F

"Face-Hand Test"
- see "Arm Drop"

Facial Paresis
Facial paresis, or prosopoplegia, may result from:

- central (upper motor neurone) lesions
- peripheral (lower motor neurone; facial (VII) nerve) lesions
- neuromuscular junction transmission disorders
- primary disease of muscle (*i.e.*, myogenic)

Facial paresis is clinically heterogeneous which may be helpful with lesion localization.

- *Upper motor neurone facial weakness* ("central facial palsy"):
  The ability to raise the eyebrow is preserved due to bilateral supranuclear connections to the frontalis muscle. A dissociation between volitional and emotional facial movements may also occur. Emotional facial palsy refers to the absence of emotional facial movement but with preserved volitional movements, as may be seen with frontal lobe (especially non-dominant hemisphere) precentral lesions (as in abulia, Fisher's sign) and in medial temporal lobe epilepsy with contralateral mesial temporal sclerosis. Volitional paresis without emotional paresis may occur when corticobulbar fibers are interrupted (precentral gyrus, internal capsule, cerebral peduncle, upper pons).

  Causes of upper motor neurone facial paresis include:

  Unilateral:

  Hemisphere infarct (with hemiparesis)
  Lacunar infarct (facio-brachial weakness, +/– dysphasia)
  Space occupying lesions: intrinsic tumor, metastasis, abscess

  Bilateral:

  Motor neurone disease
  Diffuse cerebrovascular disease
  Pontine infarct (locked-in syndrome)

- *Lower motor neurone facial weakness* (peripheral origin):

If this is due to facial (VII) nerve palsy, it results in ipsilateral weakness of frontalis (*cf.* upper motor neurone facial paresis), orbicularis oculi, buccinator, orbicularis oris and platysma. Clinically this produces:

Drooping of the side of the face with loss of the nasolabial fold

Widening of the palpebral fissure with failure of lid closure (lagophthalmos)
Eversion of the lower lid (ectropion) with excessive tearing (epiphora)
Inability to raise the eyebrow, close the eye, frown, blow out the cheek, show the teeth, laugh, and whistle
+/– dribbling of saliva from the paretic side of the mouth
Depression of the corneal reflex (efferent limb of reflex arc affected)
Speech alterations: softening of labials (p, b).

Depending on the precise location of the facial nerve injury, there may also be paralysis of the stapedius muscle in the middle ear, causing sounds to seem abnormally loud (especially low tones: hyperacusis), and impairment of taste sensation on the anterior two-thirds of the tongue if the chorda tympani is affected (ageusia, hypogeusia). Lesions within the facial canal distal to the meatal segment cause both hyperacusis and ageusia; lesions in the facial canal between the nerve to stapedius and the chorda tympani cause ageusia but no hyperacusis; lesions distal to the chorda tympani cause neither ageusia nor hyperacusis (*i.e.*, facial motor paralysis only). Lesions of the cerebellopontine angle cause ipsilateral hearing impairment and corneal reflex depression (afferent limb of reflex arc affected) in addition to facial weakness. There is also a sensory branch to the posterior wall of the external auditory canal which may be affected resulting in local hypoesthesia (Hitselberg sign).

Causes of lower motor neurone facial paresis include:

Bell's palsy: idiopathic lower motor neurone facial weakness, assumed to result from a viral neuritis
Herpes zoster (Ramsey Hunt syndrome);
Diabetes mellitus
Lyme disease (borreliosis, Bannwarth's disease)
Sarcoidosis
Leukemic infiltration, lymphoma
HIV seroconversion
Neoplastic compression (*e.g.*, cerebellopontine angle tumor; rare)
Facial nerve neuroma.

These latter conditions may need to be differentiated from Bell's palsy.

Causes of recurrent facial paresis of lower motor neurone type include:

Diabetes mellitus
Lyme disease (borreliosis, Bannwarth's disease)
Sarcoidosis
Leukemia, lymphoma.

In myasthenia gravis, a disorder of neuromuscular transmission at the neuromuscular junction, there may be concurrent ptosis, diplopia, bulbar palsy and limb weakness, and evidence of fatigable weakness.

Myogenic facial paresis may be seen in facioscapulohumeral (FSH) dystrophy, myotonic dystrophy, mitochondrial disorders. In primary

disorders of muscle the pattern of weakness and family history may suggest the diagnosis.

