scores at age 3. The processing speed confound in portions of the KABC-II is not present in the WPPSI-IV. The WPPSI-IV also extends to a lower age range than the KABC-II.

Despite its solid psychometric foundation, to date, there is a paucity of empirical literature on clinical applications of the WPPSI-IV.

## Cross-References

- [Wechsler Intelligence Scale for Children](#)

## References and Readings

- Boake, C. (2002). From the Binet-Simon to the Wechsler-Bellevue: Tracing the history of intelligence testing. *Journal of Clinical and Experimental Neuropsychology*, 24, 383–405.
- Dumont, R., & Willis, J. O. (2014). Strengths and weaknesses of the WPPSI-IV. In S. E. Raiford & D. L. Coalson (Eds.), *Essentials of WPPSI-IV assessment* (pp. 189–213). Hoboken: Wiley.
- Raiford, S. E., & Coalson, D. L. (2014). *Essentials of WPPSI-IV assessment*. Hoboken: Wiley.
- Syeda, M. M., & Climie, E. A. (2014). Test review: Wechsler preschool and primary scale of intelligence-fourth edition. *Journal of Psychoeducational Assessment*, 32, 265–272.
- Wechsler, D. (2012). *Wechsler preschool and primary scale of intelligence technical and interpretative manual* (4th ed.). Bloomington: Pearson.

## Wechsler Test of Adult Reading

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## Synonyms

WTAR

## Description

The Wechsler Test of Adult Reading (WTAR) is intended as a measure of premorbid intellectual

functioning that can be used for individuals aged 16–89. Scores from the WTAR have been used to predict IQ and index scores on the Wechsler Adult Intelligence Scale (WAIS-III) and Wechsler Memory Scale (WMS-III). The test typically takes 5–10 min and consists of 50 word items with irregular spelling. Each item is individually presented on a word card, and examinees are asked to pronounce each. The total raw score is the number of correct pronunciations (maximum score of 50). The obtained raw score is converted to a standard score that is compared to a “predicted” score. The predicted WTAR is derived from demographic data based on the test’s normative sample and then subtracted from the obtained WTAR score to assess the magnitude of difference. Obtained WTAR scores that are 20 or more points lower than predicted scores are thought to inaccurately represent premorbid IQ or ability (Strauss et al. 2006).

## Historical Background

The WTAR was developed by David Wechsler and published in 2001. The WTAR was developed using the same paradigm used for reading recognition as the National Adult Reading Test (NART), North American Adult Reading Test (NAART), and American National Adult Reading Test (ANART). All of these reading recognition measures are based on the assumption that word recognition is preserved in cases of neurologic insult and/or cognitive decline. The WTAR is one of few reading recognition measures used to specifically predict scores on the WAIS-III and WMS-III in that the same normative sample was used for all three.

## Psychometric Data

Normative data were drawn from a pool of 1134 individuals from the United States (US) and 331 individuals from the United Kingdom (UK). Groups were stratified by age, sex, and level of education. Due to higher levels of ethnic and cultural diversity in the US sample, the test

developer included ethnicity and geographic location as additional stratification variables. Internal consistency coefficients for all age groups range from 0.90 to 0.97 in the US sample and from 0.87 to 0.95 in the UK sample. Test-retest reliability coefficients provided for the US sample only range from 0.92 to 0.94 (minimal practice effects reported) (The Psychological Corporation 2001). Concurrent validity coefficients range from 0.73 to 0.90 (highest correlation with the NART). WTAR correlations with WAIS-III are highest with verbal measures (verbal IQ  $r = 0.75$ , verbal comprehension  $r = 0.74$ ), with moderate correlations found with other indexes (performance IQ  $r = 0.59$ , perceptual organization index  $r = 0.56$ , processing speed index  $r = 0.47$ , working memory index  $r = 0.62$ ). Correlations between the WTAR and the WMS-III in the US sample were as follows: working memory  $r = 0.51$ , general memory  $r = 0.49$ , immediate memory  $r = 0.47$ ). The WTAR score has been shown to predict, within ten points, full-scale IQ scores in 70.4% of cases and predict WMS-III memory scores in 55% of cases (Strauss et al. 2006). It should be noted that for both the US and UK samples, WTAR scores have been found to steadily increase between the ages of 16 and 54 and then decline between 80 and 89 years of age (The Psychological Corporation 2001).

### Current Knowledge

The WTAR score can be used alone or in combination with demographic data to predict premorbid ability (Spies and Plake 2005). However, the test publisher recommends using both to predict IQ (The Psychological Corporation 2001). It should be noted that the WTAR is not intended as a measure of reading disability, developmental delay, or intellectual disability. Normative data are available for samples in the United States and United Kingdom and include stratification data for age, gender, ethnicity, and education. The WTAR manual provides information about the development and standardization of the test;

however, little data are provided regarding the development of test items (Spies and Plake 2005).

A recent investigation validated the use of the WTAR for determining premorbid IQ on a sample of 24 patients suffering from traumatic brain injury (TBI) (Green et al. 2008). In addition, the WTAR was shown to be reliable over several months, with scores remaining unaffected by the cognitive effects of brain injury (Green et al. 2008).

### Clinical Uses

The WTAR has been shown to provide an intelligent quotient (IQ) estimate for individuals who have sustained neurologic damage such as TBI or have suffered cognitive decline related to conditions such as dementia, schizophrenia, depression, and attention deficit hyperactivity disorders (Spies and Plake 2005; The Psychological Corporation 2001). Studies have shown that WTAR scores are robust in predicting functioning in individuals with progressive dementias, including Alzheimer's, Huntington chorea, and Parkinson's disease (Strauss et al. 2006); however, persons with moderate-to-severe Alzheimer's have been shown to have much lower scores. The same has been found among individuals with more severe TBIs (Mathias et al. 2007), and preexisting learning disabilities, raising questions about the use of the WTAR with these groups.

### Future Directions

A clinical limitation of the WTAR is its reliance on pronunciation of target words, which in turn is dependent on an individual's capacities to read and speak (Green et al. 2008). Leritz et al. (2008) found that the WTAR may not accurately reflect premorbid intellectual functioning in patients with aphasia, since these individuals may maintain portions of verbally mediated intelligence that is not adequately measured through the WTAR's pronunciation format. Thus, future research is needed to fully validate the use of the WTAR with other clinical populations.

## References and Readings

- Green, R. E. A., Melo, B., Christensen, B., Ngo, L. A., Monette, G., & Bradbury, C. (2008). Measuring pre-morbid IQ in traumatic brain injury: An examination of the validity of the Wechsler test of adult reading (WTAR). *Journal of Clinical and Experimental Neuropsychology*, *30*, 163–172.
- Leritz, E. C., McGlinchey, R. E., Lundgren, K., Grande, L. J., & Milberg, W. P. (2008). Using lexical familiarity judgments to assess verbally mediated intelligence in aphasia. *Neuropsychology*, *22*, 687–696.
- Mathias, J. L., Bowden, S. C., Bigler, E. D., & Rosenfeld, J. V. (2007). Is performance on the Wechsler test of adult reading affected by traumatic brain injury? *British Journal of Clinical Psychology*, *46*, 457–466.
- Spies, R. A., & Plake, B. S. (Eds.). (2005). *The sixteenth mental measurements yearbook*. Lincoln: Buros Institute of Mental Measurements.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (Eds.). (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary*. New York: Oxford Press.
- The Psychological Corporation. (2001). *Wechsler test of adult reading*. San Antonio: Harcourt Assessment.

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## Wernicke, Karl (1848–1905)

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### Major Appointments

- Assistantship, Eye Clinic, University of Breslau, Germany (1870)
- Assistant Army Field Surgeon, Franco-Prussian War (July 1870–May 1871)
- Assistantship, Psychiatric Institute of the Allerheiligen Hospital, Breslau (1871–1875)
- Clinic for Psychiatry and Nervous Diseases, Berlin Charité Hospital (1875–1878)
- Private Neuropsychiatric practice in Berlin (1878–1885)
- Associate professor, Department of Neurology and Psychiatry, University of Breslau (1885–1890)

- Chairman of the Department of Neurology and Psychiatry, University of Breslau (1890–1904)
- Director of the Clinic of Psychiatry and Neurology, University of Halle-Wittenberg, Germany (1904–1905)

### Landmark Clinical, Scientific, and Professional Contributions

- Karl Wernicke (aka Carl Wernicke) was a pioneer of classical psychiatry and neurology. His work helped form the foundation of modern neuropsychology and cognitive neuroscience. He led a colorful and prolific life with the bulk of his work reflective of a fundamental interest in the relations among neuroanatomy, physiology, and behavior. Perhaps his greatest achievements were his role in the discovery that focal disease causes specific deficits and his research on sensory aphasia. He discovered the area of the brain that is critical for language comprehension, which is now known as Wernicke's area. His work was greatly influenced by his mentors Theodor Meynert and Karl Westphal and the work of Wilhelm Griesinger.
- In 1874, at the age of 26, Wernicke published his first work on aphasia, *Der Aphasische Symptomencomplex (The Aphasia Symptom-Complex)*. This groundbreaking book presented the discovery of sensory aphasia and received strong attention from the neurological community. In the opening monograph, he acknowledged that the work reflected an application of Meynert's neuroanatomical approach to the study of aphasia, and he acknowledged Paul Broca's work on the localization of a motor speech area, now known as Broca's area.
- From 1881 to 1883, he published a three-volume work, *Lehrbuch der Gehirnkrankheiten (Textbook of Brain Diseases)*, which has been referred to as Wernicke's most significant contribution to the area of clinical neurology. These volumes contain an anatomical/physiological review on the classification of brain diseases and a discussion of the involvement of focal

regions of the brain. This work was largely based on Meynert's doctrine which describes and discusses how anterior regions of the cerebral cortex are responsible for motor functions and posterior regions are responsible for sensory functions. Meynert's doctrine also discusses the presence of fiber tracts which link these areas. These concepts have been largely described in Meynert's 1884 publication, *Psychiatrie*.

## Wernicke's Aphasia

Wernicke's work on aphasia is largely described in his 1874 publication, *The Aphasia Symptom-Complex*, and his 1906 posthumous publication entitled *Deutsche-Klinik*. The 1906 publication summarizes his earlier and later works regarding aphasia and recapitulates the basic tenets of his theories on aphasia. In the 1874 publication, he presents a case study of a stroke survivor who was able to hear and speak fluently, but could not understand written or spoken language. In addition, although he could produce semantically and syntactically correct oral and written language, the things he said and wrote made no sense and often had no content. At postmortem, Wernicke discovered that the patient had a localized lesion in a posterior region of the left hemisphere of his brain, around where the temporal and parietal lobes meet. Wernicke concluded that this area was involved in speech comprehension, and he named the syndrome sensory aphasia. However, it is now referred to as Wernicke's aphasia or fluent aphasia. Moreover, the affected area has been labeled Wernicke's area, but it is also sometimes referred to as the receptive language area as scientists now believe that it may be involved in semantic processing.

As mentioned previously, Wernicke's work on sensory aphasia was influenced by an earlier discovery on the function of an area of the left hemisphere anterior to Wernicke's area, which is now referred to as Broca's area. In 1861 France, Paul Broca discovered that this region was involved in language production. His famous patient, called "Tan," could understand written and spoken words but could not say anything but the word

"tan." Wernicke also postulated in his publications on aphasia that Broca's area and Wernicke's area were connected. He predicted that damage to the connection between these areas would produce conduction aphasia, where a patient can speak and understand language but would misuse words and could not repeat words. This prediction later turned out to be correct.

## Wernicke's Encephalopathy

In the second volume of his *Textbook of Brain Diseases*, Wernicke described a syndrome correlated with the ingestion of sulfuric acid. He called the syndrome as acute hemorrhagic superior polioencephalitis, and it was characterized by specific mental and motor abnormalities and the paralysis of the muscles in the eyes. These symptoms were later discovered to be caused by nutritional thiamin (vitamin B1) deficiency, and the syndrome is now referred to as Wernicke's encephalopathy.

It was later discovered that Wernicke's encephalopathy is related to another condition called Korsakoff's psychosis or Korsakoff's syndrome. In a series of articles from 1887 to 1891, a Russian neuropsychiatrist named Sergei S. Korsakoff described a brain disease linked to alcohol consumption and malnutrition that presented as severe memory deficits (especially short-term memory deficits), confabulation (which is when a person makes up information to fill in gaps in his or her memory), and polyneuropathy (*psychosis polyneuritica*). Essentially, Korsakoff's psychosis is a continuation of Wernicke's encephalopathy, though a recognized episode of Wernicke's encephalopathy is not always obvious. A deficiency of thiamine is responsible for both Korsakoff's psychosis and Wernicke's encephalopathy. The symptom complex formed by both conditions has been termed the Wernicke-Korsakoff syndrome. Wernicke-Korsakoff syndrome is basically a two-stage disease, with the first, acute phase consisting of mental confusion, ocular motor paralysis, and ataxia (poor motor coordination) and the second, chronic stage consisting of severe memory deficits and confabulation.



## Short Biography

Karl Wernicke was born on May 15, 1848, in the village of Tarnowitz in Upper Silesia, which is now Tarnowskie Gory, Poland. His father was a secretary to a town official, and his mother was a housewife with a strong interest in Wernicke's education and hoped that he would study for the ministry. He attended public schools in Upper Silesia and college in Breslau and Oppeln. He studied medicine under professors Haidenhain, Meddledorph, Spiegelbert, and Lebert at the University of Breslau. He completed a six-month assistantship at the Eye Clinic at the University of Breslau under Otfried Foerster, who became his lifetime friend and patron. After his assistantship, he served as an assistant army field surgeon to Fischer during the Franco-Prussian War of 1870. After completing his military term, he accepted an assistantship in the Psychiatric Institute of the Allerheiligen Hospital under Heinrich Neumann. His colleagues included neuropsychologist Carl Friedländer and the neurohistologist Weigert, who also became his lifetime friends. During his assistantship, Neumann allowed Wernicke to study for six months with Theodor Meynert in Vienna.

Wernicke's experience with Meynert significantly influenced his interest in neuroanatomy and aphasias. After his work with Meynert and Neumann, he accepted an assistantship with Karl Westphal in a German psychiatric polyclinic in Berlin, Berlin Charité Hospital. In 1876, he completed his Habilitation, which is a level of achievement in Germany that permits medical assistants to become junior members of the medical faculty and allows them to receive financial compensation for lectures. However, in 1878, he became involved in a conflict with the hospital administration in regard to some private matter, which unfortunately resulted in his termination. Afterward, he worked for a period of seven years in the private practice of neurology. During this period of time, he did a lot of writing including his three-volume work (1881–1883), *Lehrbuch der Gehirnkrankheiten (Textbook of Brain Diseases)*.

In 1879, Wernicke also published the essay *Ueber das Bewusstsein*, which translates as *In*

*Regard to Consciousness*, and applied to the Psychiatric Institute of Dalldorf. He did not receive the position at Dalldorf. His failure was rumored to be due to Virchow's protest and the influence of a powerful Ministerial Director, Friedrich Althoff, who was involved in his dismissal from the Berlin Charité Hospital. However, in 1885 with the help of his old friend and supporter, Foerster, he was offered the opportunity to succeed Heinrich Neumann at the University of Breslau. He accepted, and in 1890 he was promoted to the position of Ordinarius, chairman of the Department of Neurology and Psychiatry. Wernicke held his faculty position at the University of Breslau for almost 20 years. His research and activity at Breslau was focused mostly on the study of mental illness and his continued interest in neuroanatomical and neuropathological explanations of psychiatric symptomology. During his time at Breslau, he also produced many of his classical works on aphasia and worked with many gifted assistants and colleagues whose respective contributions to science also greatly helped shape the history of neurology (e.g., Liepmann, Lissauer, Heilbronner, Foerster, and Kleist).

Despite his many accomplishments at the University of Breslau, Wernicke reportedly had goals for loftier positions. He set his sights on three choice university positions that became vacant in Germany at that time: Chairman of the Department of Neurology and Psychiatry at Vienna, Director of the Neurological Clinic at Berlin, and another position in Munich. However, misunderstandings and misfortune prevented Wernicke from obtaining any of the three desired positions. Misfortune continued in his last years at Breslau when he lost his assistants (including Liepmann who went to Dalldorf) due to administrative challenges. Specifically, the Psychiatry Clinic at the University of Breslau was under municipal administration which decided to sever connections with the Institute. Moreover, the state refused to supply funds for the building of a new clinic. At first, Wernicke (who held positions at the hospital and university) was still allowed to use the clinical resources and lecture at the Institute; however, those privileges also were



later withdrawn. Thus, without a university affiliation, he had only access to a municipal neurological polyclinic, and his assistants chose positions elsewhere. Rather than be discouraged, Wernicke took advantage of this newfound freedom and produced his renowned work on psychiatry entitled *Grundriss der Psychiatrie*, which translates as *Foundations of Psychiatry*.

His privileges at the University of Breslau were later restored. Nevertheless, in 1904 Wernicke left the municipal institute and took a new position as the director of the Clinic of Psychiatry and Neurology at the University of Halle-Wittenberg, succeeding Theodor Ziehen. Wernicke's years at Halle included the meticulous collection of data which applied, tested, and further advanced his hypotheses on the problems of mental illness. Moreover, his work at Halle included a further exploration into the areas of neurohistology and neurosurgery. Although his efforts were mostly devoted to the study of psychiatric problems, he strongly encouraged his assistants on their research in neurology, in particular their research on aphasia and related disorders. Perhaps his youngest and most successful assistant at the University of Halle was Karl Kleist, who assisted in writing his memorial and was responsible for supplying much of the information included in biographies on Wernicke regarding his experiences at Halle.

Wernicke was reportedly very happy with his professional life and personal life during his short time at Halle. He had a rich social life that included family and friends and often enjoyed mountain climbing, gymnastics, and cycling. In fact, it was during one of his recreational activities that he met his end. On June 15, 1905, Karl Wernicke died at the age of 57 from a cycling accident, which occurred on a narrow road in the Thüringer forest. Wernicke and his surgical colleague, Berthold Pfeifer, met a timber cart on the road, and Wernicke fell under the cart and his chest was crushed by the rear wheel. He lived for two days, but would not allow his wife to be called until the very last to spare her the anguish of his suffering and pain.

In closing, Karl Wernicke led a colorful and eventful life. He was a brilliant and prolific scientist

whose work was instrumental to the cultivation and advancement of modern neuropsychology and cognitive neuroscience.

## Cross-References

- ▶ [Aphasia](#)
- ▶ [Broca, Pierre Paul \(1824–1880\)](#)
- ▶ [Broca's Aphasia](#)
- ▶ [Korsakoff's Syndrome](#)
- ▶ [Korsakoff \(Korsakov\), Sergei Sergeievich \(1854–1900\)](#)
- ▶ [Wernicke's Aphasia](#)
- ▶ [Wernicke-Korsakoff Syndrome](#)
- ▶ [Wernicke–Lichtheim Model of Aphasia](#)

## References and Readings

- Eggert, G. H. (1977). *Wernicke's works on aphasia: A sourcebook and review: Early sources in aphasia and related disorders* (Vol. 1). The Hague: Mouton Publishers.
- Pillmann, F. (2003). Carl Wernicke (1848–1905). *Journal of Neurology*, 250(11), 1390–1391.

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## Wernicke's Aphasia

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## Synonyms

Posterior aphasia; Receptive aphasia; Sensory aphasia

## Short Description

Wernicke's aphasia is a type of aphasia characterized by fluently articulated speech with impaired language comprehension. Utterance length is normal or extended, and the patient's spontaneous speech has normal prosody and

grammar and is produced with normal effort. There is a reduction in content words, and paraphasic errors and neologisms are common, with logorrhea (excessive verbal output) in some patients. Repetition mirrors extemporaneous speech, and reading and writing are similar to auditory comprehension and spoken language. Particularly in the acute stage, Wernicke's aphasia often is associated with anosognosia (unawareness of deficits).

## Categorization

Wernicke's aphasia is a subtype of fluent aphasia.

## Natural History, Prognostic Factors, and Outcomes

The prognosis for recovery of functional communication in individual with fluent aphasia depends on the underlying cause of the aphasia as well as factors such as the size of lesion and the patient's age, premorbid language skills, and comorbid health conditions. In general, individuals with Wernicke's aphasia have a poorer prognosis for recovery of language function than those with other aphasia types.

Individuals who initially present with Wernicke's aphasia often evolve to a clinical profile of conduction or anomic aphasia, with relatively good auditory and reading comprehension, and deficits primarily in word finding and the comprehension and production of complex syntax. Language comprehension typically recovers before production, most likely because comprehension is represented bilaterally in the brain.

## Neuropsychology and Psychology of Wernicke's Aphasia

Wernicke's aphasia historically has been attributed to lesions in the posterior portion of the superior temporal gyrus (pSTG) in the language-dominant hemisphere (Brodmann's area 22). In his original work, however, Wernicke (1874) linked lesions in

this area to impairments in processing *speech* sounds (i.e., phoneme perception), not *language*, a distinction that has not been maintained in clinical definitions of Wernicke's aphasia since that time (Mesulam et al. 2015). There is strong evidence from behavioral, physiological, and neuroimaging studies that language comprehension involves a widely distributed bilateral network of cortical and subcortical structures (see review in Binder 2017), so in reality it is unlikely that impaired comprehension would result from a lesion in the pSTG alone. Instead, pSTG lesions can result in *conduction* aphasia (Buchsbaum et al. 2011), which is characterized by phonemic paraphasias and intact comprehension, consistent with the role of the pSTG in processing speech sounds. Links between pSTG lesions and Wernicke's area might reflect the prevalence of stroke as the etiology of these lesions. The middle cerebral artery (MCA) supplies not only the pSTG but also the posterior middle and inferior temporal gyri (Brodmann's areas 20, 21, and 37) and the inferior parietal lobule, including inferior portions of the angular and supramarginal gyri (Brodmann's areas 39 and 40) (Damasio 2001). Thus, Wernicke's aphasia after middle cerebral artery stroke may be due to damage to a network of regions rather than Wernicke's area specifically.

## Evaluation

As with other aphasia types, individuals with fluent aphasias are typically evaluated using a combination of standardized language tests and careful observation of extemporaneous communication. The tests and measures used depend on the goals of the assessment (e.g., diagnosis vs. prediction of functional performance vs. treatment planning), the time postonset (e.g., comprehensive test batteries are not appropriate in the context of acute stroke), and the patient's clinical presentation.

