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## **“Table Top” Sign**

Inability to place the hand flat on a level surface, recognized causes of which include ulnar neuropathy (*main en griffe*), Dupuytren’s contracture, diabetic cheiroarthropathy, and camptodactyly.

### **Cross References**

“Prayer sign”

## **Tachylalia**

Tachylalia is increased speech velocity. This has been reported in patients with cerebrotendinous xanthomatosis, particularly in the 20 to 40 year age group.

### **References**

Verrips A, Van Engelen B, de Swart B *et al.* Increased speech rate (tachylalia) in cerebrotendinous xanthomatosis: a new sign. *Journal of Medical Speech-Language Pathology* 1998; **6**: 161-164.

## **Tachyphemia**

Repetition of a word or phrase with increasing rapidity and decreasing volume; may be encountered as a feature of parkinsonian syndromes.

### **Cross References**

Parkinsonism

## **Tactile Agnosia**

A selective impairment of object recognition by touch despite (relatively) preserved somesthetic perception. This is a unilateral disorder resulting from lesions of the contralateral inferior parietal cortex.

### **References**

Reed CL, Caselli RJ, Farah MJ. Tactile agnosia: underlying impairment and implications for normal tactile object recognition. *Brain* 1996; **119**: 875-888

### **Cross References**

Agnosia

## **Tadpole Pupils**

Pupillary dilatation restricted to one segment may cause peaked elongation of the pupil, a shape likened to a tadpole’s pupil. This has been recorded in Horner’s syndrome, migraine, and Holmes-Adie pupil.

### **References**

Thompson HS, Zackon DH, Czarnecki JSC. Tadpole-shaped pupils caused by segmental spasm of the iris dilator muscle. *American Journal of Ophthalmology* 1983; **96**: 467-477

**Talantropia**

- see NYSTAGMUS

**Tandem Walking**

Tandem walking, or heel-toe walking, also known as the dynamic Romberg's test, is the ability to walk along a straight line placing one foot directly in front of the other, heel to toe, which may be likened to walking a tightrope. In ataxic disorders, cerebellar (midline cerebellum, in which axial coordination is most affected) or sensory (loss of proprioception), the ability to tandem walk is impaired, as reflected by the tendency of such patients to compensate for their incoordination by developing a broad based gait.

**Cross References**

Ataxia; Cerebellar syndromes; Proprioception; Rombergism, Romberg's sign

**Tay's Sign**

- see CHERRY RED SPOT AT THE MACULA

**Teichopsia**

- see FORTIFICATION SPECTRA

**Telegraphic Speech**

- see AGRAMMATISM

**Telopsia**

A visual illusion in which the image is altered in position.

**Cross References**

Illusion; Pelopsia; Porropsia

**Temporal Desaturation**

Temporal desaturation refers to an impairment in perception of red targets confined to the temporal visual hemifield. This may be the earliest indication of a developing temporal field defect, as in a bitemporal hemianopia due to a chiasmal lesion, or a monocular temporal field defect (junctional scotoma of Traquair) due to a distal ipsilateral optic nerve lesion.

**Cross References**

Hemianopia; Scotoma

**Temporal Pallor**

Pallor of the temporal portion of the optic nerve head may follow atrophy of the macular fibre bundle in the retina, since the macular fibers for central vision enter the temporal nerve head. This may be associated with impairment of central vision.

**Cross References**

Optic atrophy

**Terson Syndrome**

Vitreous hemorrhage in association with any form of intracranial or subarachnoid hemorrhage.

**Tetanus, Tetany**

- see *MAIN D'ACCOUCHEUR*; RISUS SARDONICUS; SPASM

**Tetraparesis, Tetraplegia**

- see QUADRIPARESIS, QUADRIPLÉGIA

**Threat Reflex**

- see BLINK REFLEX

**Tic**

A tic is an abrupt, jerky repetitive movement involving discrete muscle groups, hence a less complex movement than a stereotypy. Vocal (phonic) tics are also described. Tics vary in intensity, lack rhythmicity, and are relatively easy to imitate. They may temporarily be voluntarily suppressed by will power (perhaps accounting for their previous designation as “habit spasms”) but this is usually accompanied by a growing inner tension or restlessness, only relieved by the performance of the movement. The pathophysiology of tics is uncertain. The belief that Gilles de la Tourette syndrome was a disorder of the basal ganglia has now been superseded by evidence of dysfunction within the cingulate and orbitofrontal cortex, perhaps related to excessive endorphin release.