References

Borod JC, Koff E, Lorch MP, Nicholas M, Welkowitz J. Emotional and nonemotional facial behavior in patients with unilateral brain damage. *Journal of Neurology, Neurosurgery and Psychiatry* 1988; **51**: 826-832

Hopf HC, Muller-Forell W, Hopf NJ. Localization of emotional and volitional facial paresis. *Neurology* 1992; **42**: 1918-1923

Jacob A, Cherian PJ, Radhakrishnan K, Sankara SP. Emotional facial paresis in temporal lobe epilepsy: its prevalence and lateralizing value. *Seizure* 2003; **12**: 60-64

Cross References

Abulia; Ageusia; Bell's palsy; Bell's phenomenon, Bell's sign; *Bouche de tapir*; Cerebellopontine angle syndrome; Corneal reflex; Eight-and-a-half syndrome; Epiphora; Fisher's sign; Hitselberg sign; Hyperacusis; Lagophthalmos; Locked-in syndrome; Lower motor neurone (LMN) syndrome; Pseudobulbar palsy; Upper motor neurone (UMN) syndrome

Facilitation

Facilitation is an increase in muscle strength following repeated contraction. Clinically, facilitation may be demonstrated by the appearance of tendon-reflexes after prolonged (*ca*. 30 seconds) forced maximal contractions against resistance, *e.g*., the biceps jerk after elbow flexion, knee jerk after knee extension; and by Lambert's sign (increased force grip with sustained contraction).

This phenomenon of post-tetanic potentiation is most commonly seen in the Lambert-Eaton myasthenic syndrome (LEMS), a disorder of neuromuscular junction transmission associated with the presence of autoantibodies directed against presynaptic voltage-gated calcium ion (Ca^{2+}) channels (VGCC). The mechanism is thought to be related to an increased build up of Ca^{2+} ions within the presynaptic terminal with the repetitive firing of axonal action potentials, partially overcoming the VGCC antibody-mediated ion channel blockade, and leading to release of increasing quanta of acetylcholine.

Cross References

Fatigue; Lambert's sign

"False-Localizing Signs"

Neurological signs may be described as "false-localizing" when their appearance reflects pathology distant from the expected anatomical locus. The classic example, and probably the most frequently observed, is abducens nerve palsy (unilateral or bilateral) in the context of raised intracranial pressure, presumed to result from stretching of the nerve over the ridge of the petrous temporal bone. Many false-localizing signs occur in the clinical context of raised intracranial pressure, either idiopathic (idiopathic intracranial hypertension [IIH]) or symptomatic (secondary to tumor, hematoma, abscess).

A brief topographical overview of false-localizing signs (more details may be found in specific entries) includes:

- Motor system:
  - Kernohan's notch syndrome: false-localizing hemiparesis
  - Cerebellar syndrome with anterior cerebral artery territory infarction damaging frontocerebellar pathways
  - Brainstem compression causing diaphragm paralysis
- Cranial nerves:
  - Proptosis with middle cranial fossa tumor
  - Oculomotor (III) nerve palsy with contralateral supratentorial lesion
  - Divisional oculomotor nerve palsy with brainstem or subarachnoid space pathology
  - Trochlear nerve palsy with IIH
  - Trigeminal nerve palsy with IIH
  - Abducens nerve palsy with IIH
  - Facial nerve palsy with IIH
  - Vestibulocochlear nerve dysfunction with IIH
- Spinal cord and roots:
  - Foramen magnum/upper cervical cord lesion causing hand muscle wasting ("remote atrophy")
  - Lower cervical/upper thoracic myelopathy producing mid-thoracic girdle sensation
  - Urinary retention with rostral spinal cord compression
  - Radiculopathy with IIH, may even mimic Guillain-Barré syndrome

References

Larner AJ. False localizing signs. *Journal of Neurology, Neurosurgery and Psychiatry* 2003; **74**: 415-418

Larner AJ. A topographical anatomy of false-localizing signs. *Advances in Clinical Neuroscience & Rehabilitation* 2005; **5(1)**: 20-21

Cross References

Abducens (vi) Nerve palsy; Divisional palsy; Girdle sensation; Kernohan's notch syndrome; Oculomotor (III) nerve palsy; Proptosis; Urinary retention

Fan Sign *(Signe de l'éventail)*

- see BABINSKI'S SIGN (1)

Fasciculation

Fasciculations are rapid, flickering, twitching, involuntary movements within a muscle belly resulting from spontaneous activation of a bundle, or fasciculus, of muscle fibers (*i.e.*, a motor unit), insufficient to move the joint. Fasciculations may also be induced by lightly tapping over a partially denervated muscle belly. The term was formerly used synonymously with fibrillation, but the latter term is now reserved for contraction of a single muscle fibre, or a group of fibers smaller than a motor unit.