## Treatment

Treatment of Wernicke's aphasia can be a challenge in the acute stage, when patients are

unaware of their specific linguistic deficits. Patients may perceive the problem as being in the ears of the listener, who fails to understand and adapt to the patient's "own language." In the chronic stage, depression is a risk factor in all patients with aphasia and careful attention should be paid to vegetative and affective signs in patients who have difficulty communicating their thoughts and feelings.

Treatment is focused on improving auditory comprehension, using some combination of traditional language stimulation and training communication partners to modify their language to be more comprehensible to the patient. In regard to the former, a recent randomized clinical trial of phonological training showed small but significant improvements in comprehension in patients with Wernicke's aphasia (Woodhead et al. 2017). As in many studies of aphasia therapy, patients with more severe impairments showed greater benefit. It is noteworthy that the comparison treatment in that study was donepezil, an acetylcholinesterase inhibitor, which was associated with significant and unexpected declines in comprehension. Partner training targets include reducing syntactic complexity, phrase length, and speaking rate, factors known to affect comprehension in individuals with Wernicke's aphasia (Marshall 2001).

## Cross-References

- ▶ [Aphasia Tests](#)
- ▶ [Fluent Aphasia](#)
- ▶ [Logorrhea](#)
- ▶ [Neologism](#)
- ▶ [Paraphasia](#)
- ▶ [Speech-Language Therapy](#)
- ▶ [Wernicke–Lichtheim Model of Aphasia](#)

## References and Readings

- Binder, J. R. (2017). Current controversies on Wernicke's area and its role in language. *Current Neurology and Neuroscience Reports*, 17(8), 58.
- Buchsbaum, B. R., Baldo, J., Okada, K., Berman, K. F., Dronkers, N., D'Esposito, M., & Hickok, G. (2011).

Conduction aphasia, sensory-motor integration, and phonological short-term memory – An aggregate analysis of lesion and fMRI data. *Brain and Language*, 119(3), 119–128.

- Damasio, H. (2001). Neural basis of language disorders. In R. Chapey (Ed.), *Language intervention strategies in aphasia and related neurogenic communication disorders* (4th ed.). Philadelphia: Lippincott, Williams & Wilkins.
- Goodglass, H. (1993). *Understanding aphasia*. San Diego: Academic.
- Marshall, R. (2001). Management of Wernicke's aphasia: A context-based approach. In R. Chapey (Ed.), *Language intervention strategies in aphasia and related neurogenic communication disorders* (4th ed.). Philadelphia: Lippincott, Williams & Wilkins.
- Mesulam, M. M., Thompson, C. K., Weintraub, S., & Rogalski, E. J. (2015). The Wernicke conundrum and the anatomy of language comprehension in primary progressive aphasia. *Brain*, 138(Pt 8), 2423–2437.
- Wernicke, C. (1874). *Der aphasische Symptomenkomplex*. Breslau: Cohn & Weigert.
- Woodhead, Z. V., Crinion, J., Teki, S., Penny, W., Price, C. J., & Leff, A. P. (2017). Auditory training changes temporal lobe connectivity in 'Wernicke's aphasia': a randomised trial. *Journal of Neurology, Neurosurgery, and Psychiatry*, 88(7), 586–594.

## Wernicke-Korsakoff Syndrome

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## Synonyms

Alcoholic amnestic disorder; Korsakoff amnestic syndrome; Korsakoff (Korsakov) psychosis; Polyneuritis psychosis

## Definition

Wernicke-Korsakoff syndrome (WKS) is a neurological disorder caused by thiamine deficiency. The acute phase of the disorder is called Wernicke encephalopathy and is characterized by confusion, ataxia, and oculomotor palsy. The chronic

phase, called Korsakoff syndrome, is characterized by anterograde and retrograde amnesia and a propensity to confabulation. WKS is most frequently associated with long-standing alcohol consumption, but can also result from other disorders leading to nutritional insufficiency. In chronic alcoholics, thiamine deficiency is caused both by reduced intake and reduced absorption, the former due to inadequate nutrition and the latter caused by alcohol-related intestinal damage. As these symptoms resolve with thiamine replacement therapy, an inability to form new memories and a propensity to produce stories to fill memory gaps (confabulation) emerge. In patients who do not recover fully, Korsakoff syndrome becomes apparent, with a dense amnesia as its most striking symptom (Victor et al. 1989).

## Historical Background

In 1881, C. Wernicke described a neurological syndrome of acute onset characterized by gait disorder, ophthalmoplegia, nystagmus, polyneuropathy in the arms and legs, and a confusional state. Six years later, S.S. Korsakoff published a series of reports in which he described a neurological condition characterized by chronic changes in mental status and memory that often accompanied polyneuropathy. A majority of patients who developed this syndrome had a history of chronic alcohol abuse, but Korsakoff also described the syndrome following prolonged vomiting, typhoid fever, and intestinal obstruction. Not until several years later was it recognized that the two disorders often occur sequentially in the same patients and represent a functional syndrome now generally referred to as WKS.

## Current Knowledge

The incidence of WKS is rare and has been estimated at 0.001% of first psychiatric admissions and at 0.005% in general population studies. Studies at necropsy reveal that 1.7–2.2% of alcoholics develop the syndrome (Victor et al. 1989); this prevalence is higher than that found in clinical

studies, because in many instances diagnosis is not made during life. The overwhelming majority of cases are diagnosed in association with long-standing alcohol abuse. The condition shows no racial predilection, and it is found slightly more often in males than in females. A genetic predisposition has been hypothesized for a minority of heavy alcohol users. Peak onset is age 40–59 years for males and 30–49 years for females. There has been a decline in the incidence of WKS over time as a result of improved nutrition, more effective prophylactic interventions, and changes in cultural habits, in particular the trend for alcohol abuse to be more commonly associated with other drug abuse.

Whereas the causes for WKS are varied, thiamine deficiency is always involved (Witt 1985). The most common etiology in developed countries is chronic alcohol abuse. Several factors contribute to the development of thiamine deficiency in chronic alcoholics, including: 1. Poor nutritional habits with an inconsistent eating pattern; 2. A preference for foods high in carbohydrates and low in vitamins, which leads to a metabolic depletion of stored thiamine that is needed for the breakdown of carbohydrates; and 3. Malabsorption of thiamine in the intestine, caused by alcohol-related damage to the intestinal tract. The confluence of these factors puts an individual at risk for developing Wernicke encephalopathy (Thomson and Marshall 2006).

The acute phase of the Wernicke encephalopathy is characterized by disorientation, confusion, apathy, and the inability to maintain a coherent conversation. This confusional state is often accompanied by oculomotor problems and ataxia. These three symptoms are considered the diagnostic “classic triad” of this syndrome. However, the onset of WKS shows significant variability, which makes its diagnosis a challenge. The three classic symptoms are not always part of the early presentation, and frequently one or more of these symptoms occurs in association with nausea, vomiting, and loss of appetite (Naidoo et al. 1991). More recently, it has been suggested that a diagnosis of Wernicke encephalopathy be based on at least two of the following criteria: (1) dietary deficiencies, (2) oculomotor abnormalities,

(3) cerebellar dysfunction, and (4) altered mental status (Caine et al. 1997).

Thiamine replacement therapy is critical for patients in the early stage of Wernicke encephalopathy in order to prevent the occurrence of fatal midbrain hemorrhages. Prompt intervention will lead to clearing of the confusional state and reversal of the neurological symptoms. However, the cognitive decline cannot be prevented in most cases, and the patient is frequently left with a severe and enduring amnesia that marks the onset of the chronic Korsakoff stage of the syndrome. Changes in personality are also common, including apathy, lack of insight, and lack of concern about personal appearance. A lack of interest in alcohol is also common. A minority of patients do not develop Korsakoff syndrome and are left with minimal cognitive deficits following recovery from Wernicke encephalopathy. The onset of Korsakoff syndrome is in itself quite variable. Some patients present with a more insidious onset, without clinical evidence of antecedent Wernicke encephalopathy (Victor et al. 1989).

The main pathology of WKS consists of punctate lesions in the area of the third ventricle, the fourth ventricle, and the aqueduct – areas known to be very sensitive to thiamine deficiency. There has been much debate about the exact lesion responsible for the amnesia that accompanies WKS. The dorsomedial nucleus of the thalamus, the anterior thalamic nuclei, and the mammillary bodies has each been posited to be the critical location of the lesion responsible for amnesia (Mair et al. 1979). Most patients, however, have combined lesions that interfere with two neural circuits critical for memory: the hippocampal-anterior thalamic system and the perirhinal-dorsomedial thalamic system (Aggleton and Brown 1999).

The main manifestation of Korsakoff syndrome is a disproportionate impairment in memory (amnesia) in comparison to other cognitive functions (Butters and Cermak 1980). Most prominent is anterograde amnesia (see Anterograde Amnesia) for both verbal (names, words, or facts) and nonverbal (faces or figures) information. Even in the face of extensive repetition,

patients have difficulty learning new material, and they forget information to which they were exposed just minutes before. Patients are able to repeat information in the absence of any delay, suggesting that the deficit is not at the level of immediate registration of information. However, there is a notable decline in performance as soon as any distraction occurs. This susceptibility to interference is the principal characteristic of Korsakoff amnesia and is thought to be due to a combination of two factors: (1) failure to fully encode all aspects of incoming information and (2) failure to inhibit competition from irrelevant material at the time of retrieval (Verfaellie and Cermak 1992).

Patients with WKS also suffer from a severe retrograde amnesia (see chapter ► [“Retrograde Amnesia”](#)), characterized by difficulty retrieving personal information and knowledge of public events and facts that were learned prior to the onset of illness. The retrograde amnesia can extend back 25 years or more. A temporal gradient is typically observed with earlier established memories (childhood) being better preserved than more recent memories (Albert et al. 1979; Kopelman 1989). Several causes have been proposed for the retrograde amnesia in WKS, including (a) deficits in strategic control associated with frontal impairment and (b) poorly established memories caused by patients’ ongoing alcohol abuse. A strategic control impairment can result in an inability to plan and initiate systematic memory search. This leads to a retrieval deficit for all memories, regardless of their recency. The fact that recent memories are more affected than remote ones may be due in part to a gradually developing anterograde memory deficit caused by patients’ alcohol abuse and in part to the fact that more recent memory traces are more vulnerable because they are less well established and less frequently rehearsed.

Cognitive impairments in three domains other than memory should be noted. The first domain concerns impairments in planning and problem-solving, deficits linked to impaired frontal executive control. Impaired executive control may also be responsible for patients’ tendency to confabulate when faced with questions they cannot answer, especially in the acute phase of the



disorder. The second domain concerns visuospatial and visual perceptual deficits, which are similar to those seen in chronic alcoholics. Finally, motivation and arousal deficits are also common, and these manifest as apathy, passivity, and lack of initiative (Brokate et al. 2003).

In summary, the neuropsychological profile of WKS is characterized by a profound memory impairment in the presence of preserved intellectual ability and preserved visual confrontation naming. A severe retrograde amnesia accompanies the new learning impairment, along with reduced working memory and impaired executive functions, with a propensity to confabulation and perseveration. More subtle impairments in visuospatial and visuperceptual functioning may also occur. The work-up for possible WKS requires a comprehensive neuropsychological evaluation to establish impaired and preserved areas of cognition. One of the main diagnostic challenges concerns differentiating WKS from alcoholic dementia. To this end, it is important to compare overall post-morbid intellectual abilities with estimates of premorbid functioning. A significant discrepancy between these measures is suggestive of the presence of alcoholic dementia. Language functioning and particularly naming are typically preserved in WKS, but impaired in alcoholic dementia.

The assessment of memory function in WKS generally conforms to that described in the sections on anterograde and retrograde amnesia. Assessment of anterograde memory should also incorporate tests that assess sensitivity to and release from proactive interference. WKS patients are able to repeat information in the absence of interference, but distractions as short as 5–10 s will impair performance. Encoding is often impoverished because of an inability to use organizational strategies, and retrieval is impeded by impairments in the specification and implementation of search strategies. Tests tapping both public and personal remote memories will clarify the extent of the retrograde amnesia (Retrograde Amnesia). Because patients with WKS also have impairments in executive functioning, measures that assess hypothesis formation and set-shifting should also be included.

With respect to treatment, high dosages of thiamine can prevent the development of a severe and chronic Korsakoff syndrome at the acute phase. In the chronic phase, pharmacotherapy has targeted a number of neurotransmitters that are affected by thiamine deprivation, including serotonin and norepinephrine. Pharmaceutical interventions, however, have had limited efficacy to date. Cognitive rehabilitation efforts have also met with little success, primarily because patients with WKS have neither the motivation nor the insight necessary to implement strategies or activities aimed at compensating for their memory loss.

## Cross-References

► [Alcoholic Brain Syndrome](#)

## References and Readings

- Aggleton, J. P., & Brown, M. W. (1999). Episodic memory, amnesia, and the hippocampal-anterior thalamic axis. *Behavioral and Brain Sciences*, *22*, 425–489.
- Albert, M. S., Butters, N., & Levin, J. (1979). Temporal gradients in the retrograde amnesia of patients with alcoholic Korsakoff's disease. *Archives of Neurology*, *36*, 211–216.
- Brokate, B., Hildebrandt, H., & Eling, P. (2003). Frontal lobe dysfunction in Korsakoff's syndrome and chronic alcoholism: Continuity of discontinuity? *Neuropsychology*, *17*, 420–428.
- Butters, N., & Cermak, L. S. (1980). *Alcoholic Korsakoff's syndrome: An information processing approach to amnesia*. New York: Academic Press.
- Caine, D., Halliday, G., Kril, J. J., & Harper, C. G. (1997). Operational criteria for the classification of chronic alcoholics: Identification of Wernicke's encephalopathy. *Journal of Neurology, Neurosurgery and Psychiatry*, *62*, 51–60.
- Kopelman, M. D. (1989). Remote and autobiographical memory, temporal context memory and frontal atrophy in Korsakoff and Alzheimer patients. *Neuropsychologia*, *27*, 437–460.
- Mair, W. G., Warrington, E. K., & Weiskrantz, L. (1979). Memory disorders in Korsakoff's psychosis: A neuropathological and neuropsychological investigation of two cases. *Brain*, *102*, 749–783.
- Naidoo, E. P., Bramdev, A., & Cooper, K. (1991). Wernicke's encephalopathy and alcohol related disease. *Postgraduate Medical Journal*, *67*, 978–981.
- Thomson, A. D., & Marshall, E. J. (2006). The natural history and pathophysiology of Wernicke's



encephalopathy and Korsakoff's psychosis. *Alcohol and Alcoholism*, 41, 151–167.

Verfaellie, M., & Cermak, L. S. (1992). Neuropsychological issues in amnesia. In J. Martinez & R. Kesner (Eds.), *Learning and memory: A biological view* (pp. 467–497). San Diego: Academic Press.

Victor, M., Adams, R. A., & Collins, G. H. (1989). *The Wernicke-Korsakoff syndrome and related neurologic disorders due to alcoholism and malnutrition*. Philadelphia: F.A. Davis.

Witt, E. D. (1985). Neuroanatomical consequences of thiamine deficiency: A comparative analysis. *Alcohol and Alcoholism*, 20(2), 201–221.

## Wernicke–Lichtheim Model of Aphasia

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### Synonyms

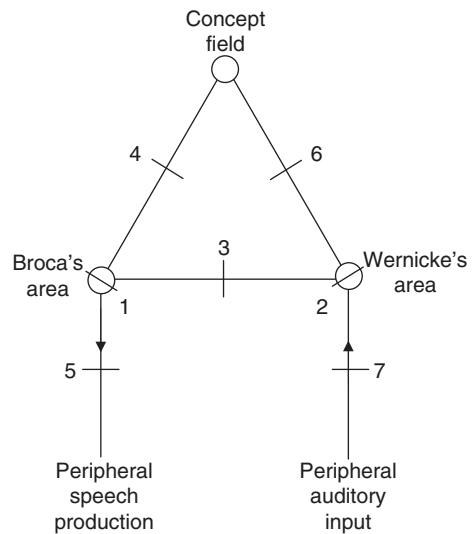
Classical model of aphasia; Connectionist model of aphasia

### Definition

The Wernicke–Lichtheim model provides a framework for classifying seven types of aphasia. The model proposes an explanation for commonly observed language production and comprehension challenges of people with aphasia, such as telegraphic speech, decreased auditory comprehension, anomia, and generation of jargon and paraphasias.

### Historical Background

In 1885, Ludwig Lichtheim published a paper describing a connectionist model of language processing stemming from the work of Pierre Paul Broca and Karl Wernicke. The model, termed either the Wernicke–Lichtheim or classical model of aphasia, was both neuroanatomical and functional in its basis and predicted the communicative consequences of damage to various brain regions. It served as the foundation for classifying



**Wernicke–Lichtheim Model of Aphasia, Fig. 1** Wernicke–Lichtheim Model of Aphasia (based on Lichtheim 1885). Note: Numbers indicate lesion sites associated with each of the proposed types of aphasia: 1 = Broca's aphasia; 2 = Wernicke's aphasia; 3 = Conduction aphasia; 4 = Transcortical motor aphasia; 5 = Subcortical motor aphasia; 6 = Transcortical sensory aphasia; 7 = Subcortical sensory aphasia

the types of aphasia already observed as well as providing a means for predicting aphasia types not yet observed but assumed logically possible on the basis of theoretical considerations. Lichtheim presented the model in the form of a simple diagram identifying three language-related centers within the brain, the neural pathways connecting them, and the possible lesion sites associated with seven distinct types of aphasia (Fig. 1).

In developing the model, Lichtheim endorsed Wernicke's views about the brain having two main centers in the left hemisphere underpinning language processing. Specifically, Lichtheim assumed the left superior temporal gyrus (i.e., Wernicke's area) contained information about the auditory form of words and was instrumental in decoding speech input and guiding speech production; he believed that the left inferior frontal gyrus (i.e., Broca's area) was responsible for storing the motor programs needed to control the vocal apparatus during speech production. Lichtheim specified a third structure – the concept field – as the storage site for information about the multiple

sensory associations forming the basis of word meanings. Lichtheim believed that the concept field was widely distributed throughout the brain. Neural fibers connected the two language centers and the concept field to one another as well as to the peripheral structures responsible for sensing auditory stimuli and for controlling muscle movements involved in speech production.

## Current Knowledge

The Wernicke–Lichtheim model has provided generations of clinicians with a framework for classifying different aphasia syndromes. Characterizations of aphasia syndromes relate to the nature of information flow between the various components of the model. Basic operation of the model for spoken language comprehension begins in Wernicke’s area by accessing phonological properties of words. This information then travels to the concept field for the activation of conceptual representations. Finally, information travels to Broca’s area, where grammatical processing takes place and where instructions are held for word pronunciation. For spoken language production, activations in the concept field stimulate retrieval of phonological representations in Wernicke’s area, followed by the activation of articulatory instructions in Broca’s area.

The Wernicke–Lichtheim model proposes seven aphasia syndromes. Two types of aphasia – Broca’s aphasia and Wernicke’s aphasia – result from lesions to the two main language centers specified in the model. In Broca’s aphasia, language comprehension remains relatively intact, but production is characterized by errors in sequencing and coordination that cause articulatory dysfluency. In Wernicke’s aphasia, comprehension deficits occur along with the production of neologisms and paraphasias. The remaining five types of aphasia result from lesions to pathways connecting the language centers to one another, to the concept field, or to peripheral structures. Conduction aphasia results from a lesion in the fibers connecting Wernicke’s and Broca’s areas; it is characterized by impairments in repetition. Transcortical sensory aphasia results

from disruption between Wernicke’s area and the concept area. Such a lesion causes comprehension challenges, although the ability to repeat remains intact. Similarly, transcortical motor aphasia – resulting from a lesion between the concept area and Broca’s area – again spares repetition skills but is otherwise similar to Broca’s aphasia in symptom features. Subcortical sensory aphasia stems from a lesion between the auditory periphery and Wernicke’s area and leads to auditory agnosia or pure word deafness. With this aphasia type, a person fails to understand spoken words but does not make errors in word production. Finally, subcortical motor aphasia results from a lesion between Broca’s area and the peripheral oral musculature and is alternatively labeled as dysarthria, a motor speech disorder.

## Future Directions

Debate about the accuracy and sufficiency of the Wernicke–Lichtheim model as an explanation of language processing in the brain has persisted for the 135 years since its conception. Evidence of its resilience emerged in 1971, when Benson and Geschwind adopted the basic framework of the model and added three additional aphasia types – global aphasia, mixed transcortical aphasia, and anomia – to form the Boston classification system. This classification system has persisted as a widely used method of categorizing aphasia syndromes, but many professionals lack confidence in the notion that people with acquired damage to the language-dominant hemisphere fit neatly into the identified aphasia syndromes. In particular, advances in neuroimaging technology have allowed researchers to determine that literally hundreds of brain regions exist relevant to language processing and that a myriad of environmental and biological factors influence neural development and modification of these regions. The challenge for future investigators will be to devise an explanation of brain language relations that accounts for dynamic neural changes due to environmental, learning, biological, and physiological influences and that can explain both normal and disordered language processing.

## Cross-References

- ▶ [Aphasia](#)
- ▶ [Aphasia Diagnostic Profiles](#)
- ▶ [Auditory Agnosia](#)
- ▶ [Broca's Aphasia](#)
- ▶ [Conduction Aphasia](#)
- ▶ [Dysarthria](#)
- ▶ [Pure Word Deafness](#)
- ▶ [Subcortical Aphasia](#)
- ▶ [Transcortical Motor Aphasia](#)
- ▶ [Transcortical Sensory Aphasia](#)
- ▶ [Wernicke's Aphasia](#)

## References and Readings

- Benson, D. F., & Geschwind, N. (1971). Aphasia and related cortical disturbances. In A. B. Baker & H. Baker (Eds.), *Clinical neurology*. New York: Harper and Row.
- Brookshire, R. H. (2007). *Introduction to neurogenic communication disorders* (7th ed.). St. Louis: Mosby.
- Caplan, D. (1987). *Neurolinguistics and linguistic aphasiology: An introduction*. New York: Cambridge University Press.
- Lichtheim, L. (1885). On aphasia. *Brain*, 7, 433–484.
- Wernicke, C. (1874). *The aphasic symptom complex*. Breslau: Kohn and Weigart. Reprinted in translation in R. S. Cohen & M. W. Warofsky (Eds.), *Boston studies in the philosophy of science* (Vol. 4). Boston: Reidel.

