# P

## Pagophagia
- see PICA

## Palatal Reflex
- see GAG REFLEX

## Palatal Tremor
Palatal tremor, also known as palatal myoclonus, is characterized by rhythmic, unilateral or bilateral, palatal contractions which continue during sleep; this may be classified as a focal myoclonic syndrome. A distinction may be made between essential and symptomatic palatal tremor, also known as primary and secondary isolated palatal tremor.

Palatal tremor may be asymptomatic, or there may be a clicking sound in the inner ear, especially in essential palatal tremor. There may be associated contractions of external ocular muscles (oculopalatal myoclonus), larynx, neck, diaphragm (respiratory myoclonus, diaphragmatic flutter, or Leeuwenhoek's disease), trunk, and limbs, which may bring the palatal tremor to attention. Palatal tremor may be accompanied by pendular nystagmus and oscillopsia. A distinct clinical entity of progressive ataxia and palatal tremor (PAPT) has been described.

Palatal myoclonus is associated with lesions interrupting pathways between the red nucleus, inferior olivary nucleus and dentate nucleus (Guillain-Mollaret triangle). Hypertrophy of the inferior olivary nucleus may be evident neuroradiologically (structural or functional imaging) and pathologically. This is a consequence of a lesion in the dentato-olivary pathway which leads to transsynaptic degeneration and hypermetabolism of the olivary nucleus. In PAPT there may be cerebellar atrophy in addition to bilateral olivary hypertrophy.

Although many cases are essential/idiopathic, recognised symptomatic causes of palatal tremor include vascular lesions, trauma, neoplasia, demyelination, epilepsy and, rarely, adult-onset Alexander's disease.

Drug treatment of palatal tremor is often unsuccessful, although reports of benefit with 5-hydroxytryptophan, carbamazepine, sodium valproate, clonazepam, baclofen, and even sumatriptan have appeared. Botulinum toxin injections may also help.

**References**

Samuel M, Torun N, Tuite PJ, Sharpe JA, Lang AE. Progressive ataxia and palatal tremor (PAPT): clinical and MRI assessment with review of palatal tremors. *Brain*. 2004; **127**: 1252–68.
Zadikoff C, Lang AE, Klein C. The "essentials" of essential palatal tremor: a reappraisal of the noslogy. *Brain*. 2006; **129**: 832–40.

**Cross References**

Eight-and-a-half syndrome; Myoclonus; Nystagmus; Oscillopsia; Tinnitus; Tremor

## Palilalia
Palilalia is a disorder of articulation characterized by the involuntary repetition of syllables within a word, whole words, or phrases, hence a reiterative speech disorder. The term stutter may be used for repetition of single syllables, and the term palilogia has sometimes been used for the repetition of phrases, to distinguish from palilalia. These phenomena may be encountered in:

- Parkinson's disease (along with bradylalia, slowness of speech).
- Progressive supranuclear palsy.



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- Tourette syndrome (along with vocal and motor tics).
- Pick's disease, as part of the so-called PES syndrome (palilalia, echolalia, stereotypy) or the PEMA syndrome (palilalia, echolalia, mutism, amimia).
- Late stages of Alzheimer's disease.
- Postencephalitic parkinsonism (von Economo's disease).
- Fahr's disease (bilateral basal ganglia calcification).
- Thalamic/midbrain infarcts.
- Seizure disorders: ictal or post-ictal phenomenon.
- Normal finding in children below the age of about 6 years

In pathological states, palilalia may reflect difficulty in set shifting, as seen in frontal lobe (frontal convexity) syndromes.

**References**

Landi D, Benvenga A, Quattrocchi CC, et al. Complex epileptic palilalia: a case report. *Seizure*. 2012; **21**: 655–57.

Shin HY, Yoon JH, Lee WY. Palilalia in Parkinson's disease. *J Neurol*. 2009; **256**(Supp l2): S143. (Abstract P403).

Yasuda Y, Akiguchi I, Ino M, Nabatabe H, Kameyama M. Paramedian thalamic and midbrain infarcts associated with palilalia. *J Neurol Neurosurg Psychiatry*. 1990; **53**: 797–9.

**Cross References**

Bradylalia; Echolalia; Frontal lobe syndromes; Hypomimia; Mutism; Parkinsonism; Stereotypy; Stutter; Tic

## Palilogia

- see PALILALIA

## Palinacousis, Palinacusis

Palinacousis or palinacusis is a paroxysmal auditory illusion of perseveration or persistence of an external auditory stimulus after it has stopped, a phenomenon also known as auditory perseveration. Although sometimes classified as an illusory experience, musical hallucinations may occur concurrently. Palinacousis may occur as an aura, seizure, or post-ictal phenomenon, and be associated with structural and/or functional pathology in the medial geniculate body or temporal lobe.

**Reference**

Fields MC, Marcuse LV. Palinacousis. *Handb Clin Neurol*. 2015; **129**: 457–67.

**Cross References**

Aura; Hallucination; Illusion

## Palinopsia

Palinopsia is an illusory visual phenomenon characterized by the persistence or recurrence of visual images immediately after the stimulus has been removed, hence visual perseveration. This is distinct from the physiological after-image. It may be associated with polyopia. The description of the symptom may lead to it being mistaken for diplopia ("pseudodiplopia"). A hallucinatory form of palinopsia has also been described.

Palinopsia occurs most frequently in the context of a left homonymous hemianopia, secondary to right occipitotemporal or occipitoparietal lesions: these may be vascular, neoplastic, metabolic, ictal, or drug- or toxin-induced (*e.g.* carbon monoxide poisoning). Palinopsia has also been described with retinal and optic nerve disease, and occasionally in normal individuals.

**Reference**

Gersztenkorn D, Lee AG. Palinopsia revamped: a systematic review of the literature. *Surv Ophthalmol*. 2015; **60**: 1–35.

**Cross References**

Hemianopia; Illusion; Perseveration; Polyopia; Visual perseveration

## Pallesthesia

Pallesthesia is the appreciation of vibration sensation; its loss may be described as pallanaesthesia. This may be an age-related sign, or a consequence of peripheral neuropathic disorders.

**Cross References**

Age-related signs; Vibration

## Palmaris Brevis Sign

Palmaris brevis sign may be useful in localising the site of an ulnar nerve lesion. Innervated by the superficial "sensory" division of the ulnar nerve in the wrist (distal canal of Guyon), contraction of the palmaris brevis muscle may be evident with compressive lesions of the deep motor branch of the ulnar nerve which cause intrinsic hand muscle weakness but no sensory loss ("Ramsay Hunt syndrome"): the patient is asked to "contract" the hypothenar eminence with the fifth digit forcibly abducted, and the examiner looks for corrugation of the skin over the eminence. In sensory superficial division ulnar nerve lesions, this sign is lost.

**References**

Iyer VG. Palmaris brevis sign in ulnar neuropathy 1998. *Muscle Nerve*. 1998; **21**: 675–77.
Larner AJ. Pitfalls in the diagnosis of ulnar neuropathy: remember the deep palmar branch. *Br J Hosp Med*. 2010; **71**: 654–5.