Brief and localized fasciculations can be a normal finding (*e.g.*, in the intrinsic foot muscles, especially abductor hallucis, and gastrocnemius, but not tibialis anterior), particularly if unaccompanied by other neurological symptoms and signs (wasting, weakness, sensory disturbance, sphincter dysfunction). Persistent fasciculations most usually reflect a pathological process involving the lower motor neurones in the anterior (ventral) horn of the spinal cord and/or in brainstem motor nuclei, typically motor neurone disease (in which cramps are an early associated symptom). Facial and perioral fasciculations are highly characteristic of Kennedy's disease (X-linked bulbospinal neuronopathy). However, fasciculations are not pathognomonic of lower motor neurone pathology since they can on rare occasions be seen with upper motor neurone pathology.

The pathophysiological mechanism of fasciculations is thought to be spontaneous discharge from motor nerves, but the site of origin of this discharge is uncertain. Although ectopic neural discharge from anywhere along the lower motor neurone from cell body to nerve terminal could produce fasciculation, the commonly encountered assumption that it originates in the anterior horn cell body is not supported by the available evidence, which points to a more distal origin in the intramuscular nerve terminals. In addition, denervation of muscle fibers may lead to nerve fibre sprouting (axonal and collateral) and enlargement of motor units which makes fasciculations more obvious clinically.

Fasciculations may be seen in:

- Motor neurone disease with lower motor neurone involvement (*i.e.*, progressive muscular atrophy, progressive bulbar atrophy variants)
- Spinal muscular atrophy
- Cervical radiculopathy (restricted to myotomal distribution)
- Multifocal motor neuropathy with conduction block
- Benign fasciculation syndrome: typically seen only after exercise and without associated muscle atrophy or weakness
- Cramp fasciculation syndrome
- Kennedy's disease (X-linked bulbospinal neuronopathy; especially perioral)
- Almost any lower motor neurone disease, especially compression
- Metabolic causes: thyrotoxicosis, tetany, after acetylcholinesterase inhibitors, anesthetic muscle relaxants.

Fasciculations may need to be distinguish from myokymia or neuromyotonia.

References

Blexrud MD, Windebank AJ, Daube JR. Long-term follow-up of 121 patients with benign fasciculations. *Annals of Neurology* 1993; **34**: 622-625

Desai J, Swash M. Fasciculations: what do we know of their significance? *Journal of the Neurological Sciences* 1997; **152 (suppl1)**: S43-S48

Layzer RB. The origin of muscle fasciculations and cramps. *Muscle Nerve* 1994; **17**: 1243-1249

Cross References
Calf hypertrophy; Cramp; Fibrillation; Lower motor neurone (LMN) syndrome; Myokymia; Neuromyotonia

Fast Micrographia

In "fast" micrographia, written letters are microscopic from the outset, sometimes approximating to a straight line, though produced at normal speed without fatigue. This pattern has been observed in progressive supranuclear palsy and with globus pallidus lesions, and contrasts with the "slow" micrographia, writing becoming progressively slower and smaller, seen in idiopathic Parkinson's disease.

References
Quinn NP. Fast micrographia and pallidal pathology. *Journal of Neurology, Neurosurgery and Psychiatry* 2002; **72**: 135 (abstract)

Cross References
Micrographia

Fatigue

The term fatigue may be used in different contexts to refer to both a sign and a symptom.

The sign of fatigue, also known as peripheral fatigue, consists of a reduction in muscle strength with repeated muscular contraction. This most characteristically occurs in disorders of neuromuscular junction transmission (*e.g.*, myasthenia gravis), but it may also be observed in disorders of muscle (*e.g.*, myopathy, polymyositis) and neurogenic atrophy (*e.g.*, motor neurone disease). In myasthenia gravis, fatigue may be elicited in the extraocular muscles by prolonged upgaze causing eyelid drooping; in bulbar muscles by prolonged counting or speech causing hypophonia; and in limb muscles by repeated contraction, especially of proximal muscles (*e.g.*, shoulder abduction) leading to weakness in previously strong muscles. Fatigue in myasthenia gravis is understood as a decline in the amount of acetylcholine released from motor nerve terminals with successive neural impulses, along with a reduced number of functional acetylcholine receptors (AChR) at the motor end-plates, due to binding of AChR antibodies and/or complement mediated destruction of the postsynaptic folds.

