F

"Face-Hand Test"
- see "ARM DROP"

Facial Paresis, Facial Weakness
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 thus clinically heterogeneous: not all facial weakness is Bell's palsy.

- *Upper motor neurone facial weakness* ("central facial palsy"):

  The ability to raise the eyebrow is preserved due to the 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. Conversely, volitional paresis without emotional paresis may occur when corticobulbar fibres 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 tumour, 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).

A.J. Larner, *A Dictionary of Neurological Signs*,
DOI 10.1007/978-3-319-29821-4_6

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 the corneal 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 hypoaesthesia (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 (neuroborreliosis, Bannwarth's disease).
- Sarcoidosis.
- Leukaemic infiltration, lymphoma.
- HIV seroconversion.
- Neoplastic compression (*e.g.* cerebellopontine angle tumour; rare).
- Facial nerve neuroma.

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

In bilateral facial weakness, particular consideration should be given to the possibility of:

- Guillain-Barré syndrome.
- HIV.
- Lyme disease.
- Sarcoidosis.
- Malignant infiltration.
- Amyloidosis.

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

- Diabetes mellitus.
- Lyme disease.
- Sarcoidosis.
- Leukaemia, 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, and 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 non-emotional facial behaviour in patients with unilateral brain damage. *J Neurol Neurosurg Psychiatry.* 1988; **51**: 826–32.

Hopf HC, Muller-Forell W, Hopf NJ. Localization of emotional and volitional facial paresis. *Neurology*. 1992; **42**: 1918–23.

Jacob A, Cherian PJ, Radhakrishnan K, Sankara SP. Emotional facial paresis in temporal lobe epilepsy: its prevalence and lateralizing value. *Seizure*. 2003; **12**: 60–4.
Masterson L, Vallis M, Quinlivan R, Prinsley P. Assessment and management of facial nerve palsy. *BMJ*. 2015; **351**: h3725.

Cross References

Abulia; Ageusia; Bell's palsy; Bell's phenomenon, Bell's sign; *Bouche de tapir*; Cerebellopontine angle syndrome; Clapham's sign; 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 describes the increase in impulse transmission across the neuromuscular junction detected following repeated muscle contraction (*cf.* fatigue). Clinically, facilitation may be demonstrated by the brief appearance of tendon reflexes which are absent at rest following prolonged (*ca*. 10–30 s) forced maximal contraction against resistance, *e.g.* the biceps jerk after elbow flexion, knee jerk after knee extension; and by Lambert's sign (increased force of grip with sustained contraction; this increase in strength of affected muscles detected in the first few seconds of maximal voluntary contraction may also be known as augmentation) Increments in compound muscle action potentials are seen after exercise or high frequency repetitive nerve stimulation.

These phenomenona of post-tetanic potentiation are 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

Augmentation; Fatigue; Lambert's sign

"False-Localising Signs"

Neurological signs may be described as "false-localising" when their appearance reflects pathology distant from the expected anatomical locus. The classic example, and probably the most frequently observed, is abducens (VI) 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-localising signs occur in the clinical context of raised intracranial pressure, either idiopathic (idiopathic intracranial hypertension [IIH]) or symptomatic (secondary to tumour, haematoma, abscess). A brief topographical overview of false-localising signs, moving from central to peripheral, includes:

- Motor system:
  - Amyotrophy of parietal lobe origin affecting the upper limb (Silverstein syndrome).
  - Kernohan's notch syndrome: false-localising hemiparesis.
  - Cerebellar syndrome with anterior cerebral artery territory infarction damaging frontocerebellar pathways.
  - Brainstem compression causing diaphragm paralysis.
- Sensory system:
  - Sensory level with parietal lobe lesion (with or without amyotrophy of upper limb muscles: Christiansen-Silverstein syndrome).
  - Hemianopia due to raised intracranial pressure if temporal lobe herniation causes posterior cerebral artery compromise.
  - Radicular symptoms (pseudoradicular syndrome) with thalamic lesion.

- Cranial nerves:
  - Proptosis with middle cranial fossa tumour.
  - 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.

(More details may be found in specific entries.)

Of note, some of these so-called false localising signs may simply reflect incorrect diagnosis: for example, virtually all reported cases of Silverstein or Christiansen-Silverstein syndrome predate the advent of modern neuroimaging techniques.