The etiological differential diagnosis of tic includes:

- Idiopathic
- Gilles de la Tourette syndrome
- Tics related to structural brain damage
- Drug-induced tics.

Treatment of tics is most usually with dopamine antagonists (haloperidol, sulpiride) and opioid antagonists (naltrexone); clonidine (central  $\alpha_2$  adrenergic receptor antagonist) and tetrabenazine (dopamine-depleting agent) have also been reported to be beneficial on occasion.

The word tic has also been used to describe the paroxysmal, lancinating pains of trigeminal neuralgia (tic douloureux).

**References**

Lees AJ. *Tics and related disorders*. Edinburgh: Churchill Livingstone, 1985

Weeks RA, Turjanski N, Brooks DJ. Tourette's syndrome: a disorder of cingulate and orbitofrontal function? *Quarterly Journal of Medicine* 1996; **89**: 401-408

**Cross References**

Klazomania; Stereotypy

**“Tie Sign”**

- see VISUAL DISORIENTATION

### Tinel's Sign (Hoffmann-Tinel Sign)

Tinel's sign (Hoffmann-Tinel sign) is present when tingling (paresthesia) is experienced when tapping lightly with a finger or a tendon hammer over a compressed or regenerating peripheral nerve. The tingling (Tinel's "sign of formication") is present in the cutaneous distribution of the damaged nerve ("peripheral reference"). Although originally described in the context of peripheral nerve regeneration after injury, Tinel's sign may also be helpful in diagnosing focal entrapment neuropathy, such as carpal tunnel syndrome. However, it is a "soft" sign; like other provocative tests for carpal tunnel syndrome (e.g., Phalen's sign) it is not as reliable for diagnostic purposes as electromyography (EMG). One study found a specificity of 59-77%, and sensitivity of 60-67%.

A "motor Tinel sign" has been described, consisting of motor EMG activity and jerking of muscles evoked by manipulation of an entrapped nerve trunk.

The neurophysiological basis of Tinel's sign is presumed to be the lower threshold of regenerating or injured (demyelinated) nerves to mechanical stimuli, which permits ectopic generation of orthodromic action potentials, as in Lhermitte's sign.

### References

- Heller L, Ring H, Costeff H, Solzi P. Evaluation of Tinel's and Phalen's signs in the diagnosis of the carpal tunnel syndrome. *European Neurology* 1986; **25**: 40-42
- Montagna P. Motor Tinel sign: a new localizing sign in entrapment neuropathy. *Muscle Nerve* 1994; **17**: 1493-1494
- Pearce JMS. Hoffmann and Tinel's sign of formication. In: Pearce JMS. *Fragments of neurological history*. London: Imperial College Press, 2003: 375-377

### Cross References

Flick sign; Lhermitte's sign; Phalen's sign

### Tinnitus

Tinnitus is the perception of elementary nonenvironmental sound or noise in the ear. This is most usually a subjective phenomenon (i.e., heard only by the sufferer), occurring in the absence of acoustic stimulation. It may occur in conjunction with either conductive or sensorineural hearing loss. However, in about one-fifth of sufferers, tinnitus is objective (i.e., heard also by an observer). This may result from:

- Vascular causes: e.g., arteriovenous malformation, fistula; carotid or vertebrobasilar bruit
- Mechanical causes: e.g., palatal myoclonus (ear click).

The common causes of subjective tinnitus are:

- Middle/inner ear disease: cochlear hydrops (Ménière's disease), presbycusis, acoustic tumor
- Pulsatile: normal heartbeat, glomus jugulare tumor, raised intracranial pressure, cervical/intracranial aneurysm, arteriovenous malformation.

**Cross References**

Hallucination; Palatal myoclonus

**“Tip-of-the-Tongue” Phenomenon**

- see CIRCUMLOCUTION

**Titubation**

- see HEAD TREMOR

**Todd's Paralysis, Todd's Paresis**

Todd's paralysis (Todd's paresis) is a transient localized weakness (usually hemiparesis), lasting seconds to minutes (exceptionally 24 to 48 hours), observed following a focal motor seizure or Jacksonian seizure originating in the central motor strip, or febrile convulsion, a phenomenon first described by RB Todd in 1854. The pattern and duration of post-ictal signs is quite heterogeneous. Aphasia is also described. A postictal “paralytic” conjugate ocular deviation may be observed after adverse seizures. Todd's paresis is of localizing value, being contralateral to the epileptogenic hemisphere.