(A gradual decline in the amplitude and speed of initiation of voluntary movements, hypometria and hypokinesia, as seen in disorders of the basal ganglia, especially Parkinson's disease, may also be described as fatigue, *e.g.*, "slow" micrographia may be ascribed to "fatigue." Progressive supranuclear palsy is notable for lack of fatigue.)

Fatigue as a symptom, or central fatigue, is an enhanced perception of effort and limited endurance in sustained physical and mental activities. This may occur in multiple sclerosis (MS), post-polio syndrome, post-stroke syndromes, and chronic fatigue syndrome (CFS). In MS and CFS, fatigue may be a prominent and disabling complaint even though neurological examination reveals little or no clinical deficit. This type of fatigue is ill-understood: in MS, frequency-

dependent conduction block in demyelinated axons has been suggested, as has hypothalamic pathology. Current treatment is symptomatic (amantadine, modafinil, 3,4-diaminopyridine) and rehabilitative (graded exercise).

Fatigue may be evaluated with various instruments, such as the Krupp Fatigue Severity Score.

References

Chaudhuri A, Behan PO. Fatigue in neurological disorders. *Lancet* 2004; **363**: 978-988

Zifko UA. Management of fatigue in patients with multiple sclerosis. *Drugs* 2004; **64**: 1295-1304

Cross References

Dystonia; Hypokinesia; Hypometria; Micrographia; Weakness

Femoral Stretch Test

The femoral stretch test, or reverse straight leg raising, consists of extension of the hip with the knee straight with the patient lying prone, a maneuver which puts traction on the femoral nerve or L3 root and may exacerbate pain in a femoral neuropathy or L3 radiculopathy, perhaps due to a retroperitoneal hemorrhage.

Cross References

Lasègue's sign

Fencer's Posture, Fencing Posture

Epileptic seizures arising in or involving the supplementary motor area may lead to adversial head and eye deviation, abduction and external rotation of the contralateral arm, flexion at the elbows, and posturing of the legs, with maintained consciousness, a phenomenon christened by Penfield the "fencing posture" because of its resemblance to the *en garde* position. These may also be known as "salutatory seizures."

Cross References

Seizures

Festinant Gait, Festination

Festinant gait or festination is a gait disorder characterized by rapid short steps (Latin: *festinare*, to hurry, hasten, accelerate) due to inadequate maintenance of the body's centre of gravity over the legs. To avoid falling and to maintain balance the patient must "chase" the centre of gravity, leading to an increasing speed of gait and a tendency to fall forward when walking (propulsion). A similar phenomenon may be observed if the patient is pulled backward (retropulsion). Festination may be associated with freezing of gait.

Festination is common in idiopathic Parkinson's disease; it is associated with longer duration of disease and higher Hoehn & Yahr stage. Festination may be related to the flexed posture and impaired postural reflexes commonly seen in these patients. It is less common in symptomatic causes of parkinsonism, but has been reported, for example in aqueduct stenosis.

References

Leheta O, Boschert J, Krauss JK, Whittle IR. Festination as the leading symptom of late onset idiopathic aqueduct stenosis. *Journal of Neurology, Neurosurgery and Psychiatry* 2002; **73**: 599-600

Cross References

Freezing; Parkinsonism; Postural reflexes

Fibrillation

Fibrillation was previously synonymous with fasciculation, but the term is now reserved for the spontaneous contraction of a single muscle fibre, or a group of fibers smaller than a motor unit, hence this is more appropriately regarded as an electrophysiological sign without clinical correlate.

Cross References

Fasciculation

Finger Agnosia

Finger agnosia is a type of tactile agnosia, in which there is inability to identify which finger has been touched when the eyes are closed, despite knowing that a finger has been touched; or inability to point to or move a finger when it is named; or inability to name the fingers (patient's own fingers or those of another person). This is a disorder of body schema, and may be regarded as a partial form of autotopagnosia.

Finger agnosia is most commonly observed with lesions of the dominant parietal lobe. It may occur in association with acalculia, agraphia, and right-left disorientation, with or without alexia and difficulty spelling words, hence as one feature of Gerstmann syndrome. Isolated cases of finger agnosia in association with left corticosubcortical posterior parietal infarction have been reported. Since this causes no functional deficit, it may be commoner than reported.