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## West Syndrome

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## Synonyms

Blitz-Nick-Salaam Krämpfe; Infantile spasms

## Definition

West syndrome (WS) is the classic syndrome associated with infantile spasms. It is characterized by three primary features: (1) spasms, (2) mental deterioration, and (3) hypsarrhythmia on EEG. Severe cognitive impairment is the most striking aspect of this condition, and it is associated with a high rate of Autism Spectrum Disorder (ASD).

## Epidemiology

WS can be cryptogenic or symptomatic, and the causes are numerous. It has been associated with various brain malformations, neurocutaneous disorders, metabolic disorders, brain trauma, neurotoxic exposure, and less commonly, brain tumors. Neurocutaneous disorders (e.g., tuberous sclerosis complex) and conditions involving cerebral dysgenesis (e.g., lissencephaly) are believed to be most commonly associated with WS. About 20% of patients with WS are classified as cryptogenic.

Prevalence rates of WS vary significantly between studies; however, on average, the incidence is about 0.31 per 1,000 births. Gender rates are often equal, though males may be at slightly greater risk. Incidence rates in different regions of the world are similar. Up to 7% of patients with WS have been found to have a family member with WS, and up to 33% have a family member with some form of epilepsy.

## Natural History, Prognostic Factors, and Outcomes

The typical age of onset for infantile spasms is between 3 and 12 months, and 93% of cases are diagnosed prior to 2 years of age. Up to 20% of children with epilepsy onset prior to 2 years of age will be diagnosed with WS. Patients with WS secondary to prenatal complications have an earlier age of onset.

Infantile spasms are often the first manifestation of WS, though they are sometimes preceded

by other seizure types. Developmental regression is frequently the first noticeable feature by caregivers and often prompts further investigation for spasms. Developmental deterioration is commonly first noticed as a loss of eye contact and may include lack of responsiveness, limited smiling, decreased alertness, and/or hypotonia. It is not uncommon for primary care physicians to miss the diagnosis of WS, either by dismissing the observed signs of deterioration or by not examining for spasms, misdiagnosing the spasms, or by not associating the features of WS.

Many patients with WS experience spontaneous recovery within weeks of the onset. Up to 25% can experience spontaneous recovery after a year, and the majority of patients with WS are without spasms by the age of 5 years. Resolution of the hypsarrhythmia pattern on EEG is gradual, though it is uncommon for patients to experience normalization of their EEG. Up to 75% can exhibit on-going, focal electrographic abnormalities, and about 50–60% of patients continue to experience seizures of various types. Patients with symptomatic WS are more likely to have on-going seizures. A large number of patients with WS go on to develop the characteristic seizures and EEG abnormalities associated with Lennox-Gastaut syndrome.

Patients with cryptogenic WS have the most favorable prognosis, both in terms of spasm control and developmental outcome. An estimated 51% of patients with cryptogenic WS experience normal development, whereas only 6% of patients with symptomatic WS have normal development. The absence of additional seizure types has also been associated with better functional outcomes.

The mortality rate in WS has been reported to range as high as 20%, and it usually occurs within the first 2 years from onset. The underlying etiology is believed to be the most common cause of death in patients with WS.

## Neuropsychology and Psychology of WS

Up to 80% of patients with WS experience some degree of cognitive or behavioral impairment that is already present at the beginning stages of the

condition. Cognitive functioning in WS is variable and is often associated with etiological factors. While cognitive impairment is most common in patients with symptomatic WS, the impact of repetitive epileptic spasms and hypsarrhythmia on brain development places patients with cryptogenic WS at risk as well.

Developmental regression in WS is often first observed as a deterioration in visual-input processing. This has been well demonstrated through research documenting deficits in visual attention. Additional research has suggested global deficits in the visual systems of patients with WS that affect visual scanning and acuity. These visual deficits have been hypothesized to be associated with dysfunction in posterior brain regions, possibly due to the emergence of electrographic abnormalities during a critical period of maturation in visual pathways. More recent research has suggested that patients with WS also experience impairment in auditory processing systems. There is some indication that patients with symptomatic WS may be at greater risk for global auditory impairment, while patients with cryptogenic WS may only experience impairments in higher-order aspects of auditory processing. These primary deficits in visual and auditory processing at the onset of the disorder are associated with cognitive impairment and may interfere with later cognitive development.

Cognitive outcome in WS is variable, but it is associated with a high rate of intellectual disability, with rates ranging as high as 80%. Patients with symptomatic WS experience poorer outcomes and are more likely to exhibit global cognitive impairment. Impairment in specific aspects of cognitive functioning has also been reported and is more likely in children with cryptogenic WS. A study completed in 1999 by Gaily et al. revealed mild-to-moderate intellectual disability in 3 out of 15 patients with cryptogenic WS (ages 4–5.9 years). Based upon results of the Wechsler Preschool and Primary Scale of Intelligence (WPPSI), Full Scale IQ ranged from 52 to 126, with one child in the low average range (21st percentile), seven children in the average range (27th–73rd percentile), one child in the high average range (79th percentile), and three children in the superior range (95th–96th percentile). The

patients in the sample demonstrated an average VIQ/PIQ discrepancy of almost 12 points; however, the direction of the split was inconsistent and the magnitude was similar for each. Other than the three children with intellectual disability, five children (33% of the sample) displayed a specific cognitive impairment, as defined by at least two impaired scores on the NEPSY. Most prevalent were impairments in memory and learning, with seven of the total 15 children displaying impairments in both verbal and visual memory. Another five children had impairments in either verbal or visual memory. Follow-up neuropsychological assessments were completed with 7 of the 15 children (ages 6.2–8.5 years). Most of the children displayed similar cognitive profiles, and all eight children with either intellectual disability or specific cognitive impairments at the time of their initial neuropsychological assessments needed special education services.

In a sample of 11 children with tuberous sclerosis complex (TSC), Humphrey et al. (2014) found that overall developmental functioning, as measured by the Early Learning Composite (ELC) of the Mullen Scales of Early Learning, declined significantly in children who developed spasms. Prior to the onset of spasms, functioning was similar in all children, but, following onset, the children with spasms exhibited almost a two standard deviation decline in functioning (mean ELC = 65.9). This decline was accounted for by a lack of developmental progress rather than regression, indicating that the children with spasms continued to acquire skills but at a pace that was less than expected. The severity of the developmental decline was significantly related to the duration of the spasms. The children with TSC who did not develop spasms displayed no change in developmental trajectory and continued to display functioning in the average range for their age (mean ELC = 94.4).

In addition to cognitive impairments, about 28% of children with WS have some sort of behavioral disturbance. This includes diagnoses of Autism Spectrum Disorder (ASD) and features of hyperactivity. A comprehensive review of clinical data by Riikonen and Amnell (1981) suggested a prevalence of ASD in 13.5% of their sample, with

about 58% of those children experiencing a reported discontinuation of symptoms as they got older. Children with WS and TSC have been found to have much higher rates of ASD (up to 58%). Hyperactivity was noted in about 15% of cases and tended to be pervasive throughout the development. Cognitive impairment was more common in children with hyperactivity, and there was a tendency for those children to have more EEG abnormalities in the temporal lobes.

## Evaluation

The diagnosis of WS should occur from completion of a thorough clinical history of developmental progress and the presence of spasms. As indicated above, regression in development often first exhibits as loss of eye contact and can include reduced responsiveness, hypotonia, and limited smiling and/or alertness. Spasms are typically characterized by clusters of brief, bilaterally symmetrical contractions of the axial musculature. They can vary in type and be classified as either flexion or extension spasms. Flexion spasms involve flexion of the neck and all four extremities with adduction of the arms. Extension spasms consist of sudden extension of the neck and extremities, with abduction of the arms. The position of the patient's body often determines the type of spasm that will be exhibited. While flexion spasms are more common, most children display a mix of the two spasm types (about 42%). Spasms typically occur upon or after waking from sleep and rarely occur during sleep.

In addition to obtaining a thorough clinical history, it is recommended that a comprehensive physical examination be conducted to consider the presence of related conditions, such as neurocutaneous disorders (e.g., TSC). EEG, MRI, and laboratory studies typically follow. An underlying etiology can only be clearly identified in about 40% of cases. Patients with cryptogenic WS present with a normal neurological examination, normal imaging, normal development prior to the onset of spasms, and no identifiable etiology. Classifying a patient as either symptomatic or cryptogenic can help identify the expected clinical course.

Routine EEG is typically sufficient to identify the characteristic electrographic abnormalities in WS; however, video-EEG is sometimes necessary for diagnosis and may be the best investigation to assess treatment response. Interictal epileptiform activity and background activity is asynchronous and often described as “chaotic” or in a state of “hypsarrhythmia,” with asymmetrical asynchronous high amplitude disorganized waveforms and multifocal independent discharges. A burst suppression pattern during sleep may be the first observed feature on EEG. The ictal EEG often consists of generalized activity followed by attenuation or abrupt attenuation.

**Treatment**

Treatment of WS has historically been controversial, and there is currently no consensus on the best course of therapy. Multiple medications have been found to be effective, and response to any form of therapy is typically apparent within 1 or 2 weeks. Adrenocorticotrophic hormone (ACTH) therapy has received a significant amount of support in the literature, with rates of spasm control ranging as high as 50–70%. A smaller number of patients may also experience normalization of their EEG. However, it should be highlighted that the side effects of ACTH and corticosteroids can be significant and should be carefully monitored. In cases of symptomatic WS, outcomes are known to be poor and are less likely to be affected by treatment. Because of this, steroid therapy may not be worth the risk.

Vigabatrin has been found to be especially effective at controlling spasms in patients with WS. Response to vigabatrin tends to be better in patients who begin therapy within 12 months of the onset of spasms, and patients with symptomatic WS appear to experience the greatest benefits. This is particularly true for patients with TSC. While treatment lag may be related to outcome in certain populations (e.g., TSC), it does not appear to predict response to therapy.

Surgical treatment of WS, such as focal resection or hemispherotomy, may be an option when

**West Syndrome, Table 1** West Syndrome

West syndrome	
EEG signature	Hypsarrhythmic pattern with asymmetrical asynchronous high amplitude disorganized waveforms and multifocal independent discharges A burst suppression pattern during sleep may be the first observed feature The ictal EEG often consist of generalized activity followed by attenuation or abrupt attenuation
Age of onset	3–12 months (up to 2 years in some cases)
Clinical features	Seizure semiology: (a) Flexor spasms: flexion of the head, trunk, and elevation of the upper and lower extremities (b) Extensor spasms: extension of the head body and limbs (c) Mixed spasms: variations of flexor and extensor spasms Seizure clusters often occur multiple times per day MRI may be normal (cryptogenic) or may demonstrate atrophy or a structural abnormality (symptomatic)
Management	ACTH or vigabatrin (drug of choice for patients with TSC as the underlying cause)
Response to treatment with AEDs	Variable
Seizure freedom	Variable
Functional outcome	Poor (relatively better for cryptogenic)

focal abnormalities can be identified on imaging. Corpus callosotomy may also be considered in some patients, especially those experiencing drop attacks. Limited information, however, is available about the long-term effectiveness of surgery in WS, either in terms of seizure outcome or developmental prognosis (Table 1).

**See Also**

- ▶ [Lennox-Gastaut Syndrome](#)
- ▶ [Tuberous Sclerosis](#)





## Further Reading

- Gaily, E., Appelqvist, K., Kantola-Sorsa, E., Liukkonen, E., Kyyrönen, P., Sarpola, M., et al. (1999). Cognitive deficits after cryptogenic infantile spasms with benign seizure evolution. *Developmental Medicine & Child Neurology*, *41*, 660–664.
- Guzzetta, F. (2006). Cognitive and behavioral outcomes in West syndrome. *Epilepsia*, *47*(Suppl 2), 49–52.
- Humphrey, A., MacLean, C., Ploubidis, G. B., Granader, Y., Clifford, M., Haslop, M., Neville, B. G. R., Yates, J. R. W., Bolton, P. F., & the Tuberous Sclerosis 2000 Study Group. (2014). Intellectual development before and after the onset of infantile spasms: A controlled prospective longitudinal study in tuberous sclerosis. *Epilepsia*, *55* (1), 108–116.
- Millichap, J. J. (2017). West syndrome. In J. M. Pellock, D. R. Nordli Jr, R. Sankar, & J. W. Wheless (Eds.), *Pellock's pediatric epilepsy: Diagnosis and therapy* (4th ed., pp. 433–449). New York: Demos Medical.
- Riikonen, R., & Amnell, G. (1981). Psychiatric disorders in children with earlier infantile spasms. *Developmental Medicine & Child Neurology*, *23*, 747–760.

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## Western Aphasia Battery

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### Synonyms

WAB; WAB-R

### Description

The Western Aphasia Battery is a comprehensive test of language function for individuals with aphasia and aged 18–89 years. Test administration time is 30–60 min, depending on the severity of the patient's aphasia and coexisting deficits (e.g., apraxia, dysarthria). As stated in the test manual, the aim of the WAB is to “evaluate the main clinical aspects of language function, content, fluency, auditory comprehension, repetition, naming, reading, writing, and calculations.” The WAB is designed to test all language modalities: reading, writing, listening, speaking, and gestural

communication. It also tests what Shewan and Kertesz originally referred to as “higher cortical functioning” (1980), including a block design subtest as well as Raven's Coloured Progressive Matrices and tests of drawing and calculation.

A revised edition (WAB-R) was published by Pearson Assessments in 2006. The WAB-R has the same overall structure as the original WAB and also includes supplementary tests of reading and writing of irregular words and nonwords and a bedside screening test that takes approximately 15 min to administer.

### Historical Background

The structure of the WAB was originally based on the clinical and neurolinguistic principles, concepts, and subtests structure developed by Goodglass and Kaplan for *The Assessment of Aphasia and Related Disorders* (1972; currently the Boston Diagnostic Assessment of Aphasia). The WAB first appeared as an unpublished test by Andrew Kertesz in 1968. Over the next 14 years, Kertesz and his colleagues published a series of articles in which the test was described, as well as a summary text, “Aphasia and Associated Disorders: Taxonomy, Localization, and Recovery” (Kertesz 1979). The text contained detailed discussion of the theoretical and clinical foundations of the test and lesion characteristics and other information for the participants in the original standardization sample. At some point after its publication, the test was separated from the text which was no longer in print, and was published as the original Western Aphasia Battery (1982). In 2006, Pearson Assessment reunited the text and test as the Western Aphasia Battery-Revised, adding tests of reading and writing of irregular words and non-words, and updating scoring instructions.

### Psychometric Data

The original WAB standardization sample included 150 adults with aphasia, 21 patients with neurological disorders not affecting the brain (e.g., spinal cord injury), 17 patients with

lesions to the nonlanguage-dominant hemisphere, and 21 adults with lesions that were either diffuse or involved other brain regions (e.g., basal ganglia) (Shewan and Kertesz 1980). A second sample, described in the 1982 test manual, included 31 hospital patients with no history of brain damage or language impairment, 70 adults with non-dominant-hemisphere lesions, 17 adults with diffuse brain lesions, and 117 adults with acute aphasia divided into anomic, conduction, Wernicke's, Broca's, and Global aphasia. The WAB-R does not include new normative data.

In support of the construct validity of the test, scores for the neurologically intact adults differed significantly from those of all aphasia types except for those with anomia, and the non-left-hemisphere lesion groups were not significantly different from each other. Construct validity is also supported by the finding that the WAB has high correlations with other aphasia tests. The WAB meets strict criteria (Cronbach's  $\alpha \geq 0.90$ ) for inter-rater reliability and internal consistency, and seven of nine subtests meet strict criteria for test-retest reliability. Criterion-related validity is supported by the finding that individuals with non-dominant-hemisphere lesions do not score in the aphasic range. The test claims to have predictive validity for communication in daily living, but this has not been tested directly.

The WAB has been translated into many languages, including Spanish (Kertesz et al. 1990), Korean (Kim and Na 2004), Portuguese (Neves Mde et al. 2014), Tagalog (Ozaeta 2012), and Persian (Nilipour et al. 2014). Results of a study comparing WAB English-Zulu translations across experts (Barratt et al. 2012) highlighted the importance of considering not only literal meaning but also cultural interpretations of WAB items when attempting to translate the test into different languages.

## Clinical Uses

The WAB is a comprehensive assessment tool for the classification and diagnosis of aphasia. It is administered by a speech-language pathologist.

The following link leads to a review of the WAB that lists languages other than English into which the WAB has been translated: <https://www.strokengine.ca/indepth/in-depth-review-of-the-wab>.

## Cross-References

- ▶ Aphasia
- ▶ Aphasia Diagnostic Profiles
- ▶ Boston Diagnostic Aphasia Examination
- ▶ Speech-Language Pathology

## References and Readings

- Barratt, J., Khoza-Shangase, K., & Msimang, K. (2012). Speech-language assessment in a linguistically diverse setting: Preliminary exploration of the possible impact of informal 'solutions' within the South African context. *The South African Journal of Communication Disorders*, 59, 34–44.
- Goodglass, H., & Kaplan, E. (1972). *Assessment of aphasia and related disorders*. Philadelphia: Lee & Febiger.
- Goodglass, H., Kaplan, E., & Barresi, B. (2000). *Boston diagnostic aphasia examination* (3rd ed.). San Antonio: The Psychological Corporation.
- Kertesz, A. (1979). *Aphasia and associated disorders: Taxonomy, localization and recovery*. New York: Grune & Stratton.
- Kertesz, A. (1982). *Western Aphasia battery* (1st ed.). San Antonio: The Psychological Corporation.
- Kertesz, A., Pascual-Leone, P., & Pascual-Leone, G. (1990). *Western Aphasia battery en versión y adaptación castellana*. Valencia: Nau Libres.
- Kertesz, A. (2006). *Western Aphasia Battery* (Revised ed.). San Antonio, TX: Pearson Assessment.
- Kim, H., & Na, D. L. (2004). Normative data on the Korean version of the Western Aphasia battery. *Journal of Clinical and Experimental Neuropsychology*, 26(8), 1011–1020.
- Neves Mde, B., Van Borsel, J., Pereira, M. M., & Paradela, E. M. (2014). Cross-cultural adaptation of the western aphasia battery – revised screening test to Brazilian Portuguese: A preliminary study. *Codas*, 26(1), 38–45.
- Nilipour, R., Pourshahbaz, A., & Ghoreyshi, Z. S. (2014). Reliability and validity of bedside version of Persian WAB (P-WAB-1). *Basic Clinical Neuroscience*, 5(4), 253–258.
- Ozaeta, C. (2012). Development of the tagalog version of the Western Aphasia battery-revised. Electronic theses and dissertations. Paper 2230.
- Shewan, C. M., & Kertesz, A. (1980). Reliability and validity characteristics of the Western Aphasia Battery

(WAB). *The Journal of Speech and Hearing Disorders*, 45(3), 308–324.

Spreen, O., & Risser, A. H. (2003). *Assessment of aphasia*. New York: Oxford University Press.

## Whiplash

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## Synonyms

Whiplash-associated disorder

## Definition

Whiplash clinical criterion is an injury to soft tissues of the cervical spine region sustained from rapid acceleration and deceleration forces to the head and neck. This is a common injury resulting from a motor vehicle crash, particularly rear-end collisions, but can also occur as a result of a fall or contact sports. The rapid back-and-forth movement of the head and neck is believed to injure muscles, ligaments, tendons, nerves, and, more rarely, other bony structures such as disks or vertebrae. The 1995 Quebec Task Force on Whiplash is widely cited because it provides a comprehensive discussion of whiplash and its management and outlines a grading system to characterize the heterogeneity and severity among acquired whiplash-associated disorders (WAD) (e.g., WAD range 0–IV) (Spitzer et al. 1995).

## Current Knowledge

### Natural History and Course of Symptoms

The incidence of whiplash injuries has increased dramatically since the beginning of

the twentieth century because of the invention of the automobile and the development of a sophisticated transportation system. The current annual incidence rate of WAD in Europe and North America is at least 300 per 100,000 persons following motor vehicle crashes (Holm et al. 2008).

Whiplash injury results in acute neck pain and other symptoms such as reduced range of motion, headache, dizziness, paresthesia, weakness, visual complaints, and cognitive complaints (Sterner and Gerdle 2004). Not surprisingly, the mechanism of injury of whiplash can also cause a comorbid concussive brain injury, making it difficult at times to know the etiology of a patient's presenting complaints, as whiplash and concussion have many overlapping symptoms. Although whiplash symptoms may resolve within several weeks to months post injury for many, up to 50% of persons with lower-grade injury (WAD I–III) report symptoms 1 year post injury (Carroll et al. 2008).

Although few would deny the validity of acute soft tissue injury associated with whiplash, there is considerable controversy about whether or not chronic whiplash disorders are biologically or psychosocially determined. This is akin to the debate regarding recovery and outcome following concussion. As in the literature for mild brain injury, there is growing evidence that chronic symptoms following whiplash are associated with biological (injury characteristics) and social-psychological (expectations, posttraumatic stress, depression, insurance and compensation system) variables (Kasch et al. 2011; Sterling et al. 2011). Discovering the factors that contribute to outcome is important, particularly given how frequent symptoms persist following whiplash injury (Carroll et al. 2008).

Regarding biological predictors of outcome, higher severity of initial pain and higher grade of injury may correlate with outcome following whiplash (Carroll et al. 2008; Kasch et al. 2008, 2011; Williamson et al. 2008). Psychological factors such as low self-efficacy, acute or post-traumatic stress-related disorders, dysphoria, passive coping, catastrophizing, and excessive pain behaviors (i.e., fear of movement) appear to

negatively influence outcome (Carroll et al. 2008; Spearing et al. 2012; Sterling et al. 2011; Williamson et al. 2008). Reviews by Carroll et al. (2008) and Williams et al. (2007) provide more detailed discussion of chronic whiplash-associated disorders.

### Treatment

Treatment for WADs are typically noninvasive, but medical or surgical techniques can be employed if deemed medically necessary (e.g., high-grade WAD injury) (Conlin et al. 2005). Although rest and immobilization techniques may be used to treat whiplash, studies also support the benefit of mobilization treatments. Providers also frequently use pharmacological interventions, such as muscle relaxers, nonsteroidal inflammatory medications, tricyclics, and other antidepressant agents, and physical therapy focusing on stretching, range of motion, exercise, and posture.