The differential diagnosis of transient postictal hemiparesis includes stroke, hemiplegic migraine, and, in children, alternating hemiplegia.

**References**

Binder DK. A history of Todd and his paralysis. *Neurosurgery* 2004; **54**: 480-486

Kellinghaus C, Kotagal P. Lateralizing value of Todd's palsy in patients with epilepsy. *Neurology* 2004; **62**: 289-291

Rolak LA, Rutecki P, Ashizawa T, Harati Y. Clinical features of Todd's post-epileptic paralysis. *Journal of Neurology, Neurosurgery and Psychiatry* 1992; **55**: 63-64

**Cross References**

Hemiparesis; Seizures

**Toe Walking**

Toe walking, or cock walking, is walking on the balls of the toes, with the heel off the floor. A tendency to walk on the toes may be a feature of hereditary spastic paraparesis, and the presenting feature of idiopathic torsion dystonia in childhood.

**Cross References**

Dystonia

**Torpor**

- see OBTUNDATION

**Torticollis**

Torticollis (wryneck, cervical dystonia, nuchal dystonia) is a movement disorder characterized by involuntary contraction of neck musculature, involving especially sternocleidomastoid, trapezius, and splenius capitis. In the majority of cases (> 50%) this produces head rotation, but laterocollis, retrocollis, tremulous (“no-no”) and complex

(i.e., variable) forms are seen; antecollis is unusual. Contractions are usually unilateral, may be associated with local pain, and, as with other types of dystonia, may be relieved by a “sensory trick” (*geste antagoniste*).

Causes of torticollis include:

Idiopathic (the majority)

Secondary to acquired cervical spine abnormalities, trauma

Cervical spinal tumor

Tardive effect of neuroleptics

The treatment of choice is botulinum toxin injections into the affected muscles. Injections benefit up to 70-80% of patients, but need to be repeated every three months or so.

### Cross References

Antecollis; Dystonia; Geste antagoniste; Laterocollis; Retrocollis

### Tortopia

- see ENVIRONMENTAL TILT

### Transcortical Aphasias

Transcortical aphasias may be categorized as either motor or sensory.

- *Transcortical motor aphasia* (TCMA):

There is a dissociation between preserved repetition (*cf.* conduction aphasia) and impaired fluency, manifest as delayed initiation, even mutism, impaired lexical selection, and reduced capacity to generate unconstrained syntactic forms. TCMA is associated with pathology (usually infarction) in the supplementary motor area, superior to Broca’s area (left lateral frontal cortex) or in subcortical structures including white matter projections and dorsal caudate nucleus; it has clinical similarities with Broca’s aphasia.

- *Transcortical sensory aphasia* (TCSA):

There is a dissociation between preserved repetition (*cf.* conduction aphasia) and impairments of spoken and written language comprehension without phonemic paraphasia. TCSA is associated with pathology (usually infarction) in the ventral and ventrolateral temporal lobe involving the fusiform gyrus and the inferior temporal gyrus, and posterior convexity lesions involving the posterior middle temporal gyrus and the temporo-occipital junction. It has similarities Wernicke’s aphasia.

Some authorities prefer to label these conditions as extrasylvian aphasic syndromes, to distinguish them from the perisylvian aphasic syndromes (Broca, Wernicke, conduction); moreover, these syndromes are not “transcortical” in any literal sense.

Dynamic aphasia (*q.v.*) may be a lesser version of TCMA, in which there are no paraphasias and minimal anomia, preserved repetition and automatic speech, but reduced spontaneous speech. This may be associated with lesions of dorsolateral prefrontal cortex (“frontal aphasia”) in the context of frontal lobe degeneration. There

may be incorporational echolalia, when the patient uses the examiner's question to help form an answer.