References

Della Sala S, Spinnler H. Finger agnosia: fiction or reality? *Archives of Neurology* 1994; **51**: 448-450

Cross References

Agnosia; Autotopagnosia; Gerstmann syndrome

Finger Drop

- see WRIST DROP

Finger-Floor Distance

In patients with leg (+/– back) pain suspected of having lumbosacral nerve root compression, a finger-floor distance of > 25 cm when the patient bends forward and attempts to touch the floor with the fingers has been found an independent predictor of radiological (MR imaging) compression. This was not the case for the straight leg raising test.

References

Vroomen PCAJ, de Krom MCTFM, Wilmink JT, Kester ADM, Knottnerus JA. Diagnostic value of history and physical examination in patients suspected of lumbosacral nerve root compression. *Journal of Neurology, Neurosurgery and Psychiatry* 2002; **72**: 630-634

Cross References

Lasègue's sign

"Finger-Nose Test"

- see ATAXIA; CEREBELLAR SYNDROMES

Fisher's Sign

Fisher's sign is the paucity of facial expression conveying emotional states or attitudes (emotional facial paresis). It follows nondominant (right) hemisphere lesions and may accompany emotional dysprosody of speech.

Cross References

Abulia; Aprosodia, aprosody; Facial paresis

Flaccidity

Flaccidity is a floppiness which implies a loss of normal muscular tone (hypotonia). This may occur transiently after acute lesions of the corticospinal tracts (flaccid paraparesis), before the development of spasticity, or as a result of lower motor neurone syndromes. It is difficult to separate the change in tone from weakness.

Cross References

Hypotonia, Hypotonus; Lower motor neurone (LMN) syndrome

Flail Arm

Flail arm refers to a severe and symmetric wasting and weakness of the arms without significant functional involvement of other regions, seen in one variant of motor neurone disease, the "flail arm syndrome," also known as Vulpian-Bernhart's form. Men are reported to be much more frequently affected than women, and this group may show improved survival compared to other MND patients. Alternative designations for this syndrome include amyotrophic brachial diplegia, dangling arm syndrome, and neurogenic man-in-a-barrel syndrome.

References

Hu MTM, Ellis CM, Al-Chalabi A, Leigh PN, Shaw CE. Flail arm syndrome: a distinctive variant of amyotrophic lateral sclerosis. *Journal of Neurology, Neurosurgery and Psychiatry* 1998; **65**: 950-951

Cross References

Amyotrophy; "man-in-a-barrel"

Flail Foot

- see CAUDA EQUINA SYNDROME; FOOT DROP

Flap, Flapping Tremor

- see ASTERIXIS

Flexibilitas Cerea (Waxy Flexibility)
- see CATATONIA

Flexion-Adduction Sign
Neuralgic amyotrophy (Parsonage-Turner syndrome) may cause arm pain, which may be prevented by holding the arm flexed at the elbow and adducted at the shoulder.

- Waxman SG. The flexion-adduction sign in neuralgic amyotrophy. *Neurology* 1979; **29**: 1301-1304

Flexor Posturing
- see DECORTICATE RIGIDITY

Flick Sign
A flicking, shaking movement of the hands made by patients with carpal tunnel syndrome to try to relieve the paresthesia and pain caused by the condition, typically noted on waking at night.

Cross References

Phalen's sign; Tinel's sign

Floccillation
- see CARPHOLOGIA

Flycatcher Tongue
- see TROMBONE TONGUE

Flynn Phenomenon
Flynn phenomenon is paradoxical constriction of the pupils in darkness. This has been documented in various conditions including congenital achromatopsia, following optic neuritis, and in autosomal dominant optic atrophy.

References

Frank JW, Kushner BJ, France TD. Paradoxical pupillary phenomena: a review of patients with pupillary constriction to darkness. *Archives of Ophthalmology* 1988; **106**: 1564-1566

Cross References

Pupillary reflexes

Foot Drop
Foot drop, often manifest as the foot dragging during the swing phase of the gait, causing tripping and/or falls, may be due to upper or lower motor neurone lesions, which may be distinguished clinically.

- *Stiff foot drop*, with upper motor neurone lesions:
  leads to a circumducting gait; it may be possible to see or hear the foot dragging or scuffing along the floor, and this may cause excessive wear on the point of the shoe. There will be other upper motor neurone signs (hemiparesis; spasticity, clonus, hyperreflexia, Babinski's sign).
- *Floppy foot drop*, with lower motor neurone lesions:

leads to a stepping gait (steppage) to try to lift the foot clear of the floor, and a slapping sound on planting the foot. At worst, there is a flail foot in which both the dorsiflexors and the plantar flexors of the foot are weak (*e.g.*, in high sciatic nerve or sacral plexus lesions). Other lower motor neurone signs may be present (hypotonia, areflexia or hyporeflexia).