## Palmomental Reflex

The palmomental reflex consists of contraction of the mentalis muscle induced by stroking the ipsilateral palm with a blunt object. It may indicate damage to the contralateral paracentral cortex or its connections, but since it is observed in about one quarter of normal adults and is very common in the normal elderly, and may occur in other conditions, both its sensitivity and specificity are low. It may be considered a frontal release sign or primitive reflex, but is less specific than the grasp reflex. Induction of the reflex by stimulation of areas other than the palm is more likely to be associated with cerebral damage.

**References**

Brodsky H, Vuong KD, Thomas M, Jankovic J. Glabellar and palmomental reflexes in parkinsonian disorders. *Neurology*. 2004; **63**: 1096–8.
Owen G, Mulley GP. The palmomental reflex: a useful clinical sign? *J Neurol Neurosurg Psychiatry*. 2002; **73**: 113–5.

**Cross References**

Age-related signs; Frontal release signs

## Papilloedema

Papilloedema is swelling (oedema) of the optic nerve head due to raised intracranial pressure (*cf*. other causes of disc swelling, which may cause pseudopapilloedema). A number of stages of papilloedema are described: in the acute stage, the only findings may be oedema at the superior and inferior poles of the disc, absence of spontaneous retinal venous pulsation, and enlargement of the blind spot. As papilloedema progresses the whole disc is involved and splinter haemorrhages may be evident at the disc margin. These early stages may be asymptomatic, or may be associated with transient losses of vision (obscurations), often provoked by activities or movements which further raise intracranial pressure, thus compromising retinal perfusion pressure. Enlargement of the blind spot and constriction of the visual field may be evident, but visual acuity is often unimpaired (*cf*. disc swelling due to papillitis). Chronic papilloedema produces gliosis of the optic nerve head and eventually optic atrophy ("sequential optic atrophy") with nerve fibre damage and permanent visual field defects.

A rating scale for papilloedema (Frisén) has been described, but one study found low agreement between reviewers using this scale.

**Reference**

Sinclair AJ, Burdon MA, Nightingale PG, et al. Rating papilloedema: an evaluation of the Frisén classification in idiopathic intracranial hypertension. *J Neurol*. 2012; **259**: 1406–12.

**Cross References**

Blind spot; Disc swelling; Obscurations; Optic atrophy; Pseudopapilloedema; Retinal venous pulsation; Scotoma

## Paraballismus

- see BALLISM, BALLISMUS; HEMIBALLISMUS

## Paradoxical Abdominal Wall Movement, Paradoxical Breathing

The normal movement of the diaphragm (*i.e.* down in inspiration, causing outward abdominal wall movement) may be reversed (paradoxical) in conditions which cause diaphragm weakness (*i.e.* inward abdominal wall movement on inspiration), *e.g.* Guillain-Barré syndrome, acid-maltase deficiency, phrenic nerve injury, hence paradoxical abdominal movement, abdominal paradox, paradoxical breathing, or paradoxical diaphragm movement. This may be detectable clinically or by X-ray screening of the diaphragm. Vital capacity is lower when lying compared to standing. Paradoxical diaphragm movement is a potentially alarming sign since it may indicate incipient respiratory failure.

**Reference**

Ahmed R, McNamara S, Gandevia S, Halmagyi GM. Paradoxical abdominal wall movement in bilateral diaphragmatic paralysis. *Pract Neurol*. 2012; **12**: 184–6.

**Cross Reference**

Myopathy

## Paradoxical Flexor Reflex

- see GORDON'S SIGN

## Paradoxical Head Tilt

- see BIELSCHOWSKY'S SIGN, BIELSCHOWSKY'S TEST

## Paradoxical Triceps Reflex

- see INVERTED REFLEXES

## Paraesthesia

Paraesthesia (plural: paraesthesiae or paraesthesias) describes an abnormal sensation, often described as a tingling sensation, or likened to "pins and needles" or electricity, pricking, tickling, or even crawling (formication), *i.e.* positive sensory symptoms. The sensation is not pleasant but nor is it painful (*cf*. dysaesthesia). Some patients may describe this sensation as "numbness" or "deadness", in which case care needs to be taken to differentiate it from anaesthesia (*i.e.* a negative phenomenon). Some authorities reserve the term for spontaneous rather than evoked positive sensory phenomena, as a distinction from dysaesthesia.

Paraesthesia is a feature of neuropathy, and may occur in the distribution of a compressed or entrapped nerve, perhaps reflecting the mechanosensitivity of nerves in this situation (*e.g.* Phalen's sign, Tinel's sign), or distally in a "glove and stocking distribution" (acroparaesthesia). Paraesthesia is a more reliable indicator of the diagnosis of neuropathy than pain. Paraesthesia may also be provoked by hyperventilation (especially perioral, hands and feet). Central lesions may also produce paraesthesia (*e.g.* Lhermitte's sign).

**Cross References**

Anaesthesia; Dysaesthesia; Lhermitte's sign; Phalen's sign; Tinel's sign

**Paragrammatism**
Paragrammatism is the substitution of morphological elements and function words in the context of fluent speech (*e.g.* in Wernicke's aphasia), as differentiated from agrammatism, the omission of function words and bound morphemes in nonfluent speech (*e.g.* in Broca's aphasia).
**Cross References**
Agrammatism; Aphasia; Broca's aphasia; Wernicke's aphasia

**Paragraphia**
- see AGRAPHIA

**Parakinesia, Parakinesis**
These terms have been used in different ways by different authors, to describe:

- a volitional purposeful act designed to camouflage, mask, or draw attention away from an involuntary movement, such as chorea;
- strange movements of presumed psychogenic origin. In this context, it should be remembered that many movements previously thought to conform to this definition have subsequently been recognised to have an organic basis (*e.g.* tics, klazomania).

The terms are now seldom used, other than to describe the involuntary movement of a paralysed arm following yawning, parakinesia brachialis oscitans.
**Reference**
Zorzetto FP, Braatz VL, Walusinski O, Teive HA. Parakinesia brachialis oscitans during thrombolytic therapy. *BMJ Case Rep*. 2013; **2013**: pii: bcr2012007079.
**Cross References**
Chorea, Choreoathetosis; Dyskinesia; Klazomania; Yawning

**Paralexia**
- see ALEXIA

**Paralogia**
- see GANSER PHENOMENON, GANSER SYNDROME

**Paralysis**
Paralysis is a total loss of power to move a body part; equivalent to the suffix -plegia. The use of the word has not been entirely consistent over time, for example James Parkinson (1755–1824) originally used the term *paralysis agitans* to describe the disease which now bears his name. The periodic paralyses are a group of conditions characterized by episodic muscular weakness and stiffness (myotonia) associated with mutations in the skeletal muscle voltage-gated sodium and calcium ion channel genes (channelopathies).
**Cross References**
Myotonia; Plegia

**Paramnesia**
Paramnesia is recalling as memories things which have not in fact taken place, hence a distortion of episodic or autobiographical memory. This may be neurological or psychiatric in origin. Relation of paramnesias as the truth occurs in confabulation.
**Cross References**
Amnesia; Confabulation; Reduplicative paramnesia

## Paramyotonia

Paramyotonia describes myotonia induced by cold and exercise. It is similar to myotonia in that muscle does not relax normally following contraction (voluntary, percussion), which may prompt a complaint of muscle aching or stiffness, but differs in that repetitive muscle use (*e.g.* exercise) accentuates the problem, leading to an increased delay in muscle relaxation (worsening stiffness). For example, repeated forced voluntary eyelid closure in a patient with paramyotonia may, after several attempts, lead to a failure of voluntary eyelid opening, the eyes remaining closed for a minute or so. Paramyotonia particularly affects the face and forearms. This type of muscle stiffness may also be sensitive to temperature, being made worse by cooling which may also provoke muscle weakness. Weakness may outlast exposure to cold by several hours.