References

Larner AJ. False localising signs. *J Neurol Neurosurg Psychiatry.* 2003; **74**: 415–18.
Larner AJ. A topographical anatomy of false-localising signs. *Adv Clin Neurosci Rehabil*. 2005; **5**(1): 20–1.
O'Connell JE. Trigeminal false localizing signs and their causation. *Brain*. 1978; **101**: 119–42.

Cross References

Abducens (VI) nerve palsy; Divisional palsy; "Dorsal guttering"; Girdle sensation; Kernohan's notch syndrome; Oculomotor (III) nerve palsy; Proptosis; Pseudoradicular syndrome; Urinary retention

Fan Sign (*Signe De L'éventail*)

- see BABINSKI'S SIGN (1)

Fasciculation

Fasciculation refers to rapid, flickering, twitching, involuntary movements within a muscle belly resulting from spontaneous activation of a bundle, or fasciculus, of muscle fibres (*i.e.* a motor unit), insufficient to produce movement around a joint. Fasciculation 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 fibres smaller than a motor unit. Fasciculation may need to be distinguished clinically and neurophysiologically from myokymia or neuromyotonia.

Brief and localized fasciculation 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). Such benign fasciculation may cause anxiety, particularly in clinicians.

Persistent fasciculation most usually reflects 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 spinal and bulbar muscular

atrophy (Kennedy's disease). However, fasciculation is not pathognomonic of lower motor neurone pathology since it may on rare occasions be seen with upper motor neurone pathology.

The pathophysiological mechanism of fasciculation 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 this originates from the anterior horn cell body is not entirely supported by the available evidence, which points to an additional, more distal, origin in the motor axons. Denervation of muscle fibres may lead to nerve fibre sprouting (axonal and collateral) with subsequent enlargement of motor units which makes fasciculation more obvious clinically.

Fasciculation may be seen in:

- Motor neurone disease (amyotrophic lateral ascelrosis) with lower motor neurone involvement (*i.e.* progressive muscular atrophy, progressive bulbar atrophy, flail limb variants).
- Other motor neurone disorders: spinal muscular atrophy; spinal and bulbar muscular atrophy (Kennedy's disease, X-linked bulbospinal muscular atrophy), especially perioral: chin fasciculations may be pathognomonic for Kennedy's disease; facial onset sensory and motor neuronopathy (FOSMN).
- 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. Extensive longitudinal follow-up may be required to confirm the "benign" label.
- Almost any lower motor neurone disease, especially compression.
- Metabolic causes: thyrotoxicosis, tetany, after treatment with acetylcholinesterase inhibitors, anaesthetic muscle relaxants.

References

Desai J, Swash M. Fasciculations: what do we know of their significance? *J Neurol Sci*. 1997; **152**(suppl1): S43–8.

Kleine BU, Stegeman DF, Schelhaas HJ, Zwarts MJ. Firing pattern of fasciculations in ALS: evidence for axonal and neuronal origin. *Neurology*. 2008; **70**: 353–9.

Simon NG, Kiernan MC. Fasciculation anxiety syndrome in clinicians. *J Neurol.* 2013; **260**: 1743–7.

Singh V, Gibson J, McLean B, Boggild M, Silver N, White R. Fasciculations and cramps: how benign? Report of four cases progressing to ALS. *J Neurol.* 2011; **258**: 573–8.

Cross References

Calf hypertrophy; Cramp; Fibrillation; Flail arm; Lower motor neurone (LMN) syndrome; Myokymia; Neuromyotonia

Fast Micrographia

In "fast" micrographia, written letters are small from the outset of writing, 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 "slow" micrographia, writing which becomes progressively slower and smaller, as seen in idiopathic Parkinson's disease.

References

Meenakshisundaram U, Velmurugendran CU, Prabash PR. Fast micrographia: an unusual but distinctive sign. *Ann Indian Acad Neurol*. 2013; **16**: 172–3.

Quinn NP. Fast micrographia and pallidal pathology. *J Neurol Neurosurg Psychiatry*. 2002; **72**: 135. (abstract).