Causes of floppy foot drop include:

Common peroneal nerve palsy
Sciatic neuropathy
Lumbosacral plexopathy
L4/L5 radiculopathy
Motor or sensorimotor polyneuropathy (*e.g.*, hereditary motor and sensory neuropathy)
Motor neuronopathy (anterior horn cell disease)
Mononeuropathy multiplex

These may be distinguished on clinical and/or neurophysiological grounds

References

McNamara B. Foot drop. *Advances in Clinical Neuroscience & Rehabilitation* 2003; **3(1)**: 24-25

Cross References

Cauda equina syndrome; Hemiparesis; Lower motor neurone (LMN) syndrome; Steppage, stepping gait; Upper motor neurone (UMN) syndrome

Foot Grasping

- see GRASP REFLEX

Forced Ductions

Forced ductions, performed by grasping the anesthetized sclera with forceps and then moving the eye through its range of motions, may be used to determine whether restricted eye movement is mechanical, due to a lesion within the orbit, such as thyroid ophthalmopathy or superior oblique tendon sheath (Brown's) syndrome.

Forced Grasping

- see GRASP REFLEX

Forced Groping

Forced groping describes involuntary movements of a hand, as if searching for an object or item which has touched or brushed against it; the hand may follow the object around if it moves (magnetic movements). There may be an accompanying grasp reflex. This type of behavior may be displayed by an alien hand, most usually in the context of corticobasal degeneration. Forced groping may be conceptualized as an exploratory reflex which is "released" from frontal lobe control by a pathological process, as in utilization behavior.

References

Adie WS, Critchley M. Forced grasping and groping. *Brain* 1927; **50**: 142-170

Cross References

Alien hand, alien limb; Grasp reflex; Magnetic movements; Utilization behavior

Forced Laughter and Crying

- see EMOTIONALISM, EMOTIONAL LIABILITY; PATHOLOGICAL CRYING, PATHOLOGICAL LAUGHTER

Forced Upgaze

Tonic upward gaze deviation, forced upgaze, may be seen in coma after diffuse hypoxic-ischemic brain injury with relative sparing of the brainstem. Forced upgaze may also be psychogenic, in which case it is overcome by cold caloric stimulation of the ear drums. Forced upgaze must be differentiated from oculogyric crisis.

Cross References

Oculogyric crisis

Foreign Accent Syndrome

- see APHEMIA

Formication

- see PARESTHESIA; TINEL'S SIGN

Fortification Spectra

Fortification spectra, also known as teichopsia, are visual hallucinations which occur as an aura, either in isolation (migraine without headache) or prior to an attack of migraine (migraine with aura; "classical migraine"). The appearance is a radial array likened to the design of medieval castles, not simply of battlements. Hence these are more complex visual phenomena than simple flashes of light (photopsia) or scintillations. They are thought to result from spreading depression, of possible ischemic origin, in the occipital cortex.

Cross References

Aura; Hallucination; Photopsia

Foster Kennedy Syndrome

The Foster Kennedy syndrome consists of optic atrophy in one eye with optic disc edema in the other eye, due to a tumor compressing one optic nerve (to produce atrophy) and causing raised intracranial pressure (to produce contralateral papilledema). A pseudo-Foster Kennedy syndrome is described in consecutive acute ischemic optic neuropathy.

References

Kennedy F. Retrobulbar neuritis as an exact diagnostic sign of certain tumors and abscesses in the frontal lobe. *American Journal of Medical Science* 1911; **142**: 355-368.

Cross References
Optic atrophy; Papilledema

Freezing

Freezing is the sudden inability in a patient with parkinsonism to move or to walk, *i.e.*, gait failure, as though the patient were turned to ice or the feet were nailed to the floor. This is one of the unpredictable motor fluctuations in late Parkinson's disease (associated with longer duration of disease and treatment) which may lead to falls, usually forward onto the knees, and injury. It may occur in confined spaces (*e.g.*, doorways), when trying to turn, or when trying to do two things at once. It is not seen in the early years of levodopa therapy.

Two variants are encountered, occurring either during an off period or wearing off period, or randomly, *i.e.*, unrelated to drug dosage or timing.