Neurophysiological studies may assist in the diagnosis of paramyotonia. During the delayed muscle relaxation, electrical activity is not prominent, and after muscle cooling the resting muscle membrane potential may be reduced from around the normal value of −80 mV to around −40 mV, at which point muscle fibres are inexcitable (contracture).

Paramyotonia congenita (Eulenburg's disease) is a channelopathy with mutations affecting the α-subunit of the sodium channel (SCN4A). Mutations in the same gene have been documented in hyperkalaemic periodic paralysis and $K^+$-aggravated myotonia.

Symptomatic treatment with membrane-stabilizing agents like mexiletine and tocainide, or with the carbonic anhydrase inhibitor acetazolamide, might be tried. Precautions are necessary during general anaesthesia because of the risk of diaphragm myotonia.

**References**

Ebers GC, George AL, Barchi RL, et al. Paramyotonia congenita and hyperkalaemic periodic paralysis are linked to the adult muscle sodium channel gene. *Ann Neurol*. 1991; **30**: 810–6.

Matthews E, Tan SV, Fialho D, et al. What causes paramytonia in the United Kingdom? Common and new SCN4A mutations revealed. *Neurology*. 2008; **70**: 50–3.

**Cross References**

Contracture; Myotonia; Paralysis; Warm-up phenomenon

## Paraparesis

Paraparesis describes weakness of the lower limbs, short of complete weakness or paralysis (paraplegia). This may result from lesions anywhere from cerebral cortex (frontal, parasagittal lesions) to peripheral nerves, producing either an upper motor neurone (spastic) or lower motor neurone (flaccid) picture. A spinal cord lesion (myelopathy) is probably the most common cause. Paraparesis may be symmetrical or asymmetrical. Recognised causes of paraparesis include:

- *Upper motor neurone lesions*:

  Traumatic section of the cord.
  Cord compression from intrinsic or extrinsic mass lesion, *e.g.* tumour, metastasis, abscess, empyema, haematoma (epidural, subdural).
  Inflammatory lesions: acute transverse myelitis of viral origin, multiple sclerosis, neuromyelitis optica (Devic's syndrome), systemic lupus erythematosus, Behçet's disease, giant cell arteritis (rare).
  Structural lesions: tethered cord syndrome, arteriovenous malformation.
  Metabolic: Hereditary spastic paraplegia (HSP), adrenoleukodystrophy (X-ALD), subacute combined degeneration of the cord (usually mild).
- *Lower motor neurone lesions*:

  Acute or chronic neuropathies (Guillain-Barré syndrome, chronic inflammatory demyelinating polyradiculoneuropathy).

**Reference**
Jacob A, Larner AJ. Diseases of the spinal cord. In: Cox TM, et al., editors. Oxford textbook of medicine. 6th ed. Oxford: Oxford University Press; 2016. in press.
**Cross References**
Flaccidity; Myelopathy; Paraplegia; Spasticity

## Paraphasia

Paraphasias are a feature of aphasias (disorders of language), particularly (but not exclusively) fluent aphasias resulting from posterior dominant temporal lobe lesions (*cf.* anterior lesions which tend to produce non-fluent aphasias with agrammatism).

Paraphasias refer to a range of speech output errors, both phonological and lexical, including substitution, addition, duplication, omission and transposition of linguistic units, affecting letters within words, letters within syllables, or words within sentences. Paraphasic errors may be categorised as:

- *Phonemic* or *literal*:

  Errors involve individual phonemes; impaired phonology (*i.e.* sound based) causing approximations to real words; nonwords resulting from phonemic paraphasia may be referred to as neologisms. Phonemic paraphasias may be encountered in Broca's aphasia and conduction aphasia, when the patient may recognise them to be errors, and Wernicke's aphasia.
- *Formal*:

  Target word is replaced by another word that is phonemically similar.
- *Morphemic*:

  Errors involving word stems, suffixes, prefixes, inflections and other parts of words.
- *Verbal*:

  Errors involving whole words. These may be further classified as:
  *Semantic* or *categoric*: substitution of a different exemplar from the same category (*e.g.* "orange" for "apple"; *paradigmatic*) or of a thematically related word (*e.g.* "sit" for "chair"; *syntagmatic*). Verbal paraphasias showing both semantic and phonemic resemblance to the target word are called *mixed* errors. These types may be observed in patients with Wernicke's aphasia, who often seem unaware of their paraphasias due to a failure of self-monitoring of output.

**Cross References**
Aphasia; Broca's aphasia; Conduction aphasia; Jargon aphasia; Neologism; Schizophasia; Transcortical aphasias; Wernicke's aphasia

## Paraplegia

Paraplegia is a total weakness (paralysis) of the lower limbs (*cf.* paraparesis). This may result from lower motor neurone lesions involving multiple nerve roots and/or peripheral nerves (*e.g.* paraparetic Guillain-Barré syndrome) producing a flaccid, areflexic paraplegia; but more commonly it is due to upper motor neurone lesions interrupting corticospinal pathways (corticospinal tract, vestibulospinal tract, reticulospinal tracts, and other extrapyramidal pathways), most usually in the spinal cord. The latter may acutely produce a flaccid areflexic picture ("spinal shock"), but later this develops into an upper motor neurone syndrome (hypertonia, clonus, hyperreflexia, loss of superficial reflexes [*e.g.* abdominal, cremasteric reflexes] and Babinski's sign) with possible lower motor neurone signs at the level of the lesion; bladder involvement is common (urinary retention). Because of the difficulty in distinguishing whether an acute paraplegia is of LMN or UMN origin, imaging to exclude

potentially reversible cord compression is mandatory. Recognised causes of paraplegia of upper motor neurone origin include:

- traumatic section of the cord.
- cord compression.
- inflammatory lesions: acute transverse myelitis of viral origin, multiple sclerosis, neuromyelitis optica (Devic's syndrome).
- ischaemic lesions; anterior spinal artery syndrome, venous infarction of the cord.

In paraplegia of upper motor neurone origin, enhanced flexion defence reflexes ("flexor spasms") may occur, producing hip and knee flexion, ankle and toe dorsiflexion. Eventually such flexor responses may become a fixed flexion deformity with secondary contractures ("paraplegia in flexion"). Prevention of this situation may be possible by avoiding spasms, which are often provoked by skin irritation or ulceration, bowel constipation, bladder infection, and poor nutrition. Physiotherapy and pharmacotherapy with agents such as baclofen, dantrolene and tizanidine may be used; botulinum toxin injections may be helpful for focal spasticity. "Paraplegia in extension", with extension at the hip and knee, may be seen with incomplete or high spinal cord lesions.