### References

Alexander MP. Transcortical motor aphasia: a disorder of language production. In: D'Esposito M (ed.). *Neurological foundations of cognitive neuroscience*. Cambridge: MIT Press, 2003: 165-174

Alexander MP, Hiltbrunner B, Fischer RS. Distributed anatomy of transcortical sensory aphasia. *Archives of Neurology* 1989; **46**: 885-892

Boatman D, Gordon B, Hart J, Selnes O, Miglioretti D, Lenz F. Transcortical sensory aphasia: revisited and revised. *Brain* 2000; **123**: 1634-1642

### Cross References

Aphasia; Broca's aphasia; Conduction aphasia; Dynamic aphasia; Echolalia; Paraphasia; Wernicke's aphasia

### Transverse Smile

- see "MYASTHENIC SNARL"

### *Tremblement Affirmatif, Tremblement Negatif*

- see HEAD TREMOR

### Tremor

Tremor is an involuntary movement, roughly rhythmic and sinusoidal, although some tremors (e.g., dystonic) are irregular in amplitude and periodicity. Tremors may be classified clinically:

- *Rest tremor*:  
present when a limb is supported against gravity and there is no voluntary muscle activation, e.g., the 3.5-7 Hz "pill rolling" hand tremor of Parkinson's disease; midbrain/rubral tremor.
- *Action tremor*:  
present during any voluntary muscle contraction.  
Various subtypes of action tremor are recognized:
- *Postural tremor*:  
present during voluntary maintenance of a posture opposed by gravity, e.g., arm tremor of essential tremor; 6Hz postural tremor sometimes seen in Parkinson's disease, which may predate emergence of akinesia/rigidity/rest tremor; modest postural tremor of cerebellar disease; some drug-induced tremors (including alcohol withdrawal, delirium tremens); tremor of IgM paraproteinemic neuropathy; wing-beating tremor of Wilson's disease.
- *Kinetic tremor*:  
present with movement, often with an exacerbation at the end of a goal-directed movement (intention tremor), e.g., cerebellar/midbrain tremor (3-5Hz).
- *Task-specific tremor*:  
evident only during the performance of a highly-skilled activity, e.g., primary writing tremor.

- *Isometric tremor:*  
present when voluntary muscle contraction is opposed by a stationary object, e.g., primary orthostatic tremor (14-18Hz).
- *Psychogenic tremors:*  
these are difficult to classify, with changing characteristics; the frequency with which such tremors are observed varies greatly between different clinics; the coactivation sign (increase in tremor amplitude with peripheral loading) is said to be typical of psychogenic tremor.

EMG may be useful for determining tremor frequency, but is only diagnostic in primary orthostatic tremor.

Various treatments are available for tremor, with variable efficacy. Essential tremor often responds to alcohol, and this is a reasonable treatment (previous anxieties that such a recommendation would lead to alcoholism seem unjustified); alternatives include propranolol, primidone, topiramate, alprazolam, flunarizine, and nicardipine. In Parkinson's disease, tremor is less reliably responsive to levodopa preparations than akinesia and rigidity; anticholinergics, such as benzhexol, may be more helpful (but may cause confusion). Primary orthostatic tremor has been reported to respond to clonazepam, primidone, and levodopa. Cerebellar tremor is often treated with isoniazid, but seldom with marked benefit, likewise carbamazepine, clonazepam, ondansetron, limb weights; stereotactic surgery may be the optimum treatment if preliminary experimental data are confirmed.

#### References

- Bain PG, Findley LJ. *Assessing Tremor Severity*. London: Smith-Gordon, 1993
- Barker R, Burn DJ. Tremor. *Advances in Clinical Neuroscience & Rehabilitation* 2004; **4(1)**: 13-14
- Deuschl G, Bain P, Brin M and an Ad Hoc Scientific Committee. Consensus statement of the Movement Disorder Society on tremor. *Movement Disorders* 1998; **13(suppl3)**: 2-23
- Findley LJ, Koller WC (eds.). *Handbook of Tremor Disorders*. New York: Marcel Dekker, 1995

#### Cross References

Asterixis; Coactivation sign; Head tremor; Knee tremor; Parkinsonism; Vocal tremor, Voice tremor; Wing-beating tremor

#### Trendelenburg's Sign

Trendelenburg's sign is tilting of the pelvis toward the side of the unaffected raised leg in a unilateral superior gluteal nerve lesion.

#### Triparesis

- see SEQUENTIAL PARESIS

#### Trismus

Trismus is an inability to open the jaw due to tonic spasm or contraction of the masticatory muscles, principally masseter and temporalis, effecting forced jaw closure ("lockjaw").



Recognized causes and associations of trismus include:

Dystonia of the jaw muscles (*e.g.*, drug-induced dystonic reaction)

Generalized tonic-clonic epileptic seizure

Neuromuscular diseases: polymyositis, tetanus, nemaline myopathy, trauma to the muscles of mastication, rabies, strychnine poisoning

Infection in the pterygomandibular space

Metabolic disorders: Gaucher's disease (type II)

Central disorders: brainstem encephalopathy, multiple sclerosis, pseudobulbar palsy.