Cross Reference
Micrographia

Fatigue

The term fatigue may be used in different neurological 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 or endurance 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, "wing flaps"), leading to weakness in previously strong muscles. Fatigue in myasthenia gravis is thought to be caused by 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. Decrements in compound muscle action potentials are seen after repetitive nerve stimulation.

Fatigue may also be used to describe 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, *e.g.* "slow" micrographia may be ascribed to "fatigue". Progressive supranuclear palsy is notable for lack of such 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. Fatigue may be evaluated with various instruments, such as the Krupp Fatigue Severity Score. 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, modafanil, 3,4-diaminopyridine) and rehabilitative (graded exercise).

References

Chaudhuri A, Behan PO. Fatigue in neurological disorders. *Lancet*. 2004; **363**: 978–88.
Induruwa I, Constantinescu CS, Gran B. Fatigue in multiple sclerosis – a brief review. *J Neurol Sci.* 2012; **323**: 9–15.

Cross References
Dystonia; Hypokinesia; Hypometria; Micrographia; Weakness

Fehlleistungen

- see PARAPRAXIA, PARAPRAXIS

Femoral Nerve Stretch Test

The femoral nerve stretch test (FNST), or reverse straight leg raising test, consists of extension of the hip with the knee straight with the patient lying prone, a manoeuvre which exerts traction on the femoral nerve or L3 root and may exacerbate pain in a femoral neuropathy or L3 radiculopathy, perhaps caused by a retroperitoneal haemorrhage. Crossed FNST is reported to be sensitive for high lumbar radiculopathy.

Typical ipsilateral sciatic pain induced by the performing FNST may be indicative of a L4/L5 intervertebral disc protrusion.

References

Christodoulides AN. Ipsilateral sciatica on femoral nerve stretch test is pathognomonic of an L4/5 disc protrusion. *J Bone Joint Surg Br.* 1989; **71**: 88–9.

Nadler SF, Malanga GA, Stitik TP, Keswani R, Foye PM. The crossed femoral nerve stretch test to improve diagnostic sensitivity for the high lumbar radiculopathy: 2 case reports. *Arch Phys Med Rehabil*. 2001; **82**: 522–3.

Cross Reference

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 adopted by fencers. These seizures may also be known as "salutatory seizures".

Cross Reference

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 backwards (retropulsion). Festination may also 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 of disease. 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

Giladi N, Shabtai H, Rozenberg E, Shabtai E. Gait festination in Parkinson's disease. *Parkinsonism Relat Disord.* 2001; **7**: 135–8.

Leheta O, Boschert J, Krauss JK, Whittle IR. Festination as the leading symptom of late onset idiopathic aqueduct stenosis. *J Neurol Neurosurg Psychiatry.* 2002; **73**: 599–600.

Morris ME, Iansek R, Galna B. Gait festination and freezing in Parkinson's disease: pathogenesis and rehabilitation. *Mov Disord.* 2008; **23**(Suppl2): S451–60. [Erratum *Mov Disord.* 2008; **23**: 1639–40].

Cross References

Freezing of gait; 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 fibres smaller than a motor unit, hence this is more appropriately regarded as a sign detected on neurophysiological testing (fibrillation potential) without clinical correlate.

Cross Reference

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. It

may be more common in indivduals with synaesthesia. 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 more common than reported. It may be found in Alzheimer's disease.

References

Davis AS, Trotter JS, Hertza J, Bell CD, Dean RS. Finger agnosia and cognitive deficits in patients with Alzheimer's disease. *App Neuropsychol Adult.* 2012; **19**: 116–20.

Della Sala S, Spinnler H. Finger agnosia: fiction or reality? *Arch Neurol*. 1994; **51**: 448–50.

Cross References

Agnosia; Autotopagnosia; Gerstmann syndrome; Synaesthesia

"Finger Chase"

- see ATAXIA; CEREBELLAR SYNDROMES

Finger Drop

- see WRIST DROP

Finger-Floor Distance

In patients with leg (+/− low 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 to be an independent predictor of radiological (MR imaging) compression. This was not the case for the straight leg raising test.

Reference

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. *J Neurol Neurosurg Psychiatry*. 2002; **72**: 630–4.