**Cross References**

Abdominal reflexes; Areflexia; Babinski's sign (1); Clonus; Contracture; Cremasteric reflex; Flaccidity; Hyperreflexia; Hypertonia, Hypertonus; Lower motor neurone (LMN) syndrome; Myelopathy; Paraparesis; Spasticity; Upper motor neurone (UMN) syndrome; Urinary retention

## Parapraxia, Parapraxis

Although these terms may be used in common parlance as synonymous with a "Freudian slip", slips which Freud himself referred to as *Fehlleistungen* ("faulty actions" or "misperformances"), in neurological practice they have a different meaning, referring to one of the cardinal symptoms of ideomotor apraxia: a combination of deficient action selection with errors of sequencing of actions and spatial orientation errors. Parapraxic errors include:

- Perseveration: repetition of movements.
- Substitution: of one movement for another (*e.g.* patient shows tongue when asked to close eyes).
- Surplus movements.
- Verbal overflow: explaining a movement rather than performing it.
- Omission: incomplete movements.
- *Conduit d'approche*: several attempts to perform the correct movement.
- Body part as object.

**Reference**

Klein R, Mayer-Gross W. The clinical examination of patients with organic cerebral disease. London: Cassell; 1957. p. 39.

**Cross References**

Apraxia; Body part as object; *Conduit d'approche*; Perseveration

## Paratonia

Paratonia (or paratonic rigidity, or *gegenhalten*, or oppositional rigidity) is a variable resistance to passive movement of a limb when changing its posture or position, which is evident in both flexor and extensor muscles (as in rigidity, but not spasticity), which seems to increase further with attempts to encourage the patient to relax, such that there is a resistance to any applied movement (German: to counter, stand ones ground). However, this is not a form of impaired muscle relaxation akin to myotonia and paramyotonia. For instance, when lifting the legs by placing the hands under the knees, the legs may be held extended at the knees

despite encouragement on the part of the examiner for the patient to flex the knees. The degree of resistance is said to depend on the speed of movement (as in spasticity). Generally, tendon reflexes are normal, plantar responses downgoing, and there is no clonus. A Paratonia Assessment Instrument has been described.

Paratonia is a sign of bilateral frontal lobe dysfunction, especially mesial cortex and superior convexity (premotor cortex, area 6). It may be related to executive dysfunction and planning impairments. It is not uncommon in otherwise healthy elderly individuals with diffuse frontal lobe cerebrovascular disease, and may also be seen in neurodegenerative disorders such as Alzheimer's disease.

**Reference**

Hobbelen JS, Koopmans RT, Verhey FR, Habraken KM, de Bie RA. Diagnosing paratonia in the demented elderly: reliability and validity of the Paratonia Assessment Instrument (PAI). *Int Psychogeriatr*. 2008; **20**: 840–52.

**Cross References**

Frontal release signs; Myotonia; Paramyotonia; Rigidity; Spasticity

## Paresis

Paresis denotes a degree of weakness which is less than total paralysis (−plegia), which may be of upper or lower motor neurone origin. Various prefixes denote the location of such weakness, *e.g.* hemiparesis, monoparesis, ophthalmoparesis, paraparesis, quadriparesis.

Since localised pain may inhibit voluntary muscular exertion, apparent weakness in such circumstances may be labelled "algesic pseudoparesis".

**Cross References**

Lower motor neurone (LMN) syndrome; Paralysis; Plegia; Upper motor neurone (UMN) syndrome; Weakness

## Parinaud's Syndrome

Parinaud's syndrome, also sometimes known as the dorsal midbrain syndrome, the periaqueductal grey matter syndrome, or the pretectal syndrome, consists of the following features:

- Eye movements:

  Paralysis of vertical gaze, especially upgaze: Bell's phenomenon may be spared.
  Loss of convergence; convergence spasm may cause slow abduction ("midbrain pseudo-sixth").
  Skew deviation.

  Convergence-retraction nystagmus (Körber-Salus-Elschnig syndrome); sometimes downbeat nystagmus.
- Eyelids:

  Lid retraction (Collier's "tucked lid" sign) or ptosis (ventral extension of lesion).
- Pupils:

  Mydriasis.

This constellation of signs results from dorsal midbrain lesions, such as pineal tumours, which affect the pretectum and posterior commissure and so interfere with conjugate eye movements in the vertical plane. The key anatomical substrates, damage to which causes the syndrome, are probably the interstitial nucleus of Cajal and the nucleus of the posterior commissure and their projections.

**References**

Keane JR. The pretectal syndrome: 206 patients. *Neurology*. 1990; **40**: 684–90.
Ouvrier R. Henri Parinaud (1844–1905). *J Neurol*. 2011; **258**: 1571–2.
Parinaud H. Paralysie des mouvements associés des yeux. *Archives de Neurologie Paris*. 1883; **5**: 145–72.

Pierrot-Deseilligny C, Chain F, Gray M, et al. Parinaud's syndrome. *Brain*. 1982; **105**: 667–96.

**Cross References**

Collier's sign; Light-near pupillary dissociation; Nystagmus; Supranuclear gaze palsy

## Parkinsonism

Parkinsonism describes a clinical syndrome characterized by the presence of some or all of the following features; there is overlap with so-called akinetic-rigid syndromes in which these features predominate:

- Akinesia, hypokinesia (*sine qua non*).
- Rigidity: consistent (leadpipe) or jerky (cogwheeling; Negro's sign).
- Bradykinesia.
- Tremor, usually at rest, of frequency 3.5–7.0 Hz, "pill rolling" type; there may sometimes be an additional action component to the tremor, and very occasionally there is exclusively an action tremor. "Re-emergent tremor" is also described.
- Stooped posture: forward flexion of trunk, flexion of knees, elbows; "simian posture".
- Impaired postural reflexes, with or without a history of falls; propulsion, retropulsion.
- Mask-like facies, poverty of spontaneous facial expression (hypomimia).
- Reduced blink rate (this may be a particular feature of progressive supranuclear palsy).
- Hypophonic, monotonic voice (hypokinetic dysarthria).
- Widened palpebral fissure (Stellwag's sign).
- Hypometria.
- Seborrhea.
- Sialorrhoea.
- Festinant (shuffling) gait.
- Micrographia.
- Dystonic postures, *e.g.* striatal toe.
- Apraxia.
- Akathisia.
- Cognitive impairment (usually of frontal-subcortical type).
- Hallucinations: minor (*anwesenheit*; passage type), or formed, visual > auditory. Insight into the non-reality of these experiences may be retained, hence may be described as "pseudohallucinations" rather than hallucinations.
- Autonomic dysfunction, especially orthostatic hypotension.

Conventionally parkinsonism is viewed as a disorder of the extrapyramidal system producing "extrapyramidal signs", although this term has limitations: despite the fact that some of the cardinal features of parkinsonism (bradykinesia, rigidity, postural instability, tremor) result from pathology in the basal ganglia, particularly affecting dopaminergic pathways, other features may reflect cortical involvement, at least in part (*e.g.* apraxia, micrographia).

The incidence of parkinsonism increases dramatically with age; it is also associated with an increased risk of death, particularly in the presence of a gait disturbance.