### References

Lai MM, Howard RS. Pseudobulbar palsy associated with trismus. *Postgraduate Medical Journal* 1994; **70**: 823-824

### Cross References

Dystonia; Pseudobulbar palsy

### Trombone Tongue

Trombone tongue, or flycatcher tongue, refers to an irregular involuntary darting of the tongue in and out of the mouth when the patient is requested to keep the tongue protruded. This sign may be seen in choreiform movement disorders, such as Huntington's disease and neuroanthocytosis, and in tardive dyskinesia.

### Cross References

Chorea, Choreoathetosis; Impersistence; Milkmaid's grip

### Trömner's Sign

Trömner's sign is flexion of the thumb and index finger in response to tapping or flicking the volar surface of the distal phalanx of the middle finger, held partially flexed between the examiner's finger and thumb. This is an alternative method to Hoffmann's sign ("snapping" the distal phalanx) to elicit the finger flexor response. As in the latter, it is suggestive of a corticospinal tract (upper motor neurone) lesion above C5 or C6, especially if unilateral, although it may be observed in some normal individuals.

### Cross References

Hoffmann's sign; Upper motor neurone (UMN) syndrome

### Trousseau's Sign

Trousseau described the signs and symptoms of tetany, including anesthesia, paresthesia, the *main d'accoucheur* posture, as well as noting that the latter could be reproduced by applying a bandage or inflating a cuff around the arm so as to impede circulation; the latter is now known as Trousseau's sign, and indicates latent tetany.

Trousseau also noted the concurrence of venous thrombosis and migrating thrombophlebitis with malignant disease, also referred to as Trousseau's sign; this may present with cerebral venous thrombosis.

### References

Pearce JMS. Armand Trousseau, physician and neurologist. In: Pearce JMS. *Fragments of neurological history*. London: Imperial College Press, 2003: 528-547

**Cross References**

Achromatopsia; Chvostek's sign; Main d'accoucheur

**Tullio Phenomenon**

The Tullio phenomenon is the experience of vestibular symptoms and signs (vertigo, nystagmus, oscillopsia, postural imbalance, ocular tilt reaction, +/- skew deviation) on exposure to high intensity acoustic stimuli, presumed to be due to hyperexcitability of the normal vestibular response to sound, causing pathological stimulation of the semicircular canals and/or otoliths. This unusual phenomenon may be associated with perilymph leaks or a defect in the capsule forming the roof of the anterior semicircular canal. The sound sensitivity is probably at the level of the receptors rather than the vestibular nerve.

**References**

Watson SRD, Halmagyi GM, Colebatch JG. Vestibular hypersensitivity to sound (Tullio phenomenon). Structural and functional assessment. *Neurology* 2000; **54**: 722-728

**Cross References**

Nystagmus; Ocular tilt reaction; Oscillopsia; Skew deviation; Vertigo

**“Tunnel Vision”**

A complaint of “tunnel vision” may indicate constriction of the visual field. This may be observed with enlargement of the blind spot and papilledema as a consequence of raised intracranial pressure or with a compressive optic neuropathy. The visual field enlarges the further away from the eye the visual target used to map the field is held, hence there is in fact “funnel vision.” In nonorganic visual impairment, by contrast, the visual field stays the same size with more distant targets (tunnel vision).

A tunnel vision phenomenon has also been described as part of the aura of seizures of anteromedial temporal and occipitotemporal origin. A closing in of vision may be described as a feature of presyncope.

**Cross References**

Aura; Blind spot; Hemianopia; Papilledema; Visual field defects

**Two-Point Discrimination**

Two-point discrimination is the ability to discriminate two adjacent point stimuli (e.g., using a pair of calipers) as two rather than one. The minimum detectable distance between the points (acuity) is smaller on the skin of the finger tips (i.e., greater acuity) than, say, the skin on the back of the trunk. Impairments of two-point discrimination may occur with dorsal column spinal cord lesions, in which proprioception (and possibly vibration) is also impaired. Cortical parietal lobe lesions may produce a cortical sensory syndrome of astereognosis, agraphesthesia, and impaired two-point discrimination.

**Cross References**

Astereognosis; Graphesthesia; Proprioception; Vibration