Management of whiplash may also incorporate alternative medicine and behavioral medicine components, such as massage, acupuncture, chiropractic intervention, or psychotherapy (Shearer et al. 2016; Sutton et al. 2016; Wong et al. 2016). Neuropsychological evaluation may be ordered for patients with comorbid concussions during their recovery to help inform return to work or to provide reassurance. Most pain specialists would agree that treatment approaches for whiplash should contain an amalgam of biological and psychosocial principles. Therapies may include education, relaxation, mindfulness, biofeedback, or cognitive-behavioral techniques. Psychological interventions may be helpful acutely (psychoeducation), but may be most important for addressing chronic or disabling symptoms. Just as with postconcussive syndrome, psychotherapeutic techniques for whiplash can (1) help patients with chronic symptoms manage pain perceptions through cognitive, behavioral, or psychophysiological (e.g., biofeedback) interventions; (2) teach patients how expectations can impact functioning, behavior and outcome; (3) improve coping skills; and (4) help patients resume prior psychosocial roles and responsibilities.

### Cross-References

- ▶ Mild Traumatic Brain Injury
- ▶ Postconcussion Syndrome

### References and Readings

- Carroll, L. J., Holm, L. W., Hogg-Johnson, S., Cote, P., Cassidy, J. D., Haldeman, S., . . . Its Associated, Disorders. (2008). Course and prognostic factors for neck pain in whiplash-associated disorders (WAD): Results of the bone and joint decade 2000–2010 task force on neck pain and its associated disorders. *Spine (Phila Pa 1976)*, 33(4 Suppl), S83–S92. <https://doi.org/10.1097/BRS.0b013e3181643eb8>.
- Conlin, A., Bhogal, S., Sequeira, K., & Teasell, R. (2005). Treatment of whiplash-associated disorders – Part I: Noninvasive interventions. *Pain Research and Management*, 10, 21–32.
- Holm, L. W., Carroll, L. J., Cassidy, J. D., Hogg-Johnson, S., Cote, P., Guzman, J., et al. (2008). The burden and determinants of neck pain in whiplash-associated disorders after traffic collisions: Results of the bone and joint decade 2000–2010 task force on neck pain and its associated disorders. *Spine*, 33, S52–S59.
- Kasch, H., Qerama, E., Kongsted, A., Bendix, T., Jensen, T. S., & Bach, F. W. (2008). Clinical assessment of prognostic factors for long-term pain and handicap after whiplash injury: A 1-year prospective study. *European Journal of Neurology*, 15(11), 1222–1230. <https://doi.org/10.1111/j.1468-1331.2008.02301.x>.
- Kasch, H., Qerama, E., Kongsted, A., Bach, F. W., Bendix, T., & Jensen, T. S. (2011). The risk assessment score in acute whiplash injury predicts outcome and reflects biopsychosocial factors. *Spine (Phila Pa 1976)*, 36(25 Suppl), S263–S267. <https://doi.org/10.1097/BRS.0b013e31823881d6>.
- Kwan, O., & Friel, J. (2003). A review and methodologic critique of the literature supporting ‘chronic whiplash syndrome’: Part I – Research articles. *Medical Science Monitor*, 9, RA203–RA215.
- Robinson, J. P., Burwinkle, T., & Turk, D. C. (2007). Perceived and actual memory, concentration, and attention problems after whiplash-associated disorders (grade I and III): Prevalence and predictors. *Archives of Physical Medicine and Rehabilitation*, 88, 774–779.
- Shearer, H. M., Carroll, L. J., Wong, J. J., Cote, P., Varatharajan, S., Southerst, D., . . . Taylor-Vaisey, A. L. (2016). Are psychological interventions effective for the management of neck pain and whiplash-associated disorders? A systematic review by the Ontario Protocol for Traffic Injury Management (OPTIMA) collaboration. *The Spine Journal*, 16(12), 1566–1581. <https://doi.org/10.1016/j.spinee.2015.08.011>.
- Spearing, N. M., Connelly, L. B., Gargett, S., & Sterling, M. (2012). Does injury compensation lead to worse health

- after whiplash? A systematic review. *Pain*, 153(6), 1274–1282. <https://doi.org/10.1016/j.pain.2012.03.007>.
- Spitzer, W. O., Skovron, M. L., Salmi, L. R., Cassidy, J. D., Duranceau, J., Suissa, S., et al. (1995). Scientific monograph of the Quebec task force on whiplash-associated disorders: Redefining whiplash and its management. *Spine*, 20, S1–S20.
- Sterling, M., Carroll, L. J., Kasch, H., Kamper, S. J., & Stemper, B. (2011). Prognosis after whiplash injury: Where to from here? Discussion paper 4. *Spine (Phila Pa 1976)*, 36(25 Suppl), S330–S334. <https://doi.org/10.1097/BRS.0b013e3182388523>.
- Sterner, Y., & Gerdle, B. (2004). Acute and chronic whiplash disorders – A review. *Journal of Rehabilitation Medicine*, 36, 193–210.
- Sutton, D. A., Cote, P., Wong, J. J., Varatharajan, S., Randhawa, K. A., Yu, H., . . . Stupar, M. (2016). Is multimodal care effective for the management of patients with whiplash-associated disorders or neck pain and associated disorders? A systematic review by the Ontario Protocol for Traffic Injury Management (OPTiMa) collaboration. *The Spine Journal*, 16(12), 1541–1565. <https://doi.org/10.1016/j.spinee.2014.06.019>.
- Williams, M., Williamson, E., Gates, S., Lamb, S., & Cooke, M. (2007). A systematic literature review of physical prognostic factors for the development of late whiplash syndrome. *Spine*, 32(E764), E780.
- Williamson, E., Williams, M., Gates, S., & Lamb, S. E. (2008). A systematic literature review of psychological factors and the development of late whiplash syndrome. *Pain*, 135(1–2), 20–30. <https://doi.org/10.1016/j.pain.2007.04.035>.
- Wong, J. J., Shearer, H. M., Mior, S., Jacobs, C., Cote, P., Randhawa, K., . . . Taylor-Vaisey, A. (2016). Are manual therapies, passive physical modalities, or acupuncture effective for the management of patients with whiplash-associated disorders or neck pain and associated disorders? An update of the bone and joint decade task force on neck pain and its associated disorders by the OPTiMa collaboration. *The Spine Journal*, 16(12), 1598–1630. <https://doi.org/10.1016/j.spinee.2015.08.024>.

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## White Matter

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### Definition

White matter is a component of the central nervous system and represents the axons of neuronal cells. The cell bodies of neurons are termed gray matter.

Axons, or white matter carries information to and from neuronal cell bodies. White matter acts as the communication highway upon which sensory and motor information travels throughout the central nervous system. White matter gets its name from the distinctive white coloring of the axons. This coloring is visible during tissue dissection and on imaging and is caused by the deposition of myelin protein around each axon. The presence of myelin is responsible in part for the speedy relay of information between sites in and out of the central nervous system. For example, the pain you feel when placing a hand near a hot surface is relayed to your brain quickly because of the myelinated white matter pathways. This quick relay prevents deforming damage to your hand. White matter is found throughout the brain and spinal cord.

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## Wide Range Achievement Test-4

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### Synonyms

WRAT-4

**Editorial Addendum:** The majority of text in this entry pertains to the WRAT-4. While this edition was in production, the WRAT-5 was published. The new version, which has two parallel forms, was normed on 2355 subjects ages 5 years to 85 years, 11 months; the grade-based sample includes 2150 examinees. The Manual states that the test was updated “. . . to provide greater skills coverage in the Math Computation and Sentence Comprehension subtests, to improve clinical utility, and to simplify administration (p. 3).” Scoring rules were revised, and the discontinuation criteria for untimed tests were reduced to shorten administration time. As with previous versions, both age- and grade-based standard scores can be calculated. A new feature is the “reading composite” score, a combination of Word Reading and Sentence Comprehension. Other new additions are two “discrepancy analyses” (Ability-Achievement Discrepancy and Pattern of Strengths and Weaknesses Discrepancy) that are provided “. . . to aid in the identification of specific learning disabilities (p. 69).” Reported reliability figures are excellent (average split-half and alternate form reliabilities are generally .9 or higher). As this version of the test has only been available for a short time, peer-reviewed studies have not yet appeared in the professional literature.



## Description

The Wide Range Achievement Test is a widely used academic achievement test battery, originally consisting of subtests measuring single-word reading, written spelling, and written mathematics. The current version also contains a “cloze” test of sentence comprehension, an important addition that addresses a substantive criticism leveled at earlier versions. Word Reading and Sentence Comprehension scores are combined to yield a Reading Composite score. The Math Computation portion is timed (15 min), whereas the Sentence Comprehension, Word Reading and Spelling portions are terminated after 7, 10, and 10 consecutive failures, respectively.

The WRAT-4 was normed on a stratified representative sample of over 3000 individuals of ages 5–94 years. Administration requires 15–25 min for ages 5–7 years and 30–45 min for others. Raw scores are converted to age- and grade-based standard scores; percentiles, stanines, normal curve equivalents, and grade equivalents may be derived as well. Between-subtest differences can be examined for significance. This instrument provides brief coverage of basic academic functions; it is easy to administer and score.

## Historical Background

The very early history of the WRAT is unknown (Sarah Jastak, PhD, personal communication, July 27, 2007). Work was initiated by Dr. Joseph Jastak during the 1930s. Dr. Jastak was aware of the Wechsler-Bellevue scales then under development but believed that assessment of cognition should encompass more than intelligence. The first published edition did not appear until 1946. (NB: the WRAT-R Manual refers to a 1936 edition, but no reference is given. The WRAT-4 Manual clarifies the initial publication date.) Revisions appeared in 1984 (WRAT-R), 1993 (WRAT-3), and 2006 (WRAT-4). Later versions offered updated norms, slight changes of content, expanded applicable age ranges, and more sophisticated psychometric data but, surprisingly, provided progressively smaller normative data bases.

Although the WRAT-R had two forms to be used with different ages, the WRAT-3 and WRAT-4 consist of two equivalent forms applicable across the entire age spectrum.

## Psychometric Data

Median internal consistency reliability for individual WRAT-4 subtests ranges from 0.87 to 0.93. Alternate form reliability (immediate retest) falls between 0.82 and 0.90, and average delayed (1 month) retest reliability is 0.84 for the total sample. The Manual reports a variety of studies demonstrating validity through correlations of WRAT-4 subtests with other related measures of achievement or cognitive skill such as the Woodcock-Johnson III and WAIS-III.

## Clinical Uses

The WRAT-4 Manual states that the test offers “. . . a quick, simple, psychometrically sound assessment of important fundamental academic skills.” The Manual cautions that the WRAT-4 alone cannot identify learning or cognitive disorders but can form part of a broader collection of measures used, in conjunction with historical background and behavioral observations, for this purpose.

WRAT-4 offers brevity at the expense of scope. More comprehensive achievement tests such as the Wechsler Individual Achievement Test and the Woodcock-Johnson Tests of Achievement allow evaluation of a broader array of academic skills. Clinicians may prefer the WRAT-4 when achievement testing is a necessary, but not central, part of the purpose of testing – for example, as a component of a broader neuropsychological evaluation. Psychoeducational specialists, however, may gravitate to one of the more specialized and comprehensive achievement batteries.

The Word Reading subtest is often extracted and used as an index of probable premorbid IQ (specifically, verbal IQ) under the assumption that reading is a “robust” skill – that is, resistant to the effects of brain dysfunction. Hence, it could provide a baseline against which to compare current

performance in order to infer extent of decline due to (e.g.) traumatic brain injury or dementia. WRAT-3 has also been used successfully to estimate premorbid functioning in persons with schizophrenia (see Harvey et al. 2006).

Although WRAT-4 is, at the time of writing, too new to have generated much research literature, studies using earlier versions of the WRAT have shown that the test adequately serves to provide estimates of premorbid level for those in the middle range of IQ but less so for those at the extremes (see Johnstone and Wilhelm 1995; Karaken et al. 1995). Caution must nonetheless be exercised when using the test in this way, as WRAT-3 reading scores of persons with traumatic brain injury increased upon 1-year retesting, with larger increases for the more severely injured group (Orme et al. 2004). This calls into question the status of the WRAT as a “hold” test, that is, one that is relatively immune to the impact of brain dysfunction. More recently developed word reading measures such as the NART, AMNART, and WTAR appear to be favored among clinicians and researchers.

## Cross-References

- ▶ [Academic Skills](#)
- ▶ [National Adult Reading Test and Orientation \(Left–Right\)](#)
- ▶ [Reading](#)
- ▶ [Reading Comprehension](#)
- ▶ [Wechsler Individual Achievement Test](#)
- ▶ [Wechsler Test of Adult Reading](#)
- ▶ [Woodcock-Johnson IV](#)

## References and Further Readings

- Harvey, P., Friedman, J., Bowie, C., Reichenberg, A., McGurk, S., Parrella, M., White, L., & Davis, K. (2006). Validity and stability of performance-based estimates of premorbid educational functioning in older patients with schizophrenia. *Journal of Clinical and Experimental Neuropsychology*, 28, 178–192.
- Jastak, S., & Wilkinson, G. (1984). *Wide range achievement test-revised*. Wilmington: Jastak Associates.
- Johnstone, B., & Wilhelm, K. (1995). The longitudinal stability of the WRAT-R reading subtest: Is it an appropriate estimate of premorbid intelligence? *Journal*

*of the International Neuropsychological Society*, 2, 282–285.

- Karaken, D., Gur, R., & Saykin, A. (1995). Reading on the wide range achievement test-revised and parental education as predictors of IQ: Comparison with the Barona formula. *Archives of Clinical Neuropsychology*, 10, 147–151.
- Orme, D., Johnstone, B., Hanks, R., & Novack, T. (2004). The WRAT-3 reading subtest as a measure of premorbid intelligence among persons with brain injury. *Rehabilitation Psychology*, 49, 250–253.
- Wide Range Achievement Test-4. Manual. (2006). Odessa: Psychological Assessment Resources.
- Wilkinson, G. S. (1993). *Wide range achievement test – 3, administration manual*. Wilmington: Wide Range.

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## Wide Range Assessment of Memory and Learning

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## Synonyms

WRAML-2

## Description

The wide range assessment of memory and learning-2 (WRAML-2) is a revised update to the original instrument, published in 1990, that was designed to assess memory in children. The WRAML-2 was designed as a comprehensive tool for the assessment of memory throughout the life span.

The WRAML-2 was developed in an attempt to update the test structure to conform to contemporary ideas about memory. It incorporated a variety of components of memory concepts: primacy and recency effects, immediate and delayed recall, recall of rote learning vs. meaningful learning, visual and verbal memory, sustained attention, short-term memory, recognition vs. retrieval systems, incremental trial learning, semantic vs. acoustic memory errors, working memory, learning curve, and memory decay.

The new version of the WRAML-2 contains 17 subtests to allow for a more detailed analysis of memory. The WRAML-2 Core Battery consists of two Verbal (Story Memory, Verbal Learning), two Visual (Design Memory, Picture Memory), and two Attention/Concentration (Finger Windows, Number-Letter) subtests. Three Index Scores are derived from these six subtests: a Verbal Memory Index, a Visual Memory Index, and an Attention/Concentration Index. Confirmed by factor analysis, the three indexes together form a General Memory Index.

In addition to the core subtests, the WRAML-2 offers 11 optional subtests. From a neuropsychological point these optional tests are important components of memory assessment. A newly added Working Memory Index is comprised of the Symbolic Working Memory and Verbal Working Memory subtests. Both tests are only used for individuals 9 years of age or older. In contrast, Sound Symbol and Sound Symbol Delay Recall subtests retained from the original WRAML are only used for individuals 8 years of age or younger. In addition to the three optional delay memory subtests of the original WRAML (Story Memory Delay Recall, Verbal Learning Delay Recall, and Sound Symbol Delay Recall), the WRAML-2 contains four new recognition subtests: Design Recognition, Picture Recognition, Verbal Recognition, and Story Memory Recognition. Four recognition subtests form the Verbal Recognition Index, the Visual Recognition Index, and a combined General Recognition Index.

Raw scores for each subtest are converted to age-based scaled scores. Summed subtest scaled scores can be converted to index scores. Percentile ranks and stanines are available for all age groups. Index score discrepancy analyses allow the identification of significant differences between subareas of memory ability.

The WRAML-2 was normed using a national stratified sampling technique, controlling for age, sex, race, region, and education. The standardization sample included 1,200 children and adults aged 5–90 years from 22 the US states, with 80 individuals assigned to each of 15 age groups. The sample was highly representative of the US population in terms of gender, ethnicity, and education.

## Historical Background

Both versions of the test were developed by David Sheslow and Wayne Adams. The original WRAML was designed for comprehensive assessment of memory in children and adolescents and had norms for up to 17 years of age. The original WRAML was one of the first clinical instruments for a comprehensive evaluation of memory with a child population. The new WRAML-2 extended assessment age of the WRAML from 5–17 to 5–90 years to allow appropriate follow-up from childhood through adulthood.

The manual states that the goal for the revision was also to enhance the WRAML to better reflect development in the field since its original release, while preserving those parts that had been identified by users as especially valuable. The process of revision and norming of the WRAML-2 was extensive. It started with clinician focus groups in 1998, followed by “item tryout” with a relatively small group of subjects, and a subsequent standardization procedure.

With the total of 17 subtests, the new WRAML-2 provides clinicians with a wider range of optional subtests. Two subtests measuring working memory and four subtests measuring recognition memory were added in the second edition to provide a more dynamic and inclusive view of memory functioning.

## Psychometric Data

Internal consistency reliabilities measured by Cronbach’s coefficient alpha ranged from 0.86 to 0.93 for Core Index scores. Alpha reliabilities for the Core Battery Verbal Memory Index, Visual Memory Index, Attention/Concentration Index, and General Memory Index are 0.92, 0.89, 0.86, and 0.93, respectively. The median alphas ranged from 0.81 to 0.92 for the six core subtests and from 0.40 to 0.92 for the 11 optional subtests.

Test-retest reliability indicated a learning effect from one test to another, with a the median interval of 49 days between administrations. The average gain for the General Memory Index was 6.7 standard score points. Core and optional subtest

gains ranged from 1.6 to 0.2, and 1.8 to 0.3 scaled score points, respectively.

High inter-rater reliability of 0.98 was reported for Design Memory, a test that is assumed to rely most on subjective judgment.

Internal validity was evaluated through item content, intercorrelations among subtests and index scores, exploratory and confirmatory factor analysis, and differential item functioning. Subtest item separation reliabilities ranged from 0.98 to 1.00. Most of the correlations among indexes and subtest scores showed low to moderate relationships. Factor analysis studies supported the internal validity of the test. A confirmatory factor analysis (CFA) of six core subtests demonstrated a three-factor model that was consistent with the hypothesized framework.

The manual reports a variety of studies documenting external validity of the test via correlations with other psychometric tests of memory, including the Wechsler memory scale – III (WMS-III), the Children’s memory scale (CMS), the Test of memory and learning (TOMAL), the California verbal learning test (CVLT), and the California verbal learning test – II (CVLT-II). Moderate convergent and discriminant validities were generally reported between respective indexes of the WRAML-2 and other measures. Moderate correlations were also found between the WRAML-2 and tests of cognitive ability and academic achievement, including the Wechsler adult intelligence scale – III (WAIS-III), the Wechsler intelligence scale for children – III (WISC-III), the Wide range achievement test-3 (WRAT-3), and the Woodcock-Johnson – III tests of achievement (WJ-III). The WRAML-2 manual includes five small clinical studies aimed at investigating differences between clinical groups (Alzheimer’s Disease, Learning Disabilities, Traumatic Brain Injury, Parkinson’s Disease, and Alcohol Abuse) and matched normal controls. Effect sizes are reported for WRAML-2 subtests and indexes for the clinical-control group comparisons.

## Clinical Uses

From a clinical standpoint, the WRAML-2 is a psychometrically solid, flexible instrument for

assessment of memory. It can be used as a comprehensive fixed battery for assessment of memory, or as a flexible screening measure. The manual is well organized and easy to follow. The examiner has a choice of manual scoring or computerized scoring at an additional cost.

The WRAML-2 offers a wider age range for assessment of memory than both the original WRAML (5–17 years), and the Wechsler memory scale-IV (WMS-IV; 16–89 years). Due to the extended age range, the WRAML-2 offers an economic and practical advantage for examiners performing assessments for different age groups. The wide age range of the test allows for follow-up assessments as the child gets older.

The Core Battery of the WRAML-2 takes less than an hour to administer. The test also offers a memory screening option when time limitations prevent a more extensive evaluation. The Screening Memory Index consists of four subtests: Story Memory, Picture Memory, Design Memory, and Verbal Learning. Administration requires approximately 20 min. The Screening Memory Index highly correlates at 0.91 with the General Memory Index.

Compared to the original WRAML, which has been available since 1990, the WRAML-2 has only been available for clinical use and research since 2004. Due to the instrument’s novelty, it has been sparsely mentioned in the literature (Atkinson 2008; Atkinson et al. 2008; Giesbrecht 2008; Hall 2007; Hartman 2007; McAuliffe 2007; Shaver 2005), with the majority of published studies as dissertation abstracts. The authors suggest that due to similarity between the WRAML and WRAML-2 Core subtests, many of the findings from the original test may still have clinical relevance and value. The WRAML-2 manual provides a topically arranged bibliography, with more than 50 clinical and population studies where the original WRAML was used in memory assessment.