The differential diagnosis of parkinsonism is broad, and includes:

- Idiopathic Parkinson's disease.
- Dementia with Lewy bodies.
- Multiple system atrophy.
- Progressive supranuclear palsy (Steele-Richardson-Olszewski syndrome).
- Corticobasal degeneration, cortical basal ganglionic degeneration.
- Drug-induced parkinsonism (*e.g.* neuroleptics, MPTP).
- Toxin-induced parkinsonism (*e.g.* carbon monoxide, manganese).
- Wilson's disease (hepatolenticular degeneration); non-Wilsonian hepatocerebral degeneration.

- Neuroleptic malignant syndrome.
- Normal pressure hydrocephalus.
- "Arteriosclerotic parkinsonism", resulting from multiple subcortical infarcts.
- Huntington's disease, especially juvenile onset type (Westphal variant).
- Post-encephalitic parkinsonism (encephalitis lethargica, von Economo's disease).
- Dementia pugilistica, post-traumatic parkinsonism.
- Systemic lupus erythematosus.
- Sjögren's syndrome.
- Hypoparathyroidism.
- Parkinsonism-dementia complex of Guam.

Obsessive slowness also enters the differential diagnosis but typical parkinsonian features (akinesia, rigidity) are not present in this condition.

It is crucial not to miss the diagnosis of Wilson's disease, although rare, since in the early stages this disorder is reversible with copper chelation therapy; hence copper and caeruloplasmin should be checked in all patients with young-onset (under age 40 or 50 years) parkinsonism (and dystonia).

Response to levodopa therapy is only reliably seen in idiopathic Parkinson's disease, although some patients with multiple system atrophy or progressive supranuclear palsy may benefit. The features particularly responsive in Parkinson's disease are bradykinesia and rigidity; tremor is less reliably helped.

**References**

Bennett DA, Beckett LA, Murray AM, et al. Prevalence of parkinsonian signs and associated mortality in a community population of older people. *New Engl J Med*. 1996; **334**: 71–6.

Gardner-Thorpe C. James Parkinson 1755–1824. Exeter: A Wheaton & Co. Ltd; 1987. [Includes facsimile of Parkinson's book on the shaking palsy].

Gibb WRG, Lees AJ. The relevance of the Lewy body to the pathogenesis of idiopathic Parkinson's disease. *J Neurol Neurosurg Psychiatry*. 1988; **51**: 745–52.

**Cross References**

Apraxia; Blinking; Bradykinesia; Dysarthria; Dystonia; Hypokinesia; Hypomimia; Hypophonia; Mask-like facies; Micrographia; Orthostatic hypotension; Postural reflexes; Rigidity; Seborrhoea; Sialorrhoea; Striatal toe; Supranuclear gaze palsy; Tremor

## Parosmia

Parosmia is a false smell, *i.e.* the subjective sensation of a smell which does not exist (*i.e.* an hallucination). Such smells are usually unpleasant (cacosmia), may be associated with a disagreeable taste (cacogeusia), and may be difficult for the patient to define. Causes include purulent nasal infections or sinusitis, and partial recovery following transection of olfactory nerve fibres after head injury. Transient parosmia may presage epileptic seizures of temporal lobe cortical origin (olfactory aura), particularly involving the medial (uncal) region. The symptom may also be common amongst the normal population.

**Reference**

Nordin S, Brämerson A, Millqvist E, Bende M. Prevlance of parosmia: the Skövde population-based studies. *Rhinology*. 2007; **45**: 50–3.

**Cross References**

Aura; Cacogeusia; Seizures

## Parry-Romberg Syndrome

Parry-Romberg syndrome describes hemifacial atrophy, a thinning of subcutaneous tissues on one side of the face which may also involve muscle and bone (causing enophthalmos), and sometimes brain, in which case neurological features (hemiparesis, hemianopia, focal epileptic seizures, cognitive impairment) may also be present.

The clinical heterogeneity of hemifacial atrophy probably reflects pathogenetic heterogeneity. The syndrome may result from maldevelopment of autonomic innervation or vascular

supply, or as an acquired feature following trauma, or a consequence of linear scleroderma (morphoea), in which case a *coup de sabre* may be seen.

**References**

Larner AJ, Bennison DP. Some observations on the aetiology of hemifacial atrophy ("Parry-Romberg syndrome"). *J Neurol Neurosurg Psychiatry*. 1993; **56**: 1035–6.

Stone J. Parry-Romberg syndrome: a global survey of 205 patients using the Internet. *Neurology*. 2003; **61**: 674–6.

**Cross References**

*Coup de sabre*; Enophthalmos; Hemianopia; Hemiparesis

## Past-pointing

- see DYSMETRIA

## Patellar Reflex

- see REFLEXES

## Pathological Crying, Pathological Laughter

Pathological laughter and pathological crying (PLC), or forced laughter and crying, also referred to as involuntary emotional expression disorder, have been defined as reflecting an incongruence of mood (subjective feeling) and expression or affect ("objective", observed), such that patients laugh involuntarily though not happy, or cry though not sad. There may be a sense that the patient is struggling against these displays of emotion, in contrast to the situation in other forms of emotional lability where there is said to be congruence of mood and affect, although sudden fluctuations and exaggerated emotional expression are common to both, suggesting a degree of overlap.

PLC is ascribed to a loss (release) of the normal inhibition of the motor component of facial expression (*i.e.* cortical-subcortical disinhibition). PLC may occur in the context of a pseudobulbar palsy ("pseudobulbar affect") but not invariably so. PLC has been reported in:

- Multiple sclerosis: crying > laughing; related to cognitive impairment (more extensive brain involvement, but not brainstem).
- Alzheimer's disease.
- Stroke: PLC may be the harbinger of brainstem stroke or a feature of anterior choroidal artery territory infarctions; rarely a feature of TIAs.
- Motor neurone disease.
- Head injury.
- Gelastic epilepsy.
- Traumatic brain injury.

A Pathological Laughter and Crying Scale has been developed. Suggested treatments for PLC include amitriptyline, levodopa, amantadine, and serotonin reuptake inhibitors such as fluoxetine and citalopram.

**References**

Larner AJ. Crying spells as symptoms of a transient ischaemic attack. *J Neurol Neurosurg Psychiatry*. 2000; **68**: 800–1.

Robinson RG, Parikh RM, Lipsey JR, Starkstein SE, Price TR. Pathological laughter and crying following stroke: validation of a measurement scale and a double-blind treatment study. *Am J Psychiatry*. 1993; **150**: 286–93.

Wild B, Rodden FA, Grodd W, Ruch W. Neural correlates of laughter and humour. *Brain*. 2003; **126**: 2121–38.

**Cross References**

Automatism; Emotionalism, Emotional lability; *Fou rire prodromique*; Pseudobulbar palsy

## Peduncular Hallucinosis

Peduncular hallucinosis is a rare syndrome characterised by hallucinations and brainstem symptoms. Hallucinations are vivid and naturalistic. Brainstem findings include oculomotor disturbances, dysarthria, ataxia, and impaired arousal. Episodic memory impairments also occur. Pathology may be in the midbrain, thalamus and pons.