Cross Reference

Lasègue's sign

"Finger-to-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, Facial weakness

Fist-Edge-Palm Test

In the fist-edge-palm test, sometimes known as the Luria test or three step motor sequence, the patient is requested to place the hand successively in three positions, imitating movements made by the examiner and then doing them alone: fist, vertical palm, palm resting flat on table. Copying motor sequences assesses motor programming ability. Defects in this programming, such as lack of kinetic melody, loss of sequence, or repetition of previous pose or position, are especially conspicuous with anterior cortical lesions. This test is incorporated into the Frontal Assessment Battery.

References

Dubois B, Slachevsky A, Litvan I, Pillon B. The FAB: a frontal assessment battery at bedside. *Neurol.* 2000; **55**: 1621–6.

Luria AR. Higher cortical functions in man. 2nd ed. New York: Basic Books; 1980. p. 423–4.

Cross Reference

Frontal lobe syndromes

Flaccidity
Flaccidity refers to 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 sometimes difficult to separate the change in tone from concurrent weakness.
Cross References
Hypotonia, Hypotonus; Lower motor neurone (LMN) syndrome

Flail Foot
- see CAUDA EQUINA SYNDROME; FOOT DROP

Flail Limb
Flail limb (flail arm, flail leg) refers to severe and symmetrical limb wasting and weakness without significant functional involvement of other regions. These are variant forms of motor neurone disease (amyotrophic lateral sclerosis), the "flail arm syndrome" having previously been described as Vulpian-Bernhart's form. Alternative designations for flail arm syndrome include amyotrophic brachial diplegia, dangling arm syndrome, and neurogenic man-in-a-barrel syndrome.

Men are reported to be much more frequently affected than women, and this group as a whole shows improved survival compared to other motor neurone disease patients. Cognition may be relatively well preserved, as for other isolated lower motor neurone forms of motor neurone disease.
References
Larner AJ. Neuropsychological neurology. The neurocognitive impairments of neurological disorders. Cambridge: Cambridge University Press; 2008. p. 68.
Wijesekera LC, Mathers S, Talman P, et al. Natural history and clinical features of the flail arm and flail leg ALS variants. *Neurology*. 2009; **72**: 1087–94.
Cross References
Amyotrophy; "Man-in-the-barrel"

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.
Reference
Waxman SG. The flexion-adduction sign in neuralgic amyotrophy. *Neurology*. 1979; **29**: 1301–4.

Flexor Posturing
- see DECORTICATE RIGIDITY

Flexor Spasms
- see SPASM

"Flick Sign"
A flicking, shaking movement of the hands made by patients with carpal tunnel syndrome to try to relieve the paraesthesia and pain caused by the condition, typically noted on waking at night, may be described as the "flick sign". This may be the most sensitive and specific of the various signs described in carpal tunnel syndrome.

References

D'Arcy CA, McGee S. Does this patient have carpal tunnel syndrome? *JAMA*. 2000; **283**: 3110–7.
Hi ACF, Wong S, Griffith J. Carpal tunnel syndrome. *Pract Neurol*. 2005; **5**: 210–7.

Cross References

Phalen's sign; Tinel's sign

Floccillation

- see CARPHOLOGIA, CARPHOLOGY

Flycatcher Tongue

- see TROMBONE TONGUE

Flynn Phenomenon

The 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.

Reference

Frank JW, Kushner BJ, France TD. Paradoxic pupillary phenomena: a review of patients with pupillary constriction to darkness. *Arch Ophthalmol.* 1988; **106**: 1564–6.

Cross Reference

Pupillary reflexes

"*Folles Larmes Prodromiques*"

- see *FOU RIRE PRODROMIQUE*

"Fonzarelli" Sign

This name has been given to focal thumb dystonia in Parkinson's disease, a movement reminiscent of the "thumbs up" gesture associated with the character of Arthur "Fonzie" Fonzarelli in the US TV sitcom *Happy Days* broadcast in the 1970s and 1980s.

Reference

Turner MR, Matthews L, Ebers GC. Teaching video NeuroImage: the "Fonzarelli" sign: focal thumb dystonia as an early manifestation of Parkinson disease. *Neurology*. 2008; **71**: e11.

Cross Reference

Dystonia

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).