## Cross-References

- ▶ [Attention](#)
- ▶ [Memory](#)

- ▶ [Wechsler Memory Scale All Versions](#)
- ▶ [Working Memory](#)

## References and Readings

- Atkinson, T. M. (2008). A cluster analysis of the wide range assessment of memory and learning – Second edition (WRAML-2). *Dissertation Abstracts International: Section A: Humanities and Social Sciences*, 68, 4191.
- Atkinson, T. M., Konold, T. R., & Glutting, J. J. (2008). Patterns of memory: A normative taxonomy of the wide range assessment of memory and learning – Second edition (WRAML-2). *Journal of the International Neuropsychological Society*, 14, 869–877.
- Giesbrecht, B. L. (2008). Evaluating the relationship among clinical measures of working memory. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 69, 2625.
- Hall, S. J. (2007). Comparison between memory test results among persona with Alzheimer’s disease. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 67, 4708.
- Hartman, D. E. (2007). Test review: Wide range assessment of memory and learning – 2 (WRAML-2): WRRedesigned and WReally improved. *Applied Neuropsychology*, 14, 138–140.
- Mcauliffe, P. (2007). Memory and executive functions in adolescents with Lyme disease. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 67, 6089.
- Shaver, G. W. (2005). Distinguishing simulated malingerers from head injury patients and controls on the wide range assessment of memory and learning – Second edition. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 65, 6674.
- Sheslow, D., & Adams, W. (2003). *Wide range assessment of memory and learning – Second edition, administration and technical manual*. Wilmington: Wide Range.

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## Williams Syndrome

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## Synonyms

Williams-Beuren syndrome

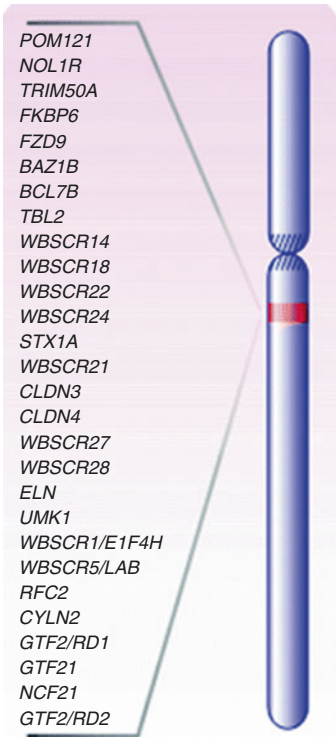
## Short Description or Definition

Williams syndrome (WS) is a neurodevelopmental disorder that is caused by a hemizygous microdeletion of chromosome 7q11.23, with an incidence of 1 in 7,500 live births. (Note that a duplication of the same region results in Duplication 7 syndrome, with a differing phenotype.) A characteristic physical, cognitive, and personality phenotype is present, with some variability. Physical features of WS include dysmorphic facial features, stunted growth, and musculoskeletal problems, as well as connective tissue abnormalities including congenital heart abnormalities. Cognitive features include delayed development, intellectual functioning typically in the range of mild intellectual disability (ID), a marked relative weakness in visuospatial constructive skills, with general language functioning at the level of overall functioning. Personality features include overfriendliness and sociability coupled with social skills difficulties, attention problems, and anxiety.

## Categorization

Classification of WS is based on results of FISH testing for the hemizygous microdeletion of the *Elastin* (ELN) gene. More detailed molecular genetic testing can reveal the breakpoints of the deletion. The genes in the classic WS deletion are illustrated in Fig. 1. There is some variability in the breakpoints, with evidence that people with deletions smaller than the entire WS region often exhibit a partial phenotype, while individuals with longer deletions that include greater telomeric effect, including gene *GTF2I*, have greater deficits in cognitive functioning (Morris et al. 2003). Examinations of the role of specific genes in the WS region also point to a role of ELN in connective tissue abnormalities, including cardiovascular symptoms (Ewart et al. 1993) and LIMK1 (Lim kinase 1) in visuospatial construction difficulties (Frangiskakis et al. 1996). While the facial gestalt associated with WS is universally found, the specific facial features show some variability, as do the medical, cognitive, and behavioral symptoms (for a review, see Mervis and Morris 2007).





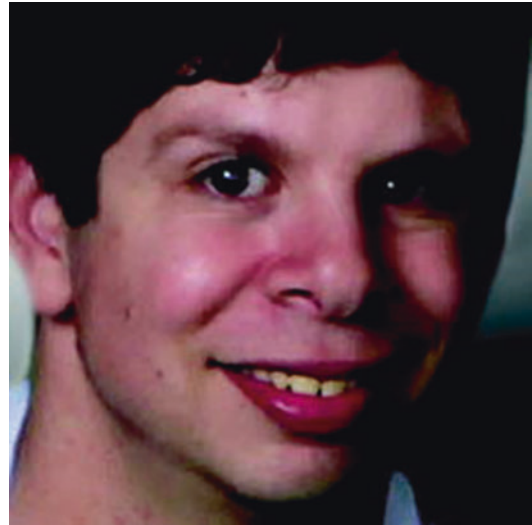
**Williams Syndrome, Fig. 1** Genetic material on the long arm of chromosome 7 deleted in Williams syndrome

## Epidemiology

The incidence of WS is 1 in 7,500 live births (Stromme et al. 2002). The vast majority of cases are spontaneous mutations. The syndrome follows an autosomal dominant pattern of inheritance in that the child of a person with WS has a 50% chance of having WS. The deletions are presumed to occur because of uneven crossover during meiosis.

## Natural History, Prognostic Factors, and Outcomes

WS was first identified in 1961 by the cardiologist Williams of New Zealand who identified a group of patients presenting with similar symptoms indicative of a heart defect, a unique facial appearance, and below-average cognitive functioning (Williams et al. 1961) (Fig. 2). Soon after, a



**Williams Syndrome, Fig. 2** Unique facial appearance of patients with Williams syndrome

second group, in Germany, described patients with similar symptoms (Beuren et al. 1962). The syndrome was subsequently named in honor of the two physicians leading the teams, Williams and Beuren. The genetic etiology of WS was identified because of the high rate of supra-valvular aortic stenosis (SVAS), which was shown to result from a mutation of the ELN gene on chromosome 7q (Ewart et al. 1993).

The facial features associated with WS, identifiable beginning in infancy, are the cardinal feature of the disorder. All children with WS show at least 9 of 17 facial features, which include broad brow, bitemporal narrowing, periorbital fullness, epicanthal folds, stellate or lacy iris pattern, strabismus, short upturned nose, full or bulbous nasal tip, malar hypoplasia, long philtrum, full prominent lips, full cheeks, wide mouth, small jaw, small and widely spaced teeth, dental malocclusion, and prominent earlobes (Morris 2006a). (Note that these features can be seen in other children with developmental delays, but only WS is associated with many of these features together.)

WS is associated with a number of medical difficulties (Morris 2006b). About half of neonates are small based on standard growth curves, and adult stature is generally short. Eighty

percent of children with WS have cardiovascular disease of some kind, with 75% with SVAS and 50% showing peripheral pulmonary stenosis (PPS). Hypotonia is commonly observed beginning in infancy and is associated with feeding difficulties and motor development delays. Older children are typically hypertonic. Fine motor difficulties and balance difficulties are commonly seen. Gastrointestinal difficulties such as gastroesophageal reflux, constipation, and colic are common, as are inguinal hernias. These features result in a 70% rate of failure to thrive in infancy. Hypercalcemia is present in approximately 15% of children and is thought to be underdiagnosed; therefore, Vitamin D supplements are not recommended, and calcium monitoring is recommended for those who have a history of hypercalcemia. Urinary tract malformation is present in about 35% of children with WS, and bladder capacity is typically reduced. There is also an increased rate of endocrine difficulties such as hypothyroidism, early puberty, and diabetes mellitus. Vision difficulties are also common, particularly strabismus. Chronic otitis media is present in about 50% of children with WS. Recent studies indicate an increased incidence of progressive sensorineural hearing loss beginning in the school-age years. Hypersensitivity to sound is commonly seen. The life expectancy in WS is normal unless there is a serious cardiovascular condition.

## Neuropsychology and Psychology of WS

Intellectual disability is present for the vast majority of individuals with WS, with functioning typically in the mild to moderate intellectual disability range, and about 25% of individuals with functioning in the borderline to average range (Mervis et al. 2000). A distinctive cognitive profile is characteristically found across development, with a relative strength in auditory short-term memory, language abilities at or above general cognitive ability levels, and a significant relative weakness in visuospatial constructive skills and fine motor dexterity (Mervis et al. 2000). While language development is typically delayed, fluid

spontaneous language is usually ultimately present (Bellugi et al. 1990). Some have pointed to language abilities as preserved; however, general consensus is that impairments in grammatical abilities, vocabulary, and pragmatics are indeed present in comparison to same-aged peers (Mervis and Morris 2007). Particular difficulties with relational vocabulary are noted. There is evidence of a strong reliance on phonological memory skills in language development (Robinson et al. 2003). In terms of adaptive functioning, personal care and domestic responsibility skills are typically areas of significant relative weakness, with milder delays also found in communication and socialization (Mervis et al. 2001). Executive functioning impairments have also been noted in the areas of set shifting, verbal and spatial working memory, and planning (Hocking et al. 2015; Lanfranchi et al. 2015; Rhodes et al. 2010, 2011a).

A distinctive personality profile is also typically present, characterized by high sociability coupled with anxiety (Klein-Tasman and Mervis 2003). The intense interest in interaction with others is observable even in infancy (Mervis et al. 2003) and continues throughout development (Ng et al. 2014). Despite this sociability, people with WS typically have difficulty establishing sustained friendships with peers (Davies et al. 1997, 1998) and social-communicative deficits are usually present and can overlap with the autism spectrum (Klein-Tasman et al. 2007, 2009; Laws and Bishop 2004). Elevated levels of anxiety, fear, or worry are commonly reported, with specific phobia rates upward of 50%. Anticipatory anxiety is particularly common. Recent research suggests that more anxiety is associated with greater social difficulty (Riby et al. 2014). Significant attention problems are present in up to 65% of people with WS (Dykens 2003; Leyfer et al. 2006). The attention and inhibitory difficulties noted (Greer et al. 2013; Rhodes et al. 2011b) are suggested to be related to difficulty inhibiting social approach (Little et al. 2013). High levels of sound sensitivity have been observed (Jarvinen et al. 2012; Zarchi et al. 2015), with accompanying problem behaviors in response to this sensory sensitivity (Gallo et al. 2008; Janes et al. 2014; Levitin et al. 2005; Riby et al. 2013).

Studies have had mixed findings regarding face-processing abilities and theory of mind development. Performance on tasks of face recognition is similar to mental age-matched controls (Deruelle et al. 1999), though there seems to be a distinctive pattern of activation when processing facial expressions (Doherty-Sneddon et al. 2009; Mills et al. 2000). Findings are mixed about whether facial processing is holistic (Tager-Flusberg et al. 2003) or piecemeal (Gagliardi et al. 2003) and whether those with WS have difficulty with disengaging from faces (Doherty-Sneddon et al. 2009, 2013; Hanley et al. 2013; Riby et al. 2011). Attention bias to emotional or threatening faces is frequently studied (Kirk et al. 2013; McGrath et al. 2016). While early research suggested theory of mind might be stronger than that of children with some other developmental disabilities, more detailed studies have found some difficulties when compared to typically developing peers (Hanley et al. 2013; Tager-Flusberg and Sullivan 2000).

Results of neuroimaging investigations have indicated overall reduction in brain size, significant reductions in cerebral and brainstem volumes, no differences in cerebellar volume, and significant reductions in occipital cortex and thalamic gray matter after controlling for brain size (Reiss et al. 2004). Increased cortical thickness in WS, as well as reduced surface area, suggested to be related to reduced brain volume overall, have also been observed (Green et al. 2016). Significantly larger amygdala, superior temporal gyrus, orbital prefrontal cortex, and dorsal anterior cingulate have also been identified. Reduced levels of functional connectivity in the WS brain have been found (Vega et al. 2015). fMRI investigation found consistent hypoactivation of an area of the parietal cortex commonly associated with visuospatial construction tasks (Meyer-Lindenberg et al. 2004), with structural MRI showing gray matter reduction in the parietooccipital/intraparietal sulcus (Kippenhan et al. 2005). Significantly diminished amygdala activity in response to viewing threatening faces and, conversely, increased activity while viewing threatening scenes, relative to age, sex, and IQ-matched controls, were observed (Meyer-

Lindenberg et al. 2005). Reduced activation in the dorsolateral prefrontal cortex, dorsal anterior cingulate cortex, and striatum in the WS group during a Go/NoGo task has also been found (Mobbs et al. 2007).

## Evaluation

FISH test is typically conducted when WS is suspected and is the accepted method of diagnosis.

## Treatment

There is no cure for WS. Optimal treatment consists of multidisciplinary involvement to address the medical, psychiatric, and neuropsychological sequelae of the disorder. As there is variability in the phenotype, the need for health-care services also varies considerably across individuals. Guidelines regarding medical management are available from the American Academy of Pediatrics (2001) and Morris (2006a, b) and are updated regularly on the Williams Syndrome Association website (For more information, see the Williams Syndrome Association website: [www.williams-syndrome.org](http://www.williams-syndrome.org)). Special education services are generally necessary, with many children educated in mainstream classrooms with support. Phonetic approaches to reading instruction are more beneficial than whole word approaches. Speech, occupational, and physical therapy are typically needed. Psychotropic medication is often used, including stimulants and nonstimulant medication for attention problems, and selective serotonin reuptake inhibitors (SSRIs) for anxiety and mood difficulty. Careful monitoring and consultation with cardiology is strongly recommended, especially for children with a history of cardiovascular involvement. Psychotherapeutic techniques such as cognitive behavioral therapy (CBT) for anxiety and social skills training may prove beneficial to address fears and improve coping skills (Phillips and Klein-Tasman 2009).

Psychoeducational and/or neuropsychological assessment can play an important role in the development of academic intervention strategies for children and adolescents with WS (see Klein-Tasman et al. 2008 for an example). At the very least, administration of intellectual, adaptive behavior, and academic achievement measures designed to assess for the presence of ID, learning disability, and relative strengths and weaknesses is recommended. Examination of receptive and expressive language, visual-spatial skills, and attentional resources may also prove helpful in the development of an Individualized Education Plan. Occupational and speech therapy are often needed. Educating teachers and other service providers about the difficulties commonly experienced in WS may help them to anticipate needs that may arise as the child progresses through school.

## Cross-References

- ▶ [Developmental Delay](#)
- ▶ [Duplication 7 Syndrome](#)
- ▶ [Intellectual Disability](#)
- ▶ [Syndrome](#)

## References and Readings

- American Academy of Pediatrics Committee on Genetics. (2001). Healthcare supervision for children with Williams syndrome. *Pediatrics*, *107*, 1192–1204.
- Bellugi, U., Bihrl, A., Jernigan, T., Trauner, D., & Doherty, S. (1990). Neuropsychological, neurological, and neuro-anatomical profile of Williams syndrome. *American Journal of Medical Genetics, Supplement*, *6*, 115–125.
- Beuren, A. J., Apitz, J., & Harmjanz, D. (1962). Supra-valvular aortic stenosis in association with mental retardation and a certain facial appearance. *Circulation*, *26*, 1235–1240.
- Davies, M., Howlin, P., & Udwin, O. (1997). Independence and adaptive behavior in adults with Williams syndrome. *American Journal of Medical Genetics*, *70*(2), 188–195.
- Davies, M., Udwin, O., & Howlin, P. (1998). Adults with Williams syndrome. Preliminary study of social, emotional and behavioural difficulties. *British Journal of Psychiatry*, *172*, 273–276.
- Deruelle, C., Mancini, J., Livet, M. O., Casse-Perrot, C., & de Schonen, S. (1999). Configural and local processing of faces in children with Williams syndrome. *Brain and Cognition*, *41*(3), 276–298.
- Doherty-Sneddon, G., Riby, D. M., Calderwood, L., & Ainsworth, L. (2009). Stuck on you: Face-to-face arousal and gaze aversion in Williams syndrome. *Cognitive Neuropsychiatry*, *14*(6), 510–523.
- Doherty-Sneddon, G., Whittle, L., & Riby, D. M. (2013). Gaze aversion during social style interactions in autism spectrum disorder and Williams syndrome. *Research in Developmental Disabilities*, *34*(1), 616–626.
- Dykens, E. M. (2003). Anxiety, fears, and phobias in persons with Williams syndrome. *Developmental Neuropsychology*, *23*(1–2), 291–316.
- Ewart, A. K., Morris, C. A., Atkinson, D., Jin, W., Sternes, K., Spallone, P., et al. (1993). Hemizyosity at the elastin locus in a developmental disorder, Williams syndrome. *Nature Genetics*, *5*(1), 11–16.
- Frangiskakis, J. M., Ewart, A. K., Morris, C. A., Mervis, C. B., Bertrand, J., Robinson, B. F., et al. (1996). LIM-kinase1 hemizyosity implicated in impaired visuospatial constructive cognition. *Cell*, *86*(1), 59–69.
- Gagliardi, C., Frigerio, E., Burt, D. M., Cazzaniga, I., Perrett, D. I., & Borgatti, R. (2003). Facial expression recognition in Williams syndrome. *Neuropsychologia*, *41*(6), 733–738.
- Gallo, F. J., Klein-Tasman, B. P., Gaffrey, M., & Curran, P. E. (2008). Expecting the worst: Observations of reactivity to sound in young children with Williams syndrome. *Research in Developmental Disabilities*, *29*, 567–581.
- Green, T., Fierro, K. C., Raman, M. M., Saggari, M., Sheau, K. E., & Reiss, A. L. (2016). Surface-based morphometry reveals distinct cortical thickness and surface area profiles in Williams syndrome. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics: The Official Publication of the International Society of Psychiatric Genetics*. <https://doi.org/10.1002/ajmg.b.32422>.
- Greer, J., Riby, D. M., Hamilton, C., & Riby, L. M. (2013). Attentional lapse and inhibition control in adults with Williams syndrome. *Research in Developmental Disabilities*, *34*(11), 4170–4177.
- Hanley, M., Riby, D. M., Caswell, S., Rooney, S., & Back, E. (2013). Looking and thinking: How individuals with Williams syndrome make judgements about mental states. *Research in Developmental Disabilities*, *34*(12), 4466–4476.
- Hocking, D. R., Reeve, J., & Porter, M. A. (2015). Characterising the profile of everyday executive functioning and relation to IQ in adults with Williams syndrome: Is the BRIEF adult version a valid rating scale? *PLoS One*, *10*(9), e0137628.
- Janes, E., Riby, D. M., & Rodgers, J. (2014). Exploring the prevalence and phenomenology of repetitive behaviours and abnormal sensory processing in children with Williams syndrome. *Journal of Intellectual Disability Research: JIDR*, *58*(8), 746–757.
- Jarvinen, A., Dering, B., Neumann, D., Ng, R., Crivelli, D., Grichanik, M., . . . , & Bellugi, U. (2012). Sensitivity of the autonomic nervous system to visual and auditory affect across social and non-social domains in Williams syndrome.

- Kippenhan, J. S., Olsen, R. K., Mervis, C. B., Morris, C. A., Kohn, P., Lindenberg, A. M., et al. (2005). Genetic contributions to human gyrification: Sulcal morphometry in Williams syndrome. *Journal of Neuroscience*, *25*(34), 7840–7846.
- Kirk, H. E., Hocking, D. R., Riby, D. M., & Cornish, K. M. (2013). Linking social behaviour and anxiety to attention to emotional faces in Williams syndrome. *Research in Developmental Disabilities*, *34*(12), 4608–4616.
- Klein-Tasman, B. P., & Mervis, C. B. (2003). Distinctive personality characteristics of 8-, 9-, and 10-year-olds with Williams syndrome. *Developmental Neuropsychology*, *23*(1–2), 269–290.
- Klein-Tasman, B. P., Mervis, C. B., Lord, C. E., & Phillips, K. D. (2007). Socio-communicative deficits in young children with Williams syndrome: Performance on the autism diagnostic observation schedule. *Child Neuropsychology*, *13*, 444–467.
- Klein-Tasman, B. P., Gallo, F. J., Phillips, K. D., & Fine, K. M. (2008). It helps to know genetic basis: Williams syndrome as an example of cognitive disability. In J. Apps, R. F. Newby, & L. Roberts (Eds.), *Telling stories: Pediatric neuropsychology case studies from the exceptional to the commonplace*. New York: Springer.
- Klein-Tasman, B. P., Phillips, K. D., Lord, C., Mervis, C. B., & Gallo, F. G. (2009). Overlap with the autism spectrum in young children with Williams syndrome. *Journal of Developmental and Behavioral Pediatrics*, *30*, 289–299.
- Janfranchi, S., De Mori, L., Mammarella, I. C., Carretti, B., & Vianello, R. (2015). Spatial-sequential and spatial-simultaneous working memory in individuals with Williams syndrome. *American Journal on Intellectual and Developmental Disabilities*, *120*(3), 193–202.
- Laws, G., & Bishop, D. (2004). Pragmatic language impairment and social deficits in Williams syndrome: A comparison with Down's syndrome and specific language impairment. *International Journal of Language & Communication Disorders*, *39*(1), 45–64.
- Levitin, D. J., Cole, K., Lincoln, A., & Bellugi, U. (2005). Aversion, awareness, and attraction: Investigating claims of hyperacusis in the Williams syndrome phenotype. *Journal of Child Psychology and Psychiatry*, *46*(5), 514–523.
- Leyfer, O. T., Woodruff-Borden, J., Klein-Tasman, B. P., Fricke, J. S., & Mervis, C. B. (2006). Prevalence of psychiatric disorders in 4 to 16-year-olds with Williams syndrome. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, *141B*, 615–622.
- Little, K., Riby, D. M., Janes, E., Clark, F., Fleck, R., & Rodgers, J. (2013). Heterogeneity of social approach behaviour in Williams syndrome: The role of response inhibition. *Research in Developmental Disabilities*, *34*(3), 959–967.
- McGrath, L. M., Oates, J. M., Dai, Y. G., Dodd, H. F., Waxler, J., Clements, C. C., . . . , & Smoller, J. W. (2016). Attention bias to emotional faces varies by IQ and anxiety in Williams syndrome. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-016-2748-y>.
- Mervis, C. B., & Morris, C. A. (2007). Williams syndrome. In M. M. Mazzocco & J. L. Ross (Eds.), *Neurogenetic developmental disorders: Variations of manifestations in childhood*. London: MIT Press.
- Mervis, C. B., Robinson, B. F., Bertrand, J., Morris, C. A., Klein-Tasman, B. P., & Armstrong, S. C. (2000). The Williams syndrome cognitive profile. *Brain and Cognition*, *44*(3), 604–628.
- Mervis, C. B., Klein-Tasman, B. P., & Mastin, M. E. (2001). Adaptive behavior of 4- through 8-year-old children with Williams syndrome. *American Journal of Mental Retardation*, *106*(1), 82–93.
- Mervis, C. B., Morris, C. A., Klein-Tasman, B. P., Bertrand, J., Kwitny, S., Appelbaum, L. G., et al. (2003). Attentional characteristics of infants and toddlers with Williams syndrome during triadic interactions. *Developmental Neuropsychology*, *23*(1–2), 243–268.
- Meyer-Lindenberg, A., Kohn, P., Mervis, C. B., Kippenhan, J. S., Olsen, R. K., Morris, C. A., et al. (2004). Neural basis of genetically determined visuo-spatial construction deficit in Williams syndrome. *Neuron*, *43*(5), 623–631.
- Meyer-Lindenberg, A., Hariri, A. R., Munoz, K. E., Mervis, C. B., Mattay, V. S., Morris, C. A., et al. (2005). Neural correlates of genetically abnormal social cognition in Williams syndrome. *Nature Neuroscience*, *8*(8), 991–993.
- Mills, D. L., Alvarez, T. D., St George, M., Appelbaum, L. G., Bellugi, U., & Neville, H. (2000). III. Electrophysiological studies of face processing in Williams syndrome. *Journal of Cognitive Neuroscience*, *12*(Suppl 1), 47–64.
- Mobbs, D., Eckert, M. A., Mills, D., Korenberg, J., Bellugi, U., Galaburda, A. M., et al. (2007). Frontostriatal dysfunction during response inhibition in Williams syndrome. *Biological Psychiatry*, *62*(3), 256–261.
- Morris, C. A. (2006a). The dysmorphology, genetics, and natural history of Williams-Beuren syndrome. In C. A. Morris, H. M. Lenhoff, & P. P. Wang (Eds.), *Williams-Beuren syndrome: Research, evaluation, and treatment* (pp. 3–17). Baltimore: John Hopkins University Press.
- Morris, C. A. (2006b). Williams syndrome [WS]. *GeneClinics: Medical Genetics Knowledge Base*. Web site: <http://www.geneclinics.org/>. Accessed 12 Jan 2009.
- Morris, C. A., Mervis, C. B., Hobart, H. H., Gregg, R. G., Bertrand, J., Ensing, G. J., et al. (2003). GTF21 hemizygoty implicated in mental retardation in Williams syndrome: Genotype-phenotype analysis of five families with deletions in the Williams syndrome region. *American Journal of Medical Genetics Part A*, *123A*(1), 45–59.
- Ng, R., Jarvinen, A., & Bellugi, U. (2014). Toward a deeper characterization of the social phenotype of Williams syndrome: The association between personality and social drive. *Research in Developmental Disabilities*, *35*(8), 1838–1849.
- Phillips, K. D., & Klein-Tasman, B. P. (2009). Mental health concerns in Williams syndrome: Intervention considerations and illustrations from case examples.