**Reference**

Benke T. Peduncular hallucinosis. A syndrome of impaired reality monitoring. *J Neurol*. 2006; **253**: 1561–71.

**Cross Reference**

Hallucination

## "Peek Sign"

One of the eye signs of myasthenia gravis: on attempted forced eye closure, orbicularis oculi may fatigue such that the patient "peeks" through the partially open palpebral fissure.

## Peliopsia

Peliopsia or pelopsia is a form of metamorphopsia characterised by the misperception of objects as being closer to the observer than they really are (*cf*. porropsia, teliopsia).

**Cross References**

Metamorphopsia; Porropsia

## Pelvic Thrusting

Pelvic thrusting may be a feature of epileptic seizures of frontal lobe origin; occasionally it may occur in temporal lobe seizures. Pelvic thrusting also occurs in pseudoseizures, particularly those of the "thrashing" variety.

Choreiform disorders may involve the pelvic region causing thrusting or rocking movements.

**Reference**

Geyer JD, Payne TA, Drury I. The value of pelvic thrusting in the diagnosis of seizures and pseudoseizures. *Neurology*. 2000; **54**: 227–9.

**Cross References**

Automatism; Chorea, Choreoathetosis; Seizure

## Pendular Nystagmus

Pendular or undulatory nystagmus is charactrerised by eye movements which are more or less equal in amplitude and velocity (sinusoidal oscillations) about a central (null) point. In acquired causes such as multiple sclerosis, this may produce oscillopsia and blurred vision. Treatment options include gabapentin and memantine.

**Reference**

Starck M, Albrecht H, Pöllmann W, Dieterich M, Straube A. Acquired pendular nystagmus in multiple sclerosis: an examiner-blind cross-over treatment study of memantine and gabapentin. *J Neurol*. 2010; **257**: 322–7.

**Cross References**

Nystagmus; Oscillopsia

## Percussion Myotonia

Percussion myotonia is the myotonic response of a muscle to a mechanical stimulus, *e.g.* when struck with a tendon hammer. For example, a blow to the thenar eminence may produce involuntary and sustained flexion of the thumb. This response, which may be seen in myotonic dystrophy, reflects the impaired muscle relaxation which characterises myotonia.

**Reference**

Barroso FA, Nogues MA. Images in clinical medicine. Percussion myotonia. *New Eng J Med*. 2009; **360**: e13.

**Cross Reference**
Myotonia

## Periodic Alternating Nystagmus

Periodic alternating nystagmus is a horizontal jerk nystagmus, which damps or stops for a few seconds and then reverses direction. Eye movements may need to be observed for up to 5 min to see the whole cycle. Periodic alternating nystagmus may be congenital (idiopathic infantile, associated with a number of genetic loci) or acquired, if the latter then its localising value is similar to that of downbeat nystagmus (with which it may coexist), especially for lesions at the cervico-medullary junction (*e.g.* Chiari malformation). Treatment of the associated lesion may be undertaken, otherwise periodic alternating nystagmus usually responds to baclofen, hence the importance of correctly identifying this particular form of nystagmus.

**References**

Halmagyi GM, Rudge P, Gresty MA, et al. Treatment of periodic alternating nystagmus. *Ann Neurol*. 1980; **8**: 609–11.

Thomas MG, Crosier M, Lindsay S, et al. The clinical and molecular genetic features of idiopathic infantile periodic alternating nystagmus. *Brain*. 2011; **134**: 892–902.

**Cross Reference**
Nystagmus

## Periodic Respiration

Periodic respiration is a cyclical waxing and waning of the depth and rate of breathing (Cheyne-Stokes breathing or respiration), over about 2 min, the crescendo-decrescendo sequence being separated by central apnoeas. A so-called variant Cheyne-Stokes pattern has hypopnoeas rather than apnoeas.

Periodic respiration may be observed in unconscious patients with lesions of the deep cerebral hemispheres, diencephalon, or upper pons, or with central or tonsillar brain herniation; it has also been reported in multiple system atrophy. Prolonged circulatory time (congestive heart failure) and hypoxaemia (*e.g.* at altitude) may also cause periodic respiration, but with a shorter cycle.

**Reference**

Pearce JMS. Cheyne-Stokes respiration. In: Fragments of neurological history. London: Imperial College Press; 2003. p. 355–8.

**Cross References**
Coma

## Periphrasis

- see CIRCUMLOCUTION

## Perseveration

Perseveration refers to any continuation or recurrence of activity without appropriate stimulus (*cf*. intrusions). Perseverations may be repeated motor behaviours (*e.g.* drawing, writing, applause sign) or speech. These are viewed as a failure to inhibit a previous response pattern. Sensory perseveration is also described, *e.g.* palinopsia in the visual system. A number of varieties of perseveration have been described, associated with lesions in different areas of the brain:

- "*Stuck-in-set*":

  Inappropriate maintenance of a current category or framework; thought to reflect a deficit in executive function; associated with frontal lobe (especially frontal convexity) damage, which is associated with an inert, apathetic pattern of behaviour, rather than the disinhibited pattern associated with orbitofrontal damage.

- "*Recurrent*":

  Unintentional repetition of a previous response to a subsequent stimulus; thought to represent an abnormal post-facilitation of a memory trace; associated with posterior left (dominant) hemisphere damage; commonly seen in aphasics, Alzheimer's disease; this overlaps with "intrusions".

- "*Continuous*":

  Inappropriate prolongation or repetition of a current behaviour without interruption; thought to represent a deficit of motor output; associated with basal ganglia damage.

**References**

Hudson AJ. Perseveration. *Brain*. 1968; **91**: 571–82.
Sandson J, Albert ML. Varieties of perseveration. *Neuropsychologia*. 1984; **22**: 715–32.

**Cross References**

Aphasia; Applause sign; Dysexecutive syndrome; Frontal lobe syndromes; Intrusion; Logoclonia; Palinopsia

## Personification of Paralysed Limbs

Critchley drew attention to the tendency observed in some hemiplegic patients to give their paralysed limbs a name or nick-name and to invest them with a personality or identity of their own. This sometimes follows a period of anosognosia and may coexist with a degree of anosodiaphoria; it is much more commonly seen with left hemiplegia. A similar phenomenon may occur with amputated limbs, and it has been reported in the context of functional limb weakness.

**References**

Critchley M. Personification of paralysed limbs in hemiplegics. *BMJ*. 1955; **ii**: 284–6.
Critchley M. The divine banquet of the brain and other essays. New York: Raven Press; 1979. p. 104–5, 116–7.
Larner AJ. Critchley revisited: personification of a neurologically dysfunctional limb. *Adv Clin Neurosci Rehabil*. 2010; **10**(2): 28.

**Cross References**

Anosodiaphoria; Anosognosia

## Pes Cavus

Pes cavus is a high-arched foot due to equinus (plantar flexion) deformity of the first ray with secondary changes in the other rays (*i.e.* deformity is more evident on the medial side of the foot in most cases). Hammer toes may also be present.

This may be due to imbalance of muscular forces during development, which may be a consequence of neurological disease. The precise pattern may differ with cause, involving the muscles of either the lower leg (*e.g.* strong peroneus longus, weak peroneus brevis and tibialis anterior) or the foot (selective denervation of lumbricals in Charcot-Marie-Tooth disease type 1A).