Treatment strategies include use of dopaminergic agents and, anecdotally, L-threodops, but these agents are not reliably helpful, particularly in random freezing. Use of visual targets (real or imagined) may help, *e.g.*, stepping over a line.

Freezing may also occur in multiple system atrophy, and has also been reported as an isolated phenomenon.

Cross References
Parkinsonism

Fregoli Syndrome

- see DELUSION

Froment's Sign

Froment has two eponymous signs:

- Activated rigidity or synkinesis (*q.v.*).
- In an ulnar nerve lesion, flexion of the distal phalanx of the thumb (flexor pollicis longus, innervated by the median nerve) is seen when attempting to squeeze a sheet of paper between the thumb and the index finger, as a compensation for the weakness of thumb adduction (adductor pollicis, innervated by the ulnar nerve), also known as Froment's prehensile thumb sign or the *signe du journal*. The term is also sometimes used for weakness of little finger adduction, evident when trying to grip a piece of paper between the ring and little finger.

Cross References
Rigidity; Synkinesia, synkinesis

Frontal Ataxia

- see ATAXIA

Frontal Lobe Syndromes

The frontal lobes of the brain have enlarged greatly during phylogeny; their diverse connections with the basal ganglia, basal forebrain, and

cerebellum, as well as other cortical areas, reflect their multiple motor and behavioral functions. Damage to the frontal lobes may produce a variety of clinical signs, most frequently changes in behavior. Such changes may easily be overlooked with the traditional neurological examination, although complained of by patient's relatives, and hence specific bedside tests of frontal lobe function should be utilized, for example:

- Verbal fluency: *e.g.*, letter/phonemic (F, A, S) probably a more specific test than category/semantic (animals, foods).
- Proverb interpretation: *e.g.*, "Make hay while the sun shines"; "Too many cooks spoil the broth"; interpretation tends to be concrete in frontal lobe disorders.
- Cognitive estimates: *e.g.*, height of the Post Office Tower, length of a man's spine, distance from London to Edinburgh; may be grossly abnormal or inappropriate.
- Copying motor sequences, to assess motor programming ability: *e.g.*, Luria fist-edge-palm test (three step motor sequence with hand).
- Alternating sequence tests: *e.g.*, alternating finger flexion/extension out of phase in two hands, or repeatedly writing m n m n m n (also used as tests of praxis, which may be affected with frontal lobe pathology); swapping a coin from hand to hand behind back in a predictable pattern and asking the patient which hand the coin is in.
- Set-shifting or go/no go tests, in which an alternating pattern is suddenly changed, *e.g.*, changing the previously predictable (left/right) pattern of coin hidden in clenched hand swapped over behind back; rhythmic tapping with pen on a surface (I tap once, you tap twice; I tap twice, you tap once); tests of response inhibition (ask patient to clap three times, s/he does so multiple times).

A useful clinico-anatomical classification of frontal lobe syndromes which reflects the functional subdivisions of the frontal lobes is as follows:

- *Orbitofrontal Syndrome* ("disinhibited"):
  Disinhibited behavior (including sexual disinhibition), impulsivity
  Inappropriate affect, *witzelsucht*, euphoria
  Emotional lability (moria)
  Lack of judgment, insight
  Distractibility, lack of sustained attention; hypermetamorphosis
  Motor perseverations are not a striking feature
- *Frontal Convexity Syndrome* ("apathetic"):
  Apathy; abulia, indifference
  Motor perseveration
  Difficulty set-shifting, stimulus boundedness
  Reduced verbal fluency
  Deficient motor programming, *e.g.*, three step hand sequence, rhythmical tapping (go/no-go test)
- *Medial Frontal Syndrome* ("akinetic"):
  Little spontaneous movement, bradykinesia, hypokinesia
  Sparse verbal output (akinetic mutism)

Urinary incontinence
Sensorimotor signs in lower limbs
Indifference to pain

Overlap between these regional syndromes may occur.

A "*dysexecutive syndrome*" has also been defined, consisting of difficulty planning, adapting to changing environmental demands (impaired cognitive flexibility, *e.g.*, in set-shifting tests), and directing attentional resources. This may be seen with dorsolateral (prefrontal) damage.

These frontal lobe syndromes may be accompanied by various neurological signs (frontal release signs or primitive reflexes). Other phenomena associated with frontal lobe pathology include imitation behaviors (echophenomena) and, less frequently, utilization behavior, features of the environmental dependency syndrome.