- Journal of Mental Health Research in Intellectual Disabilities*, 2, 110–133.
- Reiss, A. L., Eckert, M. A., Rose, F. E., Karchemskiy, A., Kesler, S., Chang, M., et al. (2004). An experiment of nature: Brain anatomy parallels cognition and behavior in Williams syndrome. *Journal of Neuroscience*, 24(21), 5009–5015.
- Rhodes, S. M., Riby, D. M., Park, J., Fraser, E., & Campbell, L. E. (2010). Executive neuropsychological functioning in individuals with Williams syndrome. *Neuropsychologia*, 48(5), 1216–1226.
- Rhodes, S. M., Riby, D. M., Fraser, E., & Campbell, L. E. (2011a). The extent of working memory deficits associated with Williams syndrome: Exploration of verbal and spatial domains and executively controlled processes. *Brain and Cognition*, 77(2), 208–214.
- Rhodes, S. M., Riby, D. M., Matthews, K., & Coghill, D. R. (2011b). Attention-deficit/hyperactivity disorder and Williams syndrome: Shared behavioral and neuropsychological profiles. *Journal of Clinical and Experimental Neuropsychology*, 33(1), 147–156.
- Riby, D. M., Jones, N., Brown, P. H., Robinson, L. J., Langton, S. R., Bruce, V., & Riby, L. M. (2011). Attention to faces in Williams syndrome. *Journal of Autism and Developmental Disorders*, 41(9), 1228–1239.
- Riby, D. M., Janes, E., & Rodgers, J. (2013). Brief report: Exploring the relationship between sensory processing and repetitive behaviours in Williams syndrome. *Journal of Autism and Developmental Disorders*, 43(2), 478–482.
- Riby, D. M., Hanley, M., Kirk, H., Clark, F., Little, K., Fleck, R., . . . , & Rodgers, J. (2014). The interplay between anxiety and social functioning in Williams syndrome. *Journal of Autism and Developmental Disorders*, 44(5), 1220–1229.
- Robinson, B. F., Mervis, C. B., & Robinson, B. W. (2003). The roles of verbal short-term memory and working memory in the acquisition of grammar by children with Williams syndrome. *Developmental Neuropsychology*, 23(1–2), 13–31.
- Stromme, P., Bjornstad, P. G., & Ramstad, K. (2002). Prevalence estimation of Williams syndrome. *Journal of Child Neurology*, 17(4), 269–271.
- Tager-Flusberg, H., & Sullivan, K. (2000). A componential view of theory of mind: Evidence from Williams syndrome. *Cognition*, 76(1), 59–90.
- Tager-Flusberg, H., Plesa-Skwerer, D., Faja, S., & Joseph, R. M. (2003). People with Williams syndrome process faces holistically. *Cognition*, 89(1), 11–24.
- Udwin, O., & Yule, W. (1991). A cognitive and behavioural phenotype in Williams syndrome. *Journal of Clinical and Experimental Neuropsychology*, 13(2), 232–244.
- Vega, J. N., Hohman, T. J., Pryweller, J. R., Dykens, E. M., & Thornton-Wells, T. A. (2015). Resting-state functional connectivity in individuals with down syndrome and Williams syndrome compared with typically developing controls. *Brain Connectivity*, 5(8), 461–475.
- Williams, J. C. P., Barrat-Boyes, B. G., & Lowe, J. B. (1961). Supraaortic stenosis. *Circulation*, 24, 1311–1318.
- Zarchi, O., Avni, C., Attias, J., Frisch, A., Carmel, M., Michaelovsky, E., . . . , & Gothelf, D. (2015). Hyperactive auditory processing in Williams syndrome: Evidence from auditory evoked potentials. *Psychophysiology*, 52(6), 782–789.

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## Wilson's Disease

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## Synonyms

Hepatolenticular degeneration

## Definition

Wilson's disease is a disorder of copper metabolism absence or dysfunction of a copper transporting P-type ATPase. The chromosome 13 autosomal recessive mutation causes a defect in copper metabolism, causing its buildup in the liver, brain, and eye.

## Current Knowledge

Numerous cognitive, psychiatric, and movement disorders may be seen with this disease. Wilson's disease neurologic manifestations include tremor, dystonia, dysmetria, dysrhythmia, ataxia, and dysarthria. Tremor is typically a proximal or "wing beating tremor." Testing for Wilson's disease involves blood and urine testing of liver function tests, ceruloplasmin, and serum and urine copper. Liver function abnormalities in the face of low ceruloplasmin and high urinary copper levels should prompt a diagnosis of Wilson's disease. Slit-lamp examination may reveal Kayser-Fleischer rings. MRI often shows abnormal T2 signal in the basal ganglia. The typical patient presents in early or middle adulthood with hepatic, psychiatric, and/or neurologic symptoms.

## Cross-References

► [Dystonia](#)

## References and Readings

Pfeiffer, R. F. (2004). Wilson's disease. In R. L. Watts & W. C. Koller (Eds.), *Movement disorders* (2nd ed., pp. 779–798). New York: McGraw-Hill.

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## Wisconsin Card Sorting Test

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## Synonyms

WCST

## Description

The Wisconsin card sorting test (WCST) is a measure of executive functioning that assesses one's ability to develop abstract concepts (abstract reasoning) and shift between sets (mental or cognitive flexibility and problem solving). The WCST can be administered in its original paper-and-pencil format, utilizing a deck of 128 preordered stimulus cards, a 64-card version, or a computer-based version of either 128 or 64 stimuli. The subjects/patients are given a set of four reference cards, each differing from the other in terms of three categories: color (red, blue, green, or yellow), shape (triangle, circle, square, or cross), or number (1, 2, 3, or 4). The subjects/patients are simply asked to match the stimulus cards to the reference cards without further direction as to how they are to be matched. The administrator or computer

provides simple feedback of “correct” or “incorrect” based on the predetermined category. Based on the feedback, the subject/patient places the next card. During administration, the rules for the correct categories are changed after the subject/patient correctly places ten cards per category. Subjects/patients are not informed of the correct sorting rule, nor are they informed that the rule will change. The task continues until the subject/patient has sorted all the cards. The number of correct and incorrect responses it takes for the subject/patient to learn the new rules and the mistakes made during this learning process are scored. Among other outcomes, the WCST measures perseveration, or the tendency to respond to a stimulus in the same way despite a shift or change in the rule governing response to that stimulus. Both the 128- and 64-item versions generate a variety of scores, including trials administered, total correct, total errors, perseverative responses, perseverative errors, non-perseverative errors, conceptual-level responses, categories completed, trials to complete first category, failures to maintain set, and a learning to learn score. A subset of these scores can also be converted to percentiles, standard, and T-scores. The 128-card version takes between 20 and 30 min to administer and 10 and 20 min to score by hand; the 64-card version takes between 10 and 15 min to administer and 10 min to score by hand, and while the computer versions take similar time to administer, they are scored instantaneously.

## Historical Background

Based on the systematic figure configurations of David A. Grant, PhD, and Esta A. Berg, PhD, from 1948, the standardized administration and scoring methods for the WCST were developed by Heaton (1981). The method was revised and expanded to include normative data by Heaton et al. (1993). The measure was initially used to evaluate perseveration and abstract thinking (Berg 1948; Grant and Berg 1948). However, the WCST has become widely used as a measure of frontal lobe functioning including strategic planning, organized searching, utilizing environmental feedback to shift cognitive sets, directing

behavior toward achieving a goal, and modulating impulsive responding.

## Psychometric Data

Initial psychometric data for the 128-card version were provided by Chelune and Baer (1986), Chelune and Thompson (1987), and Welsh et al. (1991). However, the normative sample, derived from the responses of 899 nondisabled children, adolescents, and adults, ranging in age from 6.5 to 89 years of age, and validity data were not published by Heaton and colleagues until 1993. The normative data include standard scores adjusted for age and education. Clinicians and researchers were concerned with using the norms for the 128-card version when administering and interpreting the 64-card version.

Until normative data for the 64-card version was published by Kongs et al. (2000), limited psychometric data existed for the shorter version. Both versions of the WCST have been shown to have excellent inter-scorer and intra-scorer reliability. Concurrent validity has also been examined in a number of clinical populations (e.g., schizophrenia, brain injury, dementia, etc.). Of note, issues with practice effects over multiple administrations can confound subsequent administrations of the WCST and have been widely reported. Also, another issue with the measurement, reported by researchers and clinicians, involves the difficulty of interpreting failures on the WCST, given the large number of cognitive processes that need to be intact to succeed on this task. Clinically successful performance depends on subjects/patients having normal or corrected vision, the ability to benefit from feedback, and sufficiently intact short-term memory to keep the correct category in mind over ten or more trials. Due to these issues, the WCST is recommended as a supplement to a comprehensive neuropsychological battery of tests and is not recommended as a stand-alone diagnostic measure. Further, when used in subjects/patients with known brain dysfunction, the reliability and validity are dependent upon the skill and training of the administrator/interpreter. In order to address the difficulty of

interpreting failures and patient frustration with the length of the test, (Nelson 1976) developed a modified version of the WCST (M-WCST) for use with older patients and those with more severe deficits. The M-WCST is a shorter test in which the subject/patient receives more feedback regarding the shift in set.

## Clinical Uses

The WCST has been used by a wide variety of clinicians and researchers in a wide variety of specialties to assess cognitive flexibility. For example, the WCST is used by clinical psychologists, school psychologists, neuropsychologists, neurologists, and psychiatrists. The WCST has been used to assess the development of frontal lobe function in children and frontal lobe dysfunction in adults with dementia, schizophrenia, acquired and traumatic brain injury, and stroke and persons with alcohol dependence. Difficulty has been noted in scoring the WCST, and it is recommended that administrators/interpreters are well trained and familiar with test prior to administration/scoring. In order to administer, score, and interpret the WCST, clinicians and researchers are required to hold an advanced degree in Psychology or a closely related field, have satisfactorily completed coursework in psychological testing, or hold a license or certification from an agency that requires appropriate training and experience in the ethical and competent use of psychological tests.

## Cross-References

- ▶ [Frontal Lobe](#)
- ▶ [Neuropsychological Assessment Battery](#)

## References and Readings

- Berg, E. A. (1948). A simple objective technique for measuring flexibility in thinking. *The Journal of General Psychology*, 39, 15–22.
- Chelune, G. J., & Baer, R. A. (1986). Developmental norms for the Wisconsin card sorting test. *Journal of*

- Clinical and Experimental Neuropsychology*, 8, 219–228.
- Chelune, G. J., & Thompson, L. L. (1987). Evaluation of the general sensitivity of the Wisconsin card sorting test among younger and older children. *Developmental Neuropsychology*, 3, 81–89.
- Grant, D. A., & Berg, E. A. (1948). A behavioral analysis of degree of reinforcement and ease of shifting to new responses in a Weigl-type card-sorting problem. *Journal of Experimental Psychology*, 38, 404–411.
- Heaton, R. K. (1981). *A manual for the Wisconsin card sorting task*. Odessa: Psychological Assessment Resources, Inc.
- Heaton, R. K., Chelune, G. J., Talley, J. L., Kay, G. G., & Curtiss, G. (1993). *Wisconsin card sorting test manual – Revised and expanded*. Odessa: Psychological Assessment Resources.
- Kongs, S. K., Thompson, L. L., Iverson, G. L., & Heaton, R. K. (2000). *Wisconsin card sorting test – 64 card computerized version*. Odessa: Psychological Assessment Resources.
- Nelson, H. E. (1976). A modified card sorting test sensitive to frontal lobe defects. *Cortex*, 12(4), 313–324.
- Tchanturia, K., Davies, H., Roberts, M., Harrison, A., Nakazato, M., Schmidt, U., Treasure, J., & Morris, R. (2012). Poor cognitive flexibility in eating disorders: Examining the evidence using the Wisconsin card sorting task. *PLoS One*, 7(1), e28331.
- Welsh, M. C., Pennington, B. F., & Groisser, D. B. (1991). A normative-developmental study of executive function: A window on prefrontal function in children. *Developmental Neuropsychology*, 7, 131–149.

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## Witzelsucht

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### Definition

Witzelsucht (vit'sel-zoocht) is a term used to describe a patient's tendency to tell inappropriate jokes and stories at incongruous moments. It is derived from the German words for pun or joke (*witzel*) and addiction (*sucht*). The term is sometimes used more broadly to describe silly, facetious, or euphoric affect, but this is more accurately termed “moria.” The patient is typically unconcerned with the inappropriateness and incongruity of the behavior. It is most commonly seen in

patients with frontal lobe lesions, particularly right frontal, and in frontotemporal dementia (FTD).

## References and Readings

- Mendez, M. F. (2005). Moria and Witzelsucht from frontotemporal dementia. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 17(4), 29–430.
- Vardi, J., Finkelstein, Y., Zlotogorski, Z., & Hod, I. (1994). L'homme qui rit: Inappropriate laughter and release phenomena of the frontal subdominant lobe. *Behavioral Medicine*, 20(1), 1994.

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## Wolf Motor Function Test

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## Synonyms

WMFT

## Description

The Wolf Motor Function Test (WMFT) quantifies upper extremity (UE) motor ability through timed and functional tasks (Wolf et al. 1995). When administering the WMFT, the examiner should test the less-affected UE followed by the most affected side. Items should be performed as quickly as possible; a maximum of 120 s per task is allowed (Wolf et al. 2005). The first 6 items involve timed functional tasks, items 7–14 are measures of strength, and the remaining 9 items consist of analyzing movement quality when completing various tasks (Wolf et al. 1995; Whitall et al. 2006).

Examples of WMFT items include:

- Placing forearm to table and on a box by abduction at the shoulder

- Extending the elbow by reaching across the table and with and without a 1 lb weight
- Putting the involved hand on a table and a box
- Lifting a weight into a box and pulling a weight across a table using elbow flexion
- Lifting and bringing can/cylinder close to lips and lifting a pencil and paper clip using different grasp types
- Stacking checkers, flipping cards, turning a key, folding a towel, and lifting and carrying a basket
- Gripping strength

Performance is rated on a scale (Wolf et al. 2005) from 1 (“does not attempt with UE being tested”) to 6 (“appears normal”). Lower scores indicate lower functioning levels. Since a maximum of 120 s is allocated to each item, it is estimated to take approximately 30 min with additional time for measuring grip strength (item 14). Equipment needed for testing includes a table and chair, two boxes, free weights, a can, a pencil, a bedside table, a paper clip, checkers, cards, a key with a lock, a towel, a basket, and a dynamometer for measuring hand grip strength.

The WMFT has 21 items in its original version and 17 items in the modified version. The latter is most widely used and allows assessment of clients with all levels of stroke severity. The WMFT is suitable to use with clients who have had a stroke or who have UE functional deficits. It should not be used with clients who have severe upper limb spasticity or with upper limb amputees.

## Current Knowledge

The original version of the WMFT was developed by Wolf et al. (1989) to examine the effects of constraint-induced movement therapy in clients with mild to moderate stroke and traumatic brain injury. In 1999, a graded WMFT was developed by Morris et al. (2001), to assess the motor abilities of patients who were functioning at a lower level.

## Reliability

Excellent internal consistency of the WMFT was reported by Morris et al. (2001) on 24 clients

with stroke ( $\alpha = 0.92$ ). The test-retest reliability of the WMFT was analyzed on 24 clients with stroke who were reassessed within a 2-week interval. The test-retest reliability was excellent for both functional ability and performance tests ( $r = 0.95$  and  $0.90$ , respectively). Another study of test-retest reliability was performed by Whitall et al. (2006) in 66 clients with stroke. Participants were reassessed within a 2-week interval by the same rater and under the same conditions; the test-retest reliability was excellent (ICC = 0.97).

Morris et al. (2001) evaluated the inter-rater reliability of the WMFT in 24 clients with stroke. Videotaped valuations by a physical therapist (PT) were rated by two PTs and one occupational therapist. Inter-rater reliability was excellent for both functional ability and performance tests (ICC = 0.93 and 0.99, respectively). Later, Whitall et al. (2006) estimated the inter-rater reliability of the WMFT in ten clients with stroke. The assessment of functional ability was videotaped and rated by three different raters. Inter-rater reliability was excellent (ICC = 0.99).

## Validity

No studies have reported the predictive validity of the WMFT. However, Wolf et al. (2001) examined the concurrent validity of the WMFT by comparing it to the Upper Extremity Fugl-Meyer Assessment as the gold standard in 19 clients with stroke. Adequate correlations were found between the WMFT and the Upper Extremity Fugl-Meyer Assessment ( $r = -0.57$ ). Whitall et al. (2006) also assessed the concurrent validity by comparing the WMFT to the Upper Extremity Fugl-Meyer Assessment as the gold standard in 66 clients with stroke. Correlations between the functional ability test of the WMFT and the Upper Extremity Fugl-Meyer Assessment were excellent ( $r = -0.88$ ).

Wolf et al. (2001) evaluated content validity based on whether the WMFT distinguished between individuals with impairment secondary to stroke from those without impairment. Known group's validity showed that the WMFT scores for the dominant and the non-dominant hand of individuals without



impairment were significantly higher when compared to the most and to the least affected upper limbs of clients with stroke.

### Change Scores

For use in assessing the effect of interventions, the standard error of measurement (SEM) and minimal detectable change (MDC) have been established. Based upon an SEM of 0.2 s for the WMFT Performance Time score, an MDC95 of 0.7 s was determined. Therefore, if a person experiences a change in the WMFT Performance Time score of greater than or equal to 0.7 s, the evaluator can be 95% confident that the change is not the result of chance. Individual task items have higher variability. The WMFT Functional Ability scale SEM and MDC95 is 0.1 points (Fritz et al. 2009).

### Cross-References

- ▶ [Fugl-Meyer Assessment of Sensorimotor Impairment](#)
- ▶ [Motor Assessment Scale](#)
- ▶ [Sensorimotor Assessment](#)

### References

- Barreca, S. R., Gowland, C. K., Stratford, P. W., et al. (2004). Development of the Chedoke arm and hand activity inventory: Theoretical constructs, item generation, and selection. *Topics in Stroke Rehabilitation*, 11(4), 31–42.
- Fritz, S. L., Blanton, S., Uswatte, G., Taub, E., & Wolf, S. L. (2009). Minimal detectable change scores for the Wolf Motor Function Test. *Neurorehabilitation and Neural Repair*, 23, 662–667.
- Fugl-Meyer, A. R., Jääskö, L., Leyman, I., Olsson, S., & Steglind, S. (1975). The post-stroke hemiplegic patient 1. A method for evaluation of physical performance. *Scandinavian Journal of Rehabilitation Medicine*, 7, 13–31.
- Lyle, R. C. (1981). A performance test for assessment of upper limb function in physical rehabilitation treatment and research. *International Journal of Rehabilitation and Research*, 4, 483–492.
- Morris, D., Uswatte, G., Crago, J., Cook, E., & Taub, E. (2001). The reliability of the Wolf Motor Function Test for assessing upper extremity function after stroke. *Archives of Physical Medicine and Rehabilitation*, 82, 750–755.
- Whitall, J., Savin, D., Harris-Love, M., & Waller, S. (2006). Psychometric properties of a modified wolf motor function test for people with mild and moderate upper extremity hemiparesis. *Archives of Physical Medicine and Rehabilitation*, 82, 750–755.
- Wolf, S., Catlin, P., Ellis, M., Archer, A., Morgan, B., & Piacentino, A. (2001). Assessing Wolf Motor Function Test as outcome measure for research in patients after stroke. *Stroke*, 32, 1635–1639.
- Wolf, S., Thompson, P., Morris, D., Rose, D., Winstein, C., Taub, E., et al. (2005). The EXCITE trial: Attributes of the Wolf Motor Function Test in patients with subacute stroke. *Neurorehabilitation and Neural Repair*, 19, 194–205.