  Causes of stiff foot drop include:

  - Cerebral infarct.
  - Motor neurone disease.
- *Floppy foot drop, with lower motor neurone lesions*:

  leads to a stepping gait (steppage) to try to lift the foot clear of the floor in the swing phase, 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:

- Isolated common peroneal nerve palsy: the most common cause, usually due to compression of the nerve at the head of the fibula, causing painless weakness in foot dorsiflexion and eversion, sensory loss on the anterolateral leg and dorsum of the foot, with normal reflexes; may have a Tinel sign on tapping the nerve over the fibular head.
- Sciatic neuropathy (rare).
- Lumbosacral plexopathy (rare; may be painful).
- L4/L5 radiculopathy: may also have hip abduction weakness, and sensory loss conforming to dermatomal boundaries; may be painful.
- Motor or sensorimotor polyneuropathy (*e.g.* hereditary motor and sensory neuropathy).
- Motor neuronopathy (anterior horn cell disease: look for fasciculation).
- Mononeuropathy multiplex (may be painful).

These may be distinguished on clinical and/or neurophysiological grounds. Foot drop may also occur with myopathies and muscular dystrophies.

Reference

Stevens F, Weerkamp NJ, Cals JWL. Foot drop. *BMJ*. 2015; **350**: h1736.

Cross References

Cauda equina syndrome; Dermatomal sensory loss; Hemiparesis; Lower motor neurone (LMN) syndrome; Neuropathy; Steppage, Stepping gait; Upper motor neurone (UMN) syndrome

Foot Grasping

- see GRASP REFLEX

Forced Ductions

Forced ductions, performed by grasping the anaesthetised 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 behaviour may be displayed by an alien hand, most usually in the context of corticobasal degeneration. Forced groping may be conceptualised as an exploratory reflex which is "released" from frontal lobe control by a pathological process, as in utilization behaviour.

Reference

Adie WS, Critchley M. Forced grasping and groping. *Brain*. 1927; **50**: 142–70.

Cross References

Alien hand, Alien limb; Grasp reflex; Magnetic movements; Utilization behaviour

Forced Laughter and Crying

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

Forced Upgaze

Forced upgaze describes tonic upward gaze deviation, which may be seen in coma after diffuse hypoxic-ischaemic 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 clinically from oculogyric crisis.

Cross References

Caloric testing; Coma; Oculogyric crisis

Forearm and Finger Rolling

The forearm and finger rolling tests detect subtle upper motor neurone lesions with high specificity and modest sensitivity. Either the forearms or the index fingers are rapidly rotated around each other in front of the torso for about 5 s, then the direction is reversed. Normally the appearance is symmetrical but with a unilateral upper motor neurone lesion one arm or finger remains relatively stationary, with the normal rotating around the abnormal limb. Thumb rolling may also be a sensitive test for subtle upper motor neurone pathology.

Reference

Anderson NE. The forearm and finger rolling tests. *Pract Neurol*. 2010; **10**: 39–42.

Cross References

Bed cycling test; Pronator drift; Thumb rolling test; Upper motor neurone (UMN) syndrome

Foreign Accent Syndrome

The foreign accent syndrome is a rare phonological disorder, such that speech production includes non-native vowels or consonants and hence sounds as though it is foreign, or different from the speaker's native intonation. There is no language disorder since comprehension of spoken and written language is preserved; hence it is qualitatively different from Broca's aphasia. This syndrome probably overlaps with other disorders of speech production, labelled as phonetic disintegration, pure anarthria, aphemia, apraxic dysarthria, verbal or speech apraxia, and cortical dysarthria. Case heterogeneity is noted; some cases may be non-organic.

References

Kurowski KM, Blumstein SE, Alexander M. The foreign accent syndrome: a reconsideration. *Brain Lang*. 1996; **54**: 1–25.
Monrad-Krohn GH. Dysprosody or altered "melody" of language. *Brain*. 1947; **70**: 405–15.
Ryalls J, Miller N. Foreign accent syndromes: the stories people have to tell. Hove: Psychology Press; 2014.

Cross References

Aphasia; Aphemia; Aprosodia, Aprosody

Formication

Formication is a tactile or haptic visceral hallucination, as of ants crawling over the skin. It may occur in isolation, perhaps reflecting spontaneous neuronal firing from cutaneous afferents reaching conscious threshold, or in the context of Ekbom's syndrome (delusional parasitosis).