Pes cavus may be associated with disease of genetic origin, *e.g.* hereditary motor and sensory neuropathy (HMSN, Charcot-Marie-Tooth syndrome), hereditary spastic paraparesis, Friedreich's ataxia, Marfan's syndrome; or be due to an early neurological insult, *e.g.* cerebral palsy, paralytic poliomyelitis. Familial pes cavus without other neurological signs has also been reported (a *forme fruste* of HMSN?).

Surgical treatment of pes cavus may be necessary, especially if there are secondary deformities causing pain, skin breakdown, or gait problems.

**Reference**

Berciano J, Gallardo E, Garcia A, Pelayo-Nero AL, Infante J, Combarros O. New insights into the pathophysiology of pes cavus in Charcot-Marie-Tooth disease type 1A duplication. *J Neurol*. 2011; **258**: 1594–602.

**Cross References**
Claw foot; Hammer toes

## "Petite Madeleines Phenomenon"
- see PROUST PHENOMENON

## Phalen's Sign
Phalen's sign is present when tingling (paraesthesia) is experienced in the distribution of the median nerve when the wrist is held in forced flexion (90° for 30–60 s; Phalen's maneouvre). Patients may volunteer that they experience such symptoms when carrying heavy items such as shopping bags which puts the hand in a similar posture. Hyperextension of the wrist ("reverse Phalen's manoeuvre") may also reproduce symptoms.

These are signs of compression of the median nerve at the wrist (carpal tunnel syndrome). Like other provocative tests (*e.g.* Tinel's sign), the sensitivity and specificity of Phalen's sign for this diagnosis are variable (10–91%, and 33–86%).

The pathophysiology of Phalen's sign is probably the lower threshold of injured nerves to mechanical stimuli, as for Tinel's sign and Lhermitte's sign.

**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**
Durkan's compression test; "Flick sign"; Lhermitte's sign; Paraesthesia; Tinel's sign

## "Phantom Alloaesthesia"
- see ALLOAESTHESIA

## "Phantom Boarder Sign"
- see "MIRROR SIGN"; MISIDENTIFICATION SYNDROMES

## Phantom Chromatopsia
This term has been coined to refer to the complaint of patients who are blind or nearly so that a colour, usually golden or purple, enlarges to invade the entire visual field. This is presumably cortical in origin, and has been described as an hallucination. "Phantom vision" may describe a similar phenomenon.

**Reference**
Zeki S. A vision of the brain. Oxford: Blackwell Science; 1993. p. 278, 279.
**Cross References**
Erythropsia; "Monochromatopsia"; Phantom vision

## Phantom Limb
Phantom limbs, or ghost limbs, describe the subjective report of the awareness of a non-existing or deafferented body part in a mentally otherwise competent individual. The term was coined by Weir Mitchell in the nineteenth century, but parts other than limbs (either congenitally absent or following amputation) may be affected by phantom phenomena, such as lips, tongue, nose, eye, penis, breast and nipple, teeth, and viscera. Phantom phenomena are perceived as real by the patient, may be subject to a wide range of sensations (pressure, temperature, tickle, pain), and are perceived as an integral part of the self. Such "limbless perception" is thought to reflect the mental representation of body parts generated within the brain (body schema), such that perception is carried out without somatic peripheral input. Reorganisation of cortical connections following amputation may explain phantom phenomena such as representation of a hand on the chest or face, for which there is also evidence from functional brain imaging.

**References**
Halligan PW. Phantom limbs: the body in mind. *Cogn Neuropsychiatry*. 2002; **7**: 251–68.
Melzack R. Phantom limbs. *Sci Am*. 1992; **266**: 120–6.
Ramachandran VS, Hirstein W. The perception of phantom limbs. *Brain*. 1998; **121**: 1603–30.
**Cross Reference**
Asomatognosia

## Phantom Vision
This name has been given to visual hallucinations following eye enucleation, by analogy with somaesthetic sensation experienced in a phantom limb after amputation. Similar phenomena may occur after acute visual loss, and may overlap with phantom chromatopsia. Unformed or simple hallucinations are more common than formed or complex hallucinations.
**References**
Cohn R. Phantom vision. *Arch Neurol*. 1971; **25**: 468–71.
Lepore FE. Spontaneous visual phenomena with visual loss. *Neurology*. 1990; **40**: 444–7.
**Cross References**
Hallucination; Phantom chromatopsia; Phantom limb

## Phantosmia
This term has sometimes been used to describe olfactory hallucinations.

## Pharyngeal Reflex
- see GAG REFLEX

## Phonagnosia
Phonagnosia is an inability to recognise familiar voices in the absence of hearing impairment, hence a form of auditory agnosia. The patient can recognise and understand words and sentences (*cf*. pure word deafness). Phonagnosia is the equivalent in the auditory domain of prosopagnosia in the visual domain. The neuroanatomical substrate is thought to be right parietal lobe pathology.
**Reference**
Biederman I, Herald S, Xu X, Amir O, Shilowich B. Phonagnosia, a voice homologue to prosopagnosia. *J Vision*. 2015; **15**(12): 1206.
**Cross References**
Agnosia; Prosopagnosia; Pure word deafness

## Phonemic Disintegration
Phonemic disintegration refers to an impaired ability to organise phonemes, the smallest units in which spoken language may be sequentially described, resulting in substitutions, deletions and misorderings of phonemes. Phonemic disintegration is relatively common in aphasic disorders, including Broca's aphasia, conduction aphasia, and transcortical motor aphasia. Isolated phonemic disintegration is rare. The neural substrate may be primary motor cortex of the left inferior precentral gyrus and subjacent white matter, with sparing of Broca's area.
**References**
Larner AJ, Robinson G, Kartsounis LD, et al. Clinical-anatomical correlation in a selective phonemic speech production impairment. *J Neurol Sci*. 2004; **219**: 23–9.
Taubner RW, Raymer AM, Heilman KM. Frontal-opercular aphasia. *Brain Lang*. 1999; **70**: 240–61.
**Cross References**
Aphasia; Aphemia; Broca's aphasia

## Phonetic Disintegration
- see APHEMIA; SPEECH APRAXIA

## Phonophobia

Phonophobia is a dislike, or fear, of sounds, especially loud sounds, often experienced during a migraine headache at the same time as photophobia and osmophobia.

**Cross References**

Hyperacusis; Photophobia

## Phosphene

Phosphenes are percepts in one modality induced by an inappropriate stimulus, *e.g.* when pressure is applied to the eyeball, the mechanical stimulus may induce the perception of light (Newton investigated his own vision in this way, stimulating the back of his eye with a bodkin). The perception of flashes of light when the eyes are moved has been reported in optic neuritis, presumably reflecting the increased mechanosensitivity of the demyelinated optic nerve fibres; this is suggested to be the visual equivalent of Lhermitte's sign. Eye gouging to produce phosphenes by mechanical stimulation of the retina is reported in Leber's congenital amaurosis. Noise-induced visual phosphenes have also been reported, and may be equivalent to auditory-visual synaesthesia.

**References**

Bolognini N, Convento S, Fusaro M, Vallar G. The sound-induced phosphene illusion. *Exp Brain Res.* 2013; **231**: 469–78.