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## Wonderlic Personnel Test

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### Synonyms

WPT

### Definition

The Wonderlic Personnel Test (WPT) is a standardized, self-administered assessment of general mental ability that is frequently used in industrial and business settings as an aptitude test for prospective employees.

### Historical Background

The WPT was initially introduced in 1937 by Eldon F. Wonderlic as a short-form test of general mental ability. Development of the WPT aimed to efficiently employ the lowest number of questions possible that would reliably characterize an individual's cognitive abilities. The WPT was used by the US Military in World War II to aid in the selection and placement of potential military recruits. Normative data for the WPT was first published in 1950, followed by regular publication of expanded normative data categorized by

job title and demographic information. The WPT is also well known for its use by the National Football League to assess prospective players in the predraft season ([www.wonderlic.com](http://www.wonderlic.com)).

## Current Knowledge

The WPT comprises 50 items that fall into three broad categories: verbal reasoning, numerical reasoning, and spatial reasoning. Individual test items measure cognitive skills that include abstract reasoning and problem-solving, vocabulary, and visuospatial abilities. Test items are presented with increasing difficulty levels. The total score is the number of correct items produced in a 12-min test administration time period (Wonderlic 1992). In the context of a comprehensive neuropsychological assessment, the WPT may provide a useful and efficient estimate of an individual's global cognitive abilities. The WPT may also be used as a screening instrument to facilitate the future administration of a more in-depth neuropsychological evaluation.

Measures of reliability for the WPT range from 0.82 to 0.94. There are alternate forms of the WPT, and their reliability ranges from 0.73 to 0.95 (Grubb et al. 2004). The WPT is offered in several different languages and is suitable for administration in a group format. The Wonderlic Personnel Test-Revised (WPT-R) was published in 2007. This version includes revised normative data and test items. It can be administered on a computer or in a paper-and-pencil format. The WPT web site ([www.wonderlic.com](http://www.wonderlic.com)) is a useful resource for additional information on various forms and applications of the WPT.

## Cross-References

► [Personality Assessment Inventory](#)

## References and Readings

Grubb, W. L., III, Whetzel, D. L., & McDaniel, M. A. (2004). General mental ability tests in industry. In

M. Hersen (Ed.), *Comprehensive handbook of psychological assessment*. New York: Wiley.

Wonderlic, E. F. (1992). *Manual of the wonderlic personnel test and scholastic level exam II*. Libertyville: Wonderlic Personnel Test.

Wonderlic, E. F. (n.d.) <http://www.wonderlic.com>. Accessed 3 May 2016.

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## Woodcock-Johnson IV

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## Synonyms

WJ IV; *Woodcock-Johnson IV*

## Description

The *Woodcock-Johnson IV* (WJ IV; Schrank et al. 2014b) is the most recent iteration of the well-known Woodcock-Johnson battery of tests. Unlike previous versions which included a cognitive battery and an achievement battery, the WJ IV consists of *three* co-normed assessment batteries: the *Woodcock-Johnson IV Tests of Cognitive Abilities* (WJ IV COG; Schrank et al. 2014a), the *Woodcock-Johnson Tests of Achievement* (WJ IV ACH; Schrank et al. 2014c), and the *Woodcock-Johnson Test of Oral Language* (WJ IV OL; Schrank et al. 2014d). Each of these assessment batteries can be used independently or in combination with one or both of the other batteries.

The norming sample for the WJ IV is comprised of 7,416 participants ranging from age 2 to 90 who were demographically representative of the United States population (Mather and Wendling 2014b; McGrew et al. 2014). An online scoring system is used to convert raw scores to standard scores, percentiles, age and grade equivalents, etc. based on test norms (Schrank and Dailey 2014).

The WJ IV COG is based in the Cattell-Horn-Carroll (CHC) theory of cognitive abilities

(Carroll 1993, 2005; McGrew 2005, 2009); in addition to measuring CHC broad abilities, the WJ IV incorporates measures of more specific, narrow abilities. Although the WJ IV COG consists of 18 tests, these tests make up two batteries: the Standard Battery, which is comprised of Tests 1–10, and the Extended Battery, which is comprised of Tests 11–18. According to information included in the WJ IV Technical Manual (McGrew et al. 2014), tests included in the WJ IV COG Extended Battery can be used either in combination with tests from the Standard Battery or independently. Depending on the tests the examiner selects and administers, a variety of different composite or clusters can be obtained. For example, scores from Tests 1–7 make up the General Intellectual Ability (GIA) composite. The Brief Intellectual Ability composite and *Gf-Gc* Composite are overall composite scores comprised of fewer tests. Other combinations of tests yield scores for a variety of CHC broad ability clusters (i.e., Comprehension-Knowledge [*Gc*], Fluid Reasoning [*Gf*], Short-term Working Memory [*Gwm*], Cognitive Processing Speed [*Gs*], Auditory Processing [*Ga*], Long-term Processing [*Glr*], and Visual Processing [*Gv*]), narrow ability clusters (e.g., Quantitative Reasoning [*RQ*], Auditory Memory Span [*MS*], Number Facility [*N*], Perceptual Speed [*P*], Vocabulary [*VL/LD*]), and a Cognitive Efficiency cluster (McGrew et al. 2014).

The WJ IV ACH includes 20 tests. Tests 1–11 make up the Standard Battery (which has three forms or versions), and Tests 12–20 make up the Extended Battery. Similar to the WJ IV COG, a variety of cluster scores can be obtained depending on the tests the examiner chooses to administer. Tests 1–11 yield a Broad Achievement Cluster, while other combinations of tests form a variety of clusters related to reading (i.e., Reading, Broad Reading, Basic Reading Skills, Reading Comprehension, Reading Fluency, Reading Rate.), mathematics (i.e., Mathematics, Broad Mathematics, Math Calculation Skills, Math Problem Solving), and writing skills (i.e., Written Language, Broad Written Language, Basic Writing Skills, Written Expression; McGrew et al. 2014). In addition, cross-domain clusters are

available (e.g., Academic Skills, Academic Fluency, Academic Applications, Academic Knowledge, Phoneme-Grapheme Knowledge; McGrew et al. 2014).

The WJ IV OL is made up of 12 tests (Tests 10–12 are Spanish versions of Tests 1, 2, and 6). The WJ IV OL tests comprise several clusters (i.e., Oral Language, Broad Oral Language, Oral Expression, Listening Comprehension, Phonetic Coding, and Speed of Lexical Access) as well as three Spanish clusters (which are Spanish parallels of the Oral Language, Broad Oral Language, and Listening Comprehension clusters). Two additional cross-domain cluster scores are available if certain tests from the WJ IV COG and WJ IV OL are given.

## Historical Background

According to McGrew et al. (2014), the original version of the WJ (i.e., the *Woodcock-Johnson Psycho-Educational Battery*; Woodcock and Johnson 1977), was not developed in accordance with a particular theory of intelligence or achievement; rather, a range of tasks of varying levels of complexity were developed based on a series of learning experiments. The tasks were then organized into broad abilities based on factor analysis. The *Woodcock-Johnson Psycho-Educational Battery – Revised* (WJ-R; Woodcock and Johnson 1989) was released in 1989 to be consistent with the *Gf-Gc* theory of intelligence (Horn 1991; Horn and Noll 1997). A decade later, the battery was revised again to stay up-to-date with developments in CHC theory (WJ III; Woodcock et al. 2001, 2007). The WJ IV (Schrank et al. 2014b) was released in 2014, and according to the authors of the WJ IV Technical Manual, “the WJ IV reflects the most contemporary specification of CHC theory at the time of publication” (McGrew et al. 2014, p. 4).

## Psychometric Data

Validity evidence for the WJ IV includes evidence of content validity, factor loadings of WJ IV COG

tests on *g*, cluster score intercorrelations, and exploratory and confirmatory statistic methods. Content validity was demonstrated via results of a multidimensional scaling (MDS) procedure. Median loadings of eighteen of the WJ IV COG tests on *g* ranged from 0.45 to 0.74. McGrew et al. (2014) noted that cluster intercorrelations support the intended internal structure of the WJ IV. Results of a three-stage structural validity analysis of the WJ IV provided evidence that the WJ IV structure was consistent with the intended structure based on CHC theory (McGrew 2005, 2009). There is also a wealth of evidence of convergent and divergent validity in the WJ IV Technical Manual based on correlations with other commonly used measures of intelligence, oral language, and academic achievement (McGrew et al. 2014).

Reliability for tests with time components or multiple-point items was calculated using the Rasch model. Reliability for untimed tests and tests with items scored dichotomously was calculated with split-half procedures. Overall, 38 of the 39 mean test reliability coefficients were above 0.80, 17 of which were above 0.90 (McGrew et al. 2014). Similarly, nearly all speeded tests had test-retest reliability coefficients above 0.80; the two other tests had test-retest correlations of 0.76 and 0.79 for one age band (ages 14–17; McGrew et al. 2014). Reliability coefficients for WJ IV cluster scores fell consistently above 0.85, with the vast majority of cluster scores falling above 0.90.

## Clinical Uses

The WJ IV can be used to evaluate a wide range of broad and narrow cognitive and oral language abilities and academic skills in clients ranging from age 2 to 90+. It is designed to provide users with flexibility to administer tests and make comparisons to answer a variety of referral concerns.

## See Also

- ▶ [Academic Skills](#)
- ▶ [Cognitive Functioning](#)

## References and Readings

- Carroll, J. B. (1993). *Human cognitive abilities: A survey of factor-analytic studies*. Cambridge: Cambridge University Press.
- Carroll, J. B. (2005). The three-stratum theory of cognitive abilities. In D. P. Flanagan & P. L. Harrison (Eds.), *Contemporary intellectual assessment: Theories, tests, and issues* (2nd ed., pp. 69–76). New York: Guilford Press.
- Horn, J. L. (1991). Measurement of intellectual capabilities: A review of theory. In K. S. McGrew, J. K. Werder, & R. W. Woodcock (Eds.), *WJ-R technical manual* (pp. 197–232). Rolling Meadows: Riverside.
- Horn, J. L., & Noll, J. (1997). Human cognitive capabilities: Gf-Gc theory. In D. P. Flanagan, J. L. Genshaft, & P. L. Harrison (Eds.), *Contemporary intellectual assessment: Theories, tests, and issues* (pp. 53–91). New York: Guilford Press.
- Kaufman, A. S., & Kaufman, N. L. (2004). *Kaufman assessment battery for children* (2nd ed.). San Antonio: Pearson.
- Mather, N., & Wendling, B. J. (2014a). *Examiner's manual: Woodcock-Johnson IV tests of achievement*. Rolling Meadows: Riverside.
- Mather, N., & Wendling, B. J. (2014b). *Examiner's manual: Woodcock-Johnson IV tests of cognitive abilities*. Rolling Meadows: Riverside.
- McGrew, K. S. (2005). The Cattell-Horn-Carroll theory of cognitive abilities: Past, present, and future. In D. P. Flanagan & P. L. Harrison (Eds.), *Contemporary intellectual assessment: Theories, tests, and issues* (pp. 136–181). New York: Guilford Press.
- McGrew, K. S. (2009). CHC theory and the human cognitive abilities project: Standing on the shoulders of the giants of psychometric intelligence research. *Intelligence*, 37, 1–10.
- McGrew, K. S., LaForte, E. M., & Schrank, F. A. (2014). *Technical manual: Woodcock-Johnson IV*. Rolling Meadows: Riverside.
- Roid, G. H. (2003). *Stanford Binet intelligence scales* (5th ed.). Austin: PRO-ED.
- Schrank, F. A., & Dailey, D. (2014). *Woodcock-Johnson online scoring and reporting [Online format]*. Rolling Meadows: Riverside.
- Schrank, F. A., McGrew, K. S., & Mather, N. (2014a). *Woodcock-Johnson IV tests of cognitive abilities*. Rolling Meadows: Riverside.
- Schrank, F. A., Mather, N., & McGrew, K. S. (2014b). *Woodcock-Johnson IV*. Rolling Meadows: Riverside.
- Schrank, F. A., Mather, N., & McGrew, K. S. (2014c). *Woodcock-Johnson IV tests of achievement*. Rolling Meadows: Riverside.
- Schrank, F. A., Mather, N., & McGrew, K. S. (2014d). *Woodcock-Johnson IV tests of oral language*. Rolling Meadows: Riverside.
- Wechsler, D. (2002). *Wechsler intelligence scale for children* (4th ed.). San Antonio: Pearson.
- Wechsler, D. (2008). *Wechsler adult intelligence scale* (4th ed.). San Antonio: Pearson.

- Woodcock, R. W., & Johnson, M. B. (1977). *Woodcock-Johnson psycho-educational battery*. Rolling Meadows: Riverside.
- Woodcock, R. W., & Johnson, M. B. (1989). *Woodcock-Johnson psycho-educational battery—Revised*. Rolling Meadows: Riverside.
- Woodcock, R. W., McGrew, K. S., & Mather, N. (2001, 2007). *Woodcock-Johnson III*. Rolling Meadows: Riverside.

## Word Finding

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## Synonyms

Confrontation naming; Naming; Word retrieval

## Definition

Word finding is the ability of a speaker to think of and retrieve specific words to express an intended idea.

## Current Knowledge

Word finding is a skill that takes place in conversational speech as the speaker composes a sentence with nouns, verbs, adjectives, adverbs, and other grammatical words. The process of word finding relies on activation of a complex series of lexical-semantic and phonological representations (Tippett and Hillis 2015). The ease with which words are retrieved during conversation is influenced by psycholinguistic factors such as age of acquisition, familiarity, frequency, and phonological complexity (Raymer 2011). During clinical testing, word finding is often tested in the course of verbal tasks requiring one-word responses, such as confrontation naming of pictures of objects or actions, providing words in a specific category (e.g., name animals, say words

starting with the letter “s,” name the days of the week), or answering specific questions (e.g., Which animal barks?) (Goodglass et al. 2001). Word-finding difficulties are common in all forms of aphasia associated with various neurologic conditions affecting the left cerebral hemisphere (e.g., stroke, brain injury, Alzheimer’s disease). Clinically, word finding is typically tested with confrontation picture-naming measures such as the Boston Naming Test (Kaplan et al. 2001) or word fluency tasks requiring the retrieval of words in a given category, whether semantic (e.g., animals; see Western Aphasia Battery, Kertesz 2007) or phonemic/orthographic (e.g., words beginning with the letter F; see Controlled Oral Word Association Test, Straus et al. 2006). Clinical measures, however, may not provide a clear picture of word-finding abilities as they take place in the context of conversational discourse (Tingley et al. 2003; Carragher et al. 2012).

## Cross-References

- ▶ [Anomia](#)
- ▶ [Aphasia Tests](#)

## References

- Carragher, M., Conroy, P., Sage, K., & Wilkinson, R. (2012). Can impairment-focused therapy change the everyday conversations of people with aphasia? A review of the literature and future directions. *Aphasiology*, 26, 895–916.
- Goodglass, H., Kaplan, E., & Barresi, B. (2001). *The assessment of aphasia and related disorders* (3rd ed.). Philadelphia: Lippincott, Williams, & Wilkins.
- Kaplan, E., Goodglass, H., & Weintraub, S. (2001). *The Revised Boston Naming Test*. Philadelphia, PA: Lippincott, Williams, & Wilkins.
- Kertesz, A. (2007). *The western aphasia battery-revised*. San Antonio: PsychCorp.
- Raymer, A. M. (2011). Naming and word-retrieval impairments. In L. L. LaPointe (Ed.), *Aphasia and related neurogenic language disorders* (pp. 95–110). New York: Thieme Medical Publishers.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *Compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). Oxford: Oxford University Press.



- Tingley, S. J., Kyte, C. S., Johnson, C. J., & Beitchman, J. H. (2003). Single-word and conversational measures of word-finding proficiency. *American Journal of Speech-Language Pathology, 12*, 359–368.
- Tippett, D. C., & Hillis, A. E. (2015). The cognitive processes underlying naming. In A. E. Hillis (Ed.), *The handbook of adult language disorders* (pp. 141–150). New York: Psychology Press.

## Word Memory Test

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### Definition

The Word Memory Test (WMT; Green 2003; Green et al. 1996) is a well-validated effort test. It has been employed in many studies with diverse control and clinical samples of children and adults with an emphasis on traumatic brain injuries, chronic pain, and self-reported depression (Dunn et al. 2003; Gervais et al. 2001, 2004; Gorissen et al. 2005; Green 2003, 2007; Green and Flaro 2003; Green and Iverson 2001; Green et al. 1996, 1999, 2001, 2002, 2003; Iverson et al. 1999; O’Bryant and Lucas 2006; Rohling et al. 2002a, b; Tan et al. 2002; Williamson et al. 2003). The WMT is a computerized test of the ability to learn a list of 20 word pairs. It takes about 7 min of the tester’s time and about 20 min of the patient’s time. The examinee is instructed to watch and remember a list of 20 semantically related word pairs (e.g., dog/cat, man/woman, pig/bacon, fish/fin). Each word pair is presented for 6 s. The list is presented twice. For immediate recognition (IR), the person is shown word pairs containing only one of the words from the original list (e.g., dog/rabbit, man/boy), and he/she selects the word from the original list. A similar recognition test, but with different foil words (e.g., dog/rat), is administered after a 30 min delay interval (i.e., “delayed recognition,” DR). WMT scoring includes a calculation of consistency of responding from Immediate Recognition to

Delayed Recognition (called “Consistency”). The effort measures (IR, DR, and Consistency) are followed by a series of memory tests (Multiple Choice, Paired Associates, Delayed Free Recall, and Long Delayed Free Recall), which become increasingly difficult.

### Current Knowledge

The Word Memory Test is a simple effort test. Children tend to perform very well on the test (Green and Flaro 2003), although performance might be attenuated in younger children, especially those with lower reading levels (Courtney et al. 2003). Ongoing research continues to illustrate the clinical usefulness of the test for detecting poor effort (Bauer 2007; Flaro et al. 2007; Stevens et al. 2008; Sullivan et al. 2007). Recent research has focused on better understanding factors that influence performance on the test, which might lead to false positive identification of poor effort (Batt et al. 2008; Greiffenstein et al. 2008; Greve et al. 2008). There is a wide body of literature supporting its use in clinical practice.

### Cross-References

- ▶ [Malingering](#)
- ▶ [Portland Digit Recognition Test](#)
- ▶ [Test of Memory Malingering](#)
- ▶ [Victoria Symptom Validity Test](#)

### References and Readings

- Batt, K., Shores, E. A., & Chekaluk, E. (2008). The effect of distraction on the Word Memory Test and Test of Memory Malingering performance in patients with a severe brain injury. *Journal of the International Neuropsychological Society, 14*(6), 1074–1080.
- Bauer, L., O’Bryant, S. E., Lynch, J. K., McCaffrey, R. J., & Fisher, J. M. (2007). Examining the Test of Memory Malingering Trial 1 and Word Memory Test Immediate Recognition as screening tools for insufficient effort. *Assessment, 14*(3), 215–222.
- Courtney, J. C., Dinkins, J. P., Allen, L. M., 3rd, & Kuroski, K. (2003). Age related effects in children taking the computerized assessment of response bias and word memory test. *Neuropsychology,*

- Development, and Cognition. Section C, Child Neuropsychology*, 9(2), 109–116.
- Dunn, T. M., Shear, P. K., Howe, S., & Ris, M. D. (2003). Detecting neuropsychological malingering: Effects of coaching and information. *Archives of Clinical Neuropsychology*, 18(2), 121–134.
- Flaro, L., Green, P., & Robertson, E. (2007). Word Memory Test failure 23 times higher in mild brain injury than in parents seeking custody: The power of external incentives. *Brain Injury*, 21(4), 373–383.
- Gervais, R. O., Russell, A. S., Green, P., Allen, L. M., 3rd, Ferrari, R., & Pieschl, S. D. (2001). Effort testing in patients with fibromyalgia and disability incentives. *Journal of Rheumatology*, 28(8), 1892–1899.
- Gervais, R. O., Rohling, M. L., Green, P., & Ford, W. (2004). A comparison of WMT, CARB, and TOMM failure rates in non-head injury disability claimants. *Archives of Clinical Neuropsychology*, 19(4), 475–487.
- Gorissen, M., Sanz, J. C., & Schmand, B. (2005). Effort and cognition in schizophrenia patients. *Schizophrenia Research*, 78(2–3), 199–208.
- Green, P. (2003). *Word Memory Test for Windows: User's manual and program*. Edmonton: Author.
- Green, P. (2007). Spoiled for choice: Making comparisons between forced-choice effort tests. In K. B. Boone (Ed.), *Assessment of feigned cognitive impairment: A neuropsychological perspective* (pp. 50–77). New York: The Guilford Press.
- Green, P., & Flaro, L. (2003). Word memory test performance in children. *Neuropsychology, Development, and Cognition. Section C, Child Neuropsychology*, 9(3), 189–207.
- Green, P., & Iverson, G. L. (2001). Effects of injury severity and cognitive exaggeration on olfactory deficits in head injury compensation claims. *NeuroRehabilitation*, 16(4), 237–243.
- Green, P., Allen, L. M., & Astner, K. (1996). *The Word Memory Test: A user's guide to the oral and computer-administered forms, US version 1.1*. Durham: CogniSyst.
- Green, P., Iverson, G. L., & Allen, L. (1999). Detecting malingering in head injury litigation with the Word Memory Test. *Brain Injury*, 13(10), 813–819.
- Green, P., Rohling, M. L., Lees-Haley, P. R., & Allen, L. M., 3rd. (2001). Effort has a greater effect on test scores than severe brain injury in compensation claimants. *Brain Injury*, 15(12), 1045–1060.
- Green, P., Lees-Haley, P. R., & Allen, L. M. (2002). The Word Memory Test and the validity of neuropsychological test scores. *Journal of Forensic Neuropsychology*, 2(3/4), 97–124.
- Green, P., Rohling, M. L., Iverson, G. L., & Gervais, R. O. (2003). Relationships between olfactory discrimination and head injury severity. *Brain Injury*, 17(6), 479–496.
- Greiffenstein, M. F., Greve, K. W., Bianchini, K. J., & Baker, W. J. (2008). Test of Memory Malingering and Word Memory Test: A new comparison of failure concordance rates. *Archives of Clinical Neuropsychology*, 23(7–8), 801–807.
- Greve, K. W., Ord, J., Curtis, K. L., Bianchini, K. J., & Brennan, A. (2008). Detecting malingering in traumatic brain injury and chronic pain: A comparison of three forced-choice symptom validity tests. *The Clinical Neuropsychologist*, 22(5), 896–918.
- Iverson, G. L., Green, P., & Gervais, R. (1999). Using the Word Memory Test to detect biased responding in head injury litigation. *Journal of Cognitive Rehabilitation*, 2, 4–8.
- O'Bryant, S. E., & Lucas, J. A. (2006). Estimating the predictive value of the Test of Memory Malingering: An illustrative example for clinicians. *The Clinical Neuropsychologist*, 20(3), 533–540.
- Rohling, M. L., Allen, L. M., & Green, P. (2002a). Who is exaggerating cognitive impairment and who is not? *CNS Spectrums*, 7(5), 387–395.
- Rohling, M. L., Green, P., Allen, L. M., & Iverson, G. L. (2002b). Depressive symptoms and neurocognitive test scores in patients passing symptom validity tests. *Archives of Clinical Neuropsychology*, 17(3), 205–222.
- Stevens, A., Friedel, E., Mehren, G., & Merten, T. (2008). Malingering and uncooperativeness in psychiatric and psychological assessment: Prevalence and effects in a German sample of claimants. *Psychiatry Research*, 157 (1–3), 191–200.
- Sullivan, B. K., May, K., & Galbally, L. (2007). Symptom exaggeration by college adults in attention-deficit hyperactivity disorder and learning disorder assessments. *Applied Neuropsychology*, 14(3), 189–207.
- Tan, J. E., Slick, D. J., Strauss, E., & Hultsch, D. F. (2002). How'd they do it? Malingering strategies on symptom validity tests. *The Clinical Neuropsychologist*, 16(4), 495–505.
- Williamson, D. J. G., Green, P., Allen, L., & Rohling, M. L. (2003). Evaluating effort with the Word Memory Test and Category Test- or not: Inconsistencies in a compensation-seeking sample. *Journal of Forensic Neuropsychology*, 3(3), 19–44.