Cross References

Ekbom's syndrome; Hallucination; Paraesthesia; Tinel's sign

Fortification Spectra

Fortification spectra, also known as teichopsia, are visual hallucinations which occur as an aura, either in isolation (migraine aura 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 ischaemic origin, in the occipital cortex. The term was

first used by John Fothergill (1712–1780) in his 1778 publication on "Sick Head-Ach" (sic), from which he himself suffered. The visions of Hildegard von Bingen (1098–1179), illustrated in the 12th century, are thought possibly to reflect migrainous fortification spectra.

References

Fothergill J. Remarks on that complaint commonly known under the name of the Sick-Head-ach. *Med Obs Inq.* 1778; **3**: 219–43.
Plant GT. The fortification spectra of migraine. *BMJ*. 1986; **293**: 1613–7.
Singer C. The visions of Hildegard of Bingen. In: From magic to science. Essays on the scientific twilight. London: Ernest Benn; 1928.p. 199–239.

Cross References

Aura; Hallucination; Photopsia; Teichopsia

Foster Kennedy Syndrome

The Foster Kennedy syndrome consists of optic atrophy in one eye with optic disc oedema in the other eye. Anosmia ipsilateral to the optic atrophy may also be found.

This syndrome is classically due to a tumour, typically an olfactory groove meningioma, which compresses the ipsilateral optic nerve to cause atrophy, and also causes raised intracranial pressure with consequent contralateral papilloedema.

Similar clinical appearances have been reported in other conditions, sometimes called a pseudo-Foster Kennedy syndrome, such as sequential anterior ischaemic optic neuropathy and idiopathic intracranial hypertension.

Reference

Kennedy F. Retrobulbar neuritis as an exact diagnostic sign of certain tumors and abscesses in the frontal lobe. *Am J Med Sci*. 1911; **142**: 355–68.

Cross References

Disc swelling; Optic atrophy; Papilloedema

Fou Rire Prodromique

Fou rire prodromique, or laughing madness, was first described by Féré in 1903. It consists of pathological (mood incongruent) laughter which heralds the development of stroke, typically in the brainstem as a consequence of basilar artery occlusion but sometimes in the capsular genu.

Pathological crying as a prodrome of brainstem stroke has also been described ("*folles larmes prodromiques*").

References

Coelho M, Ferro JM. Fou rire prodromique. Case report and systematic review of the literature. *Cerebrovasc Dis.* 2003; **16**: 101–4.
Larner AJ. Basilar artery occlusion associated with pathological crying: "folles larmes prodromiques"? *Neurology*. 1998; **51**: 916–7.
Larner AJ. Charles Féré (1852-1907). *J Neurol*. 2011; **258**: 524–5.

Cross Reference

Pathological crying, Pathological laughter

Freezing of Gait

Freezing of gait is defined as a brief episodic absence or a marked reduction of forward progression of the feet despite the intention to walk. This occurs in Parkinson's disease and other forms of parkinsonism (progressive supranucelar palsy, vascular parkinsonism). Various clinical patterns are reported, including trembling in place, shuffling forward, and complete akinesia.

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 at gait initiaition, in confined spaces (*e.g.* doorways), when trying to turn, or when trying to do two things at once. Freezing of gait may occur either during an off period or wearing off period, or randomly, *i.e.* unrelated to drug dosage or timing.

There are no clear treatment protocols for freezing of gait. Strategies include use of dopaminergic agents and, anecdotally, L-threodops, but these agents are not reliably helpful, particularly in random freezing. Methylphenidate has also been tried. Use of visual targets (real or imagined) may help, *e.g.* stepping over a line.

References

Lewis SJ, Barker RA. A pathophysiological model of freezing of gait in Parkinson's disease. *Parkinsonism Relat Disord.* 2009; **15**: 333–8.

Nonnekes J, Snijders AH, Nutt JG, Deuschl G, Giladi N, Bloem BR. Freezing of gait: a practical approach to management. *Lancet Neurol*. 2015; **14**: 768–78.

Okuma Y. Practical approach to freezing of gait in Parkinson's disease. *Pract Neurol*. 2014; **14**: 222–30.