References

Larner AJ, Leach JP. Phineas Gage and the beginnings of neuropsychology. *Advances in Clinical Neuroscience & Rehabilitation* 2002; **2(3)**: 26

Parkin AJ. *Explorations in cognitive neuropsychology*. Hove: Psychology Press, 1996: 220-242

Trimble MR. *Biological psychiatry* (2nd edition). Chichester: Wiley, 1996: 147-156

Cross References

Abulia; Akinesia; Akinetic mutism; Apathy; Attention; Disinhibition; Dysexecutive syndrome; Emotionalism, Emotional lability; Frontal release signs; Hypermetamorphosis; Hyperorality; Hyperphagia; Hypersexuality; Incontinence; Perseveration; Utilization behavior; *Witzelsucht*

Frontal Release Signs

Frontal release signs are so named because of the belief that they are released from frontal inhibition by diffuse pathology within the frontal lobes (usually vascular or degenerative) with which they are often associated, although they may be a feature of normal ageing. Some of these responses are present during infancy but disappear during childhood, hence the terms "primitive reflexes" or "developmental signs" are also used (Babinski's sign may therefore fall into this category). The term "psychomotor signs" has also been used since there is often accompanying change in mental status.

The frontal release signs may be categorized as:

- *Prehensile*:
  - Sucking reflex (tactile, visual)
  - Grasp reflex: hand, foot
  - Rooting reflex (turning of the head toward a tactile stimulus on the face)
- *Nociceptive*:
  - Snout reflex
  - Pout reflex
  - Glabellar (blink) reflex
  - Palmomental reflex

The corneomandibular and nuchocephalic reflexes may also be categorized as "frontal release" signs. Some are of little clinical value

(*e.g.*, palmomental reflex). Concurrent clinical findings may include dementia, gait disorder (frontal gait, *marche à petit pas*), urinary incontinence, akinetic mutism and *gegenhalten*.

Common causes of these findings are diffuse cerebrovascular disease and motor neurone disease.

References

Franssen EH. Neurologic signs in ageing and dementia. In: Burns A (ed.). *Ageing and dementia: A methodological approach*. London: Edward Arnold, 1993: 144-174

Cross References

Age-related signs; Babinski's sign (1); Corneomandibular reflex; *Gegenhalten*; Grasp reflex; *Marche à petit pas;* Palmomental reflex; Pout reflex; Rooting reflex; Sucking reflex

Fugue

Fugue, and fugue-like state, are used to refer to a syndrome characterized by loss of personal memory (hence the alternative name of "twilight state"), automatic and sometimes repetitive behaviors, and wandering or driving away from normal surroundings.

Fugue may be:

Psychogenic: associated with depression (sometimes with suicide); alcoholism, amnesia; "hysteria";

Epileptic: complex partial seizures

Narcoleptic

Some patients with frontotemporal dementia may spend the day walking long distances, and may be found a long way from home, unable to give an account of themselves, and aggressive if challenged; generally they are able to find their way home (spared topographical memory) despite their other cognitive deficits.

Cross References

Amnesia; Automatism; Dementia; Poriomania; Seizures

Functional Weakness and Sensory Disturbance

Various signs have been deemed useful indicators of functional or "nonorganic" neurological illness, including:

Collapsing or "give way" weakness
Hoover's sign
Babinski's trunk-thigh test
"Arm drop"
Belle indifférence
Sternocleidomastoid sign
Midline splitting sensory loss
Functional postures, gaits:
- Monoplegic "dragging"
- Fluctuation of impairment
- Excessive slowness, hesitation
- "Psychogenic Romberg" sign
- "Walking on ice"

Uneconomic posture, waste of muscle energy
Sudden knee buckling

Although such signs may be suggestive, their diagnostic utility has never been formally investigated in prospective studies, and many, if not all, have been reported with "organic" illness. Hence it is unwise to rely on them as diagnostic indicators.

References

Lempert T, Brandt T, Dieterich M *et al*. How to identify psychogenic disorders of stance and gait: a video study in 37 patients. *Journal of Neurology* 1991; **238**: 140-146

Stone J, Zeman A, Sharpe M. Functional weakness and sensory disturbance. *Journal of Neurology, Neurosurgery and Psychiatry* 2002; **73**: 241-245

Cross References

"Arm drop"; Babinski's trunk-thigh test; *Belle indifférence*; Collapsing weakness; Hoover's sign; Sternocleidomastoid test

Funnel Vision

- see "TUNNEL VISION"