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## Workers' Compensation

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## Synonyms

Disability benefits; Workmen's compensation

## Definition

Workers' compensation provides insurance to cover medical care and compensation for

employees who are injured in the course of employment. In exchange, the employee forfeits the right to sue their employer for negligence. Workers' compensation laws are designed so that employees who are injured or disabled on the job are provided with fixed monetary awards. This is done to prevent or eliminate the need for litigation. These laws also provide benefits for dependents of employees who might have been killed because of work-related accidents or illnesses. Some laws also protect employers and employees by limiting the amount an injured worker can claim from an employer. Most employers are required to subscribe to insurance for workers' compensation, and an employer who does not may be penalized financially.

## Cross-References

► [Independent Neuropsychological Examination](#)

## References and Readings

- DeCarlo, D. R., & Minkowitz, M. (1990). Workers' compensation and employers' liability law: National development. *Tort and Insurance*, 25(L.L.), 521–526.
- Melton, G. B., Pettila, J., Poythress, N. G., & Slobogin, C. (2007). *Psychological evaluations for the courts* (3rd ed.). New York: Guilford.
- Shuman, D. W., & Hardy, J. F. (2007). Causation, psychology, and law. In G. Young, A. Kane, & K. Nicholson (Eds.), *Causality of psychology injury: Presenting evidence in court*. New York: Springer.

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## Working Memory

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## Synonyms

Short-term memory

## Definition

Working memory (WM) is a cognitive construct that refers to the process of short-term storage and management of information. Although it is often used as a synonym of short-term memory (STM), WM is frequently used to convey an additional emphasis on the active manipulation of buffered information. In contrast, STM refers to information storage without any type of manipulation and is, in fact, a key underlying process of WM. Although a variety of WM models have been proposed that include subcomponent processes and functional interactions, there are several central themes that are relatively consistent regardless of any one specific model (e.g., limited capacity, specialized subsystems that are domain specific, and storage of information into memory as distinct from actual perception).

## Historical Background

One of the most influential models of working memory was proposed by (Baddeley and Hitch 1974; Baddeley 1986). According to this model, a *central executive* is responsible for directing attention and managing multiple cognitive processes, including at least two primary subsystems (i.e., the phonological loop, visuospatial sketchpad). The *phonological loop* maintains verbal information, while the *visuospatial sketchpad* maintains visual and spatial information. In addition, the phonological loop is comprised of two components, storage of verbal information and subvocal phonological rehearsal. Phonological rehearsal is used to actively refresh information that would otherwise rapidly decay in the STM store. Similarly, the visuospatial sketchpad is comprised of visual and spatial subsystems that are similarly organized into buffering and rehearsal components. In 2000, Baddeley added a third primary subprocess, the episodic buffer, to his model (Baddeley 2000). The episodic buffer maintains necessary information drawn from long-term memory to integrate the other working memory processes. Baddeley's model has been the most dominant and extensively investigated; however, research in WM has yielded numerous

additional models that represent diverse perspectives (e.g., see Cowan 2008; Ericsson and Kintsch 1995; Unsworth and Engle 2007).

## Current Knowledge

Working memory has become a very large focus of clinical research among a variety of populations, including children with and without learning disabilities (Andersson and Lyxell 2007; Gathercole et al. 2006; Martinussen et al. 2005; Swanson et al. 2009) since WM has considerable effects on reading and comprehension of visual material (De Beni et al. 2007; Palladino et al. 2001). Other clinical populations in which WM has been extensively studied are schizophrenia (Brebion et al. 2009; Piskulic et al. 2007; Van Snellenberg et al. 2016), multiple sclerosis (Chiaravalloti et al. 2005; Hillary et al. 2003; Parmenter et al. 2006; Gmeindl and Courtney 2012), major depressive disorder (Christopher and MacDonald 2005; Gohier et al. 2009; Rose and Ebmeier 2006), as well as cognitive aging and Alzheimer's disease (Baddeley et al. 1999; Belleville et al. 2007; Germano and Kinsella 2005; Nissim et al. 2017; Woods et al. 2018), to name a few.

In addition, several tests have been developed to assess WM; these generally fall into two categories, those that assess STM performance (e.g., delayed matching to sample, Sternberg Task, Brown-Peterson Trigrams) and those that also assess the management and manipulation of buffered information (e.g., the Paced Auditory Serial Addition Test (PASAT), n-back). Many of these assessment measures, however, are utilized primarily in research and do not have extensive information on standardization. Some measures have been developed and standardized to assess WM in clinical settings. For example, the Wechsler Intelligence Tests have a WM score comprised of Digit Span, Letter-Number Sequencing, and Arithmetic subtests (WISC-IV, Wechsler 2003; WAIS-IV, Wechsler 2008). The degree to which these individual subtests measure all the criterion of WM (e.g., extent of manipulation) has been debated in the field

(see Shelton et al. 2009), and more research is warranted regarding the extent to which laboratory tests and clinical tests measure the same WM construct.

## Future Directions

Although Baddeley and Hitch did not propose brain regions associated with these functions, attempts to localize WM systems soon followed the development of functional neuroimaging methods such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI). WM is likely represented by a dense network of regions that connect the prefrontal cortex with the other specialized regions in the brain (D'Esposito 2007). In addition, Smith and Jonides have integrated results from functional neuroimaging studies of the n-back verbal WM paradigm and presented support for localization of Baddeley's model of WM (Smith and Jonides 1998; also see Owen et al. 2005; Nee et al. 2013). Specifically, lateralized working memory systems proposed that left hemisphere activity was associated with verbal WM and right hemisphere activity was related to visuospatial WM. Memory buffering was attributed to posterior parietal activity and executive processing attributed to dorsolateral prefrontal cortex. Subvocal phonological rehearsal used to maintain the memory buffer was associated with premotor regions and Broca's area, with a similar system for buffering and maintenance of visual information in the right hemisphere. There is little evidence as to the localization of the episodic buffer. This was an ambitious framing of the evidence within Baddeley's model, and many of the assertions have received subsequent support. In addition to continued efforts to localize WM systems, future research would include advancing computational models of the integration of the different neuronal substrates responsible for WM (i.e., functional and effective connectivity; Schlosser et al. 2006) among both healthy and clinical populations. For example, WM has been considered one of the primary cognitive deficits in schizophrenia

(see Forbes et al. 2009; Lee and Park 2005), and a recent meta-analysis of neuroimaging studies reported reduced activation in the dorsolateral prefrontal cortex and increased activation in anterior cingulate and left frontal pole regions (Glahn et al. 2005). Additional models are needed to test the interactions between these brain regions.

## Cross-References

- ▶ [Attention](#)
- ▶ [Executive Functioning](#)
- ▶ [Functional Magnetic Resonance Imaging](#)
- ▶ [N-Back Paradigm](#)
- ▶ [Paced Auditory Serial Attention Test](#)
- ▶ [Recent Memory](#)

## References and Readings

- Andersson, U., & Lyxell, B. (2007). Working memory deficit in children with mathematical difficulties: A general or specific deficit? *Journal of Experimental Child Psychology*, 96(3), 197–228.
- Baddeley, A. (1986). *Working memory*. Oxford: Oxford University Press.
- Baddeley, A. (2000). The episodic buffer: A new component of working memory? *Trends in Cognitive Sciences*, 4(11), 417–423.
- Baddeley, A. D., & Hitch, G. J. (1974). Working memory. In G. A. Bower (Ed.), *The psychology of learning and motivation: Advances in research and theory* (Vol. 8, pp. 47–89). New York: Academic.
- Baddeley, A., Cocchini, G., Della Sala, S., Logie, R. H., & Spinnler, H. (1999). Working memory and vigilance: Evidence from normal aging and Alzheimer's disease. *Brain and Cognition*, 41(1), 87–108.
- Belleville, S., Chertkow, H., & Gauthier, S. (2007). Working memory and control of attention in persons with Alzheimer's disease and mild cognitive impairment. *Neuropsychology*, 21(4), 458–469.
- Brebion, G., David, A. S., Jones, H. M., & Pilowsky, L. S. (2009). Working memory span and motor and cognitive speed in schizophrenia. *Cognitive and Behavioral Neurology*, 22(2), 101–108.
- Chiaravalloti, N., Hillary, F., Ricker, J., Christodoulou, C., Kalnin, A., Liu, W. C., et al. (2005). Cerebral activation patterns during working memory performance in multiple sclerosis using fMRI. *Journal of Clinical and Experimental Neuropsychology*, 27(1), 33–54.
- Christopher, G., & MacDonald, J. (2005). The impact of clinical depression on working memory. *Cognitive Neuropsychiatry*, 10(5), 379–399.
- Cowan, N. (2008). What are the differences between long-term, short-term, and working memory? *Progress in Brain Research*, 169, 323–338.
- De Beni, R., Borella, E., & Carretti, B. (2007). Reading comprehension in aging: The role of working memory and metacomprehension. *Neuropsychology, Development, and Cognition Section B, Aging, Neuropsychology and Cognition*, 14(2), 189–212.
- D'Esposito, M. (2007). From cognitive to neural models of working memory. *Philosophical Transactions of the Royal Society of London, Series B*, 362(1481), 761–772.
- Ericsson, K. A., & Kintsch, W. (1995). Long-term working memory. *Psychological Review*, 102(2), 211–245.
- Forbes, N. F., Carrick, L. A., McIntosh, A. M., & Lawrie, S. M. (2009). Working memory in schizophrenia: A meta-analysis. *Psychological Medicine*, 39(6), 889–905.
- Gathercole, S. E., Alloway, T. P., Willis, C., & Adams, A. M. (2006). Working memory in children with reading disabilities. *Journal of Experimental Child Psychology*, 93(3), 265–281.
- Germano, C., & Kinsella, G. J. (2005). Working memory and learning in early Alzheimer's disease. *Neuropsychology Review*, 15(1), 1–10.
- Glahn, D. C., Ragland, J. D., Abramoff, A., Barrett, J., Laird, A. R., Bearden, C. E., et al. (2005). Beyond hypofrontality: A quantitative meta-analysis of functional neuroimaging studies of working memory in schizophrenia. *Human Brain Mapping*, 25(1), 60–69.
- Gmeindl, L., & Courtney, S. M. (2012). Deconstructing spatial working memory and attention deficits in multiple sclerosis. *Neuropsychology*, 26(1), 57–70.
- Gohier, B., Ferracci, L., Surguladze, S. A., Lawrence, E., El Hage, W., Kefi, M. Z., et al. (2009). Cognitive inhibition and working memory in unipolar depression. *Journal of Affective Disorders*, 116(1–2), 100–105.
- Hillary, F. G., Chiaravalloti, N. D., Ricker, J. H., Steffener, J., Bly, B. M., Lange, G., et al. (2003). An investigation of working memory rehearsal in multiple sclerosis using fMRI. *Journal of Clinical and Experimental Neuropsychology*, 25(7), 965–978.
- Lee, J., & Park, S. (2005). Working memory impairments in schizophrenia: A meta-analysis. *Journal of Abnormal Psychology*, 114(4), 599–611.
- Martinussen, R., Hayden, J., Hogg-Johnson, S., & Tannock, R. (2005). A meta-analysis of working memory impairments in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44(4), 377–384.
- Nee, D., Brown, J., Berman, M., Demiralp, E., Krawitz, A., & Jonides, J. (2013). A meta-analysis of executive components of working memory. *Cerebral Cortex*, 23(2), 264–282.
- Nissim, N., O'Shea, A., Bryant, V., Porges, E., Cohen, R., & Woods, A. J. (2017). Frontal structural neural correlates of working memory performance in older adults. *Frontiers in Aging Neuroscience*, 8, 328.



- Owen, A., McMillan, K., Laird, A., & Bullmore, E. (2005). N-back working memory paradigm: A meta-analysis of normative functional neuroimaging studies. *Human Brain Mapping, 25*(1), 46–59.
- Palladino, P., Cornoldi, C., De Beni, R., & Pazzaglia, F. (2001). Working memory and updating processes in reading comprehension. *Memory & Cognition, 29*(2), 344–354.
- Parmenter, B. A., Shucard, J. L., Benedict, R. H., & Shucard, D. W. (2006). Working memory deficits in multiple sclerosis: Comparison between the n-back task and the paced auditory serial addition test. *Journal of the International Neuropsychological Society, 12*(5), 677–687.
- Piskulic, D., Olver, J. S., Norman, T. R., & Maruff, P. (2007). Behavioural studies of spatial working memory dysfunction in schizophrenia: A quantitative literature review. *Psychiatry Research, 150*(2), 111–121.
- Rose, E. J., & Ebmeier, K. P. (2006). Pattern of impaired working memory during major depression. *Journal of Affective Disorders, 90*(2–3), 149–161.
- Schlösser, R. G., Wagner, G., & Sauer, H. (2006). Assessing the working memory network: Studies with functional magnetic resonance imaging and structural equation modeling. *Neuroscience, 139*(1), 91–103.
- Shelton, J. T., Elliott, E. M., Hill, B. D., Calmia, M. R., & Gouvier, W. D. (2009). A comparison of laboratory and clinical working memory tests and their prediction of fluid intelligence. *Intelligence, 37*, 283–293.
- Smith, E. E., & Jonides, J. (1998). Neuroimaging analyses of human working memory. *Proceedings of the National Academy of Sciences USA, 95*(20), 12061–12068.
- Swanson, H. L., Xinhua, Z., & Jerman, O. (2009). Working memory, short-term memory, and reading disabilities: A selective meta-analysis of the literature. *Journal of Learning Disabilities, 42*(3), 260–287.
- Unsworth, N., & Engle, R. W. (2007). The nature of individual differences in working memory capacity: Active maintenance in primary memory and controlled search from secondary memory. *Psychological Review, 114*(1), 104–132.
- Van Snellenberg, J., Girgis, R., Horga, G., van de Giessen, E., Slifstein, M., Ojeil, N., et al. (2016). Mechanisms of working memory impairment in schizophrenia. *Biological Psychiatry, 80*(8), 617–626.
- Wechsler, D. (2003). *Wechsler intelligence scales for children-fourth edition*. Bloomington: Pearson.
- Wechsler, D. (2004). *Wechsler intelligence scales for children* (4th ed., Integrated). Bloomington: Pearson.
- Wechsler, D. (2008). *Wechsler adult intelligence scales* (4th ed.). Bloomington: Pearson.
- Woods, A. J., Cohen, R., Marsiske, M., Alexander, G., Czaja, S., & Wu, S. (2018). Augmenting cognitive training in older adults (The ACT study): Design and methods of a phase III tDCS and cognitive training trial. *Contemporary Clinical Trials, 65*, 19–32.

## Working Memory Index

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## Synonyms

Freedom from Distractibility (WISC-III); WMI;  
 Working Memory Scale (WAIS-IV)

## Definition

A score derived from administration of selected subtests from the third and fourth edition Wechsler Adult Intelligence Scale (WAIS) and Wechsler Intelligence Scale for Children (WISC) and the fourth edition of the Wechsler Preschool and Primary Scale of Intelligence (WPPSI). The Working Memory Index (WMI) is designed to provide a measure of a person's ability to attend to information presented verbally, manipulate information in short-term immediate memory, and then formulate a response.

## Current Knowledge

*Wechsler Intelligence Scales (WIS)*: The WIS family of tests are some of the most widely used test batteries to assess general intellectual ability in adults aged 16 years or older (Wechsler Adult Intelligence Scale; WAIS), children aged 6–16 years (Wechsler Intelligence Scale for Children; WISC), and children aged 2–7 years (Wechsler Preschool and Primary Scale of Intelligence; WPPSI). Since the original development of these tests (WAIS, 1955; WISC, 1949; WPPSI, 1967), all three batteries have been revised on several occasions. The most recent revisions were published in 2012 (WPPSI-IV), 2014 (WISC-V), and 2008 (WAIS-IV).

*History*: Until recently, one of the most prominent features of the WIS was the derivation and

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

*Evolution:* Factor-based interpretation of the WIS was first included in the WISC-III (1991). The addition of a new subtest (i.e., Symbol Search) resulted in the introduction of a four factor scoring system, defined by a Verbal Comprehension Index (VCI), Perceptual Organization Index (POI), Freedom from Distractibility Index (FDI), and Processing Speed Index (PSI). The same four-factor scoring system was also included in the WAIS-III (1997) following the inclusion of two new subtests (i.e., Symbol Search and Letter-

Number Sequencing), with the exception that the FDI was renamed the Working Memory Index (WMI). For the WISC-III and WAIS-III, the index scores were initially introduced as an “alternative” system for scoring and interpretation that coexisted with the traditional IQ scores which remained unchanged. However, the publication of the WPPSI-IV, WISC-IV, and WAIS-IV represented a significant deferment from the Wechsler scale tradition. The Verbal IQ and Performance IQ scores were excluded for the first time, and only the Full scale IQ score was retained. For the first time in WIS history, the interpretation of the WIS was largely focused on the Index scores that were thought to provide a more precise measurement of multiple cognitive abilities assessed by these batteries. For the WISC-IV, the index scores include VCI, WMI, PSI, and the renamed POI – Perceptual Reasoning Index (PRI). For the WISC-V, the index scores include VCI, WMI, PSI, Visual Spatial Index (VSI), and Fluid Reasoning Index (FRI). The PRI/POI was replaced with the VSI and FRI, allowing for more nuanced communication of abilities. For the WAIS-IV, the four index scores (now known as “scales”) include the Verbal Comprehension Scale, Perceptual Reasoning Scale, Working Memory Scale, and Processing Speed Scale. For the WPPSI-IV, the index scores include the VCI, WMI, VSI, and, for children ages 4–7, the PSI and FRI.

*Subtest Composition:* The core subtests used to derive WMI vary across the WAIS-III/IV, WISC-III/IV/V, and WPPSI-IV. For the WAIS-III, the core subtests contributing to the WMI are Arithmetic, Digit Span, and Letter-Number Sequencing. For the WAIS-IV, only two of these core

**Working Memory Index,**

**Table 1** Core subtest composition of WMI

	AR	DSP	LNS	PS	PM	ZL
WAIS-III	•	•	•			
WAIS-IV	•	•				
WISC-III <sup>a</sup>	•	•				
WISC-IV		•	•			
WISC-V		•		•		
WPPSI-IV					•	•

*Note:* AR arithmetic, DSP digit span, LNS letter-number sequencing, PS picture span, PM picture memory, ZL zoo locations

<sup>a</sup>Known as Freedom from Distractibility Index on this test

subtests remain (Arithmetic, Digit Span) with Letter-Number Sequencing now a supplementary subtest. For the WISC-III, Arithmetic and Digit Span are included in the calculation of FDI. For the WISC-IV, Digit Span and Letter-Number Sequencing are included in the calculation of WMI. For the WISC-V, Digit Span and Picture Span are the core subtests comprising the WMI. For the WPPSI-IV, Picture Memory and Zoo Locations comprise the WMI. The core subtests used to derive WMI (and FDI in the WISC-III) across the WAIS, WISC, and WPPSI batteries and revisions are presented below. Only the core subtests (not supplementary) are shown in Table 1.

### See Also

- ▶ [Distractibility](#)
- ▶ [Intelligence](#)
- ▶ [Perceptual Organization Index](#)
- ▶ [Performance IQ](#)
- ▶ [Processing Speed Index](#)
- ▶ [Verbal Comprehension Index](#)
- ▶ [Verbal IQ](#)
- ▶ [Wechsler Intelligence Scale for Children](#)
- ▶ [Wechsler Memory Scale All Versions](#)

### Further Reading

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

- Kaufman, A. S., & Lichtenberger, E. O. (2006). *Assessing adolescent and adult intelligence* (3rd ed.). Hoboken: John Wiley & Sons, Inc.
- Sattler, J. M. (2008). *Assessment of children: Cognitive foundations* (5th ed.). San Diego: Author.
- Tulsky, D. S., Saklofske, D. H., & Zhu, J. (2003). Revising a standard: An evaluation of the origin and development of the WAIS-III. In D. S. Tulsky et al. (Eds.), *Clinical interpretation of the WAIS-III and WMS-III* (pp. 43–92). San Diego: Academic Press.

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## Writing

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### Definition

Writing is the orthographic representation of language symbols; that is, it is the translation of phonemes into graphemes for the purpose of communication. Impairments in writing ability are referred to as *dysgraphia*.

### Cross-References

- ▶ [Agraphia](#)