Cross Reference

Parkinsonism

Fregoli Syndrome

- see DELUSION

Frey's Syndrome

- see GUSTATORY SWEATING

Froment's Sign

Jules Froment has two eponymous signs:

- Activated rigidity or synkinesis; sometimes known as Froment's manoeuvre.
- 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* or newspaper sign The term is also sometimes used for weakness of little finger adduction (palmar interossei), evident when trying to grip a piece of paper between the ring and little finger.

Reference

Broussolle E, Rethy MP, Thobois S. Jules Froment (1878-1946). *J Neurol.* 2009; **256**: 1581–2.

Cross References

Rigidity; Synkinesia, Synkinesis; Wartenberg's sign (1)

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 behavioural functions. Damage to the frontal lobes may produce a variety of clinical signs, most frequently changes in behaviour. 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 in London, 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, the applause sign).

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 behaviour (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.
- *Medial prefrontal syndrome* ("apathetic, akinetic"):
  - little spontaneous movement, bradykinesia, hypokinesia.
  - sparse verbal output (akinetic mutism).
  - urinary incontinence.
  - sensorimotor signs in lower limbs.
  - indifference to pain.
- *Frontal convexity or dorsolateral prefrontal syndrome* ("dysexecutive"):
  - apathy; abulia, indifference.
  - motor perseveration.
  - difficulty planning, adapting to changing environmental demands, set-shifting; stimulus boundedness.
  - reduced verbal fluency.
  - deficient motor programming, *e.g.* three step hand sequence, rhythmical tapping (go/no-go test).

Overlap between these regional syndromes may occur.

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 behaviours (echophenomena) and, less frequently, utilization behaviour, features of the environmental dependency syndrome.

Frontal lobe syndromes may occur as a consequence of various pathologies:

- Neurodegenerative diseases: especially behavioural variant frontotemporal lobar degeneration; occasionally in Alzheimer's disease.
- Structural lesion: tumour (intrinsic, extrinsic), normal pressure hydrocephalus.
- Cerebrovascular event.
- Head injury.
- Inflammatory metabolic disease: multiple sclerosis, X-linked adrenoleukodystrophy.

References
Damasio AR, Anderson SW. The frontal lobes. In: Heilman KM, Valenstein E, editors. Clinical neuropsychology. 4th ed. Oxford: Oxford University Press; 2003. p. 404–46.
Rosen HJ, Allison SC, Schauer GF, Gorno-Tempini ML, Weiner MW, Miller BL. Neuroanatomical correlates of behavioural disorders in dementia. *Brain*. 2005; **128**: 2612–25.
Cross References
Abulia; Akinesia; Akinetic mutism; Alternating fist closure test; Apathy; Applause sign; Attention; Disinhibition; Dysexecutive syndrome; Emotionalism, Emotional lability; Fist-edge-palm test; Frontal release signs; Hypermetamorphosis; Hyperorality; Hyperphagia; Hypersexuality; Perseveration; Urinary incontinence; Utilization behaviour; *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 neurodegenerative) 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 categorised as:

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

The corneomandibular and nuchocephalic reflexes may also be categorised as "frontal release" signs. (More details may be found in specific entries.)

Some of these signs 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, and they may be more common in dementia with Lewy bodies than other causes of an extrapyramidal syndrome. All increase with age in normal individuals.

References
Borroni B, Broli M, Constanzi C, et al. Primitive reflex evaluation in the clinical assessment of extrapyramidal syndromes. *Eur J Neurol*. 2006; **13**: 1026–8.
Franssen EH. Neurologic signs in ageing and dementia. In: Burns A, editor. Ageing and dementia: a methodological approach. London: Edward Arnold; 1993. p. 144–74.
Van Boxtel MP, Bosma H, Jolles J, Vreeling FW. Prevalence of primitive reflexes and the relationship with cognitive change in healthy adults: a report from the Maastricht Aging Study. *J Neurol*. 2006; **253**: 935–41.
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 behaviours, 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 "non-organic" 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, excessive effort.
  - 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. *J Neurol*. 1991; **238**: 140–6.

Stone J, Warlow C, Sharpe M. The symptom of functional weakness: a controlled study of 107 patients. *Brain*. 2010; **133**: 1537–51.

Cross References

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

Funnel Vision

- see "TUNNEL VISION"