Davis FA, Bergen D, Schauf C, McDonald I, Deutsch W. Movement phosphenes in optic neuritis: a new clinical sign. *Neurology*. 1976; **26**: 1100–4.

Lessell JB, Cohen MM. Phosphenes induced by sound. *Neurology*. 1979; **29**: 1524–6.

**Cross References**

Auditory-visual synaesthesia; Gaze-evoked phenomena; Lhermitte's sign; Photism; Synaesthesia

## Photic Sneeze Reflex

- see SNEEZING

## Photism

Photisms are transient positive visual phenomenon, such as geometrical shapes or brightly coloured spectral phenomena, occurring in the context of epilepsy, migraine, or in blind visual fields (hence overlapping with photopsia). Auditory-visual synaesthesia may also be described as sound-induced photism.

**Cross References**

Auditory-visual synaesthesia; Photopsia

## Photophobia

Photophobia is an abnormal intolerance of light, often experienced with eye pain. It is associated with a wide range of causes, and may result from both peripheral and central mechanisms:

- Anterior segement eye disorders: uveitis, glaucoma, cataract.
- Vitreo-retinal disorders: retinitis pigmentosa.
- Optic neuropathies: optic neuritis.
- Intracranial disease: migraine, meningitis and other causes of meningeal irritation, central photophobia (possibly associated with a thalamic lesion), dazzle.
- Physiological photophobia: sudden exposure to light after light deprivation.

**Cross References**

Dazzle; Meningism; Retinitis pigmentosa

## Photopsia

Photopsias are simple visual hallucinations consisting of flashes of light which often occur with a visual field defect. They suggest dysfunction in the inferomedial occipital lobe, such as migraine or an epileptogenic lesion.

**Cross References**
Aura; Hallucination; Photism

### Physical Duality
Physical duality describes a rare somaesthetic metamorphopsia occurring as a migraine aura in which individuals feel as though they have two bodies.
**Cross Reference**
Metamorphopsia

### Piano-Playing Fingers
- see PSEUDOATHETOSIS

### Pica
Pica, or pagophagia, is a morbid craving for unusual or unsuitable food in association with iron deficiency. It has also been reported in tuberous sclerosis. Sufferers risk infection from contaminated foods.
**References**
Larner AJ. Neurological signs: geophagia (geophagy) and pica (pagophagia). *Adv Clin Neurosci Rehabil.* 2009; **9**(4): 20.
Von Garnier C, Stunitz H, Decker M, Battegay E, Zeller A. Pica and refractory iron deficiency anaemia: a case report. *J Med Case Rep*. 2008; **2**: 324.
**Cross Reference**
Geophagia, Geophagy

### "Picture Sign"
The "picture sign" is present when a patient believes that individuals seen on the television screen are actually present in the external world; indeed they may be reported to emerge from the television set into the room. This may occur as part of the cognitive disturbance of Alzheimer's disease or dementia with Lewy bodies, or as part of a psychotic disorder. Like the "mirror sign", the "picture sign" may be classified as a misidentification phenomenon.
**Cross References**
"Mirror sign"; Misidentification syndromes

### "Picture Within a Picture" Sign
Following a right parieto-occipital infarction, a patient complained of seeing people moving about in the left lower quadrant of the visual field whilst vision was normal in the remainder of the visual field, a phenomenon labelled as the "picture within a picture" sign. This has been categorized as a visual release hallucination.
**Reference**
Benegas MN, Liu GT, Volpe NJ, Galetta SL. "Picture within a picture" visual hallucinations. *Neurology*. 1996; **47**: 1347–8.

### *Pied En Griffe*
- see CLAW FOOT

### "Pie-In-The-Sky" Defect
This name has sometimes been given to the superior homonymous quadrantanopia ending sharply at the vertical midline due to a temporal lobe lesion interrupting Meyer's loop, that part of the optic radiation coursing through the temporal lobe.
**Cross Reference**
Quadrantanopia

### "Pill Rolling"
- see PARKINSONISM; TREMOR

## "Pinch Sign"

The "pinch sign", or "okay sign", is an inability to make a small circle ("form the letter O", divers' okay sign) by approximating the distal phalanges of the thumb and index finger, due to weakness of flexor digitorum profundus in the index finger and flexor pollicis longus in the thumb as a consequence of median nerve lesions in the forearm, *e.g.* anterior interosseous neuropathy, pronator teres syndrome. This results in a pinching posture of thumb and index finger. The "straight thumb sign" may also be present.

**Cross References**

Froment's sign; "Straight thumb sign"

## Pinhole Test

Impairments in visual acuity due to refractory defects (changes in shape of the globe or defects in the transparent media of the eye) may be improved or corrected by looking through a pinhole which restricts vision to the central beam of light.

## Pisa Syndrome

- see PLEUROTHOTONOS

## Plantar Grasp Reflex

- see GRASP REFLEX

## Plantar Response

The plantar response is most commonly elicited by stroking the sole of the foot with a blunt object. The first response of the hallux is the critical observation, which may be facilitated by having ones line of vision directly above the axis of the toe. The normal response after maturation of the corticospinal tracts (*i.e.* after about 3 years of age) is for the big toe to flex. An extensor response of the big toe in an adult (Babinski's sign) is a reliable sign of upper motor neurone pathology. This may or may not be accompanied by fanning (abduction) of the other toes (fan sign, *signe de l'éventail*), but this does not constitute part of the sign and in isolation has no clinical value.

Use of the terminology "negative Babinski's sign" or "negative Babinski response" to mean "flexor plantar response" is incorrect and should not be used. This normal plantar response is a superficial cutaneous reflex, analogous to abdominal and cremasteric reflexes, whereas the pathological response is often accompanied by activity in other flexor muscles. In some individuals the toes do not move at all, in which case the response is labelled as "mute" or absent. Assessment of the response may be confounded by withdrawal of the foot in ticklish individuals. Stroking the lateral border of the dorsum of the foot rather than the sole has been advocated.

The plantar response may be elicited in a variety of other ways which are not in routine clinical use. Of these, perhaps the most frequently used are Chaddock's sign (application of a stimulus in a circular direction around the external malleolus, or the lateral aspect of the foot from heel to little toe) and Oppenheim's sign (application of heavy pressure to the anterior surface of the tibia from patella to ankle). If the plantar response thus elicited is upgoing, this suggests a spread of the "receptive field" of the reflex. Babinski's sign is the earliest to occur in the presence of upper motor neurone pathology.

It is often difficult to form a definite judgment on the plantar response and reproducibility is also questionable. A study of 24 experienced clinicians invited to examine plantar responses "blind" found that the interobserver percentage agreement beyond chance was on average only 16.7% (95% confidence interval [CI] 0.4–33%); intraobserver percentage agreement was a little better (average 59.6%; CI 39.6–79.6%). There remains a persistent belief, particularly amongst trainees, that an experienced neurologist can make the plantar response go which ever way s/he chooses.

Differentiation of the Babinski sign from the striatal toe seen in parkinsonian syndromes may need to be made.

**References**
Maher J, Reilly M, Daly L, Hutchinson M. Plantar power: reproducibility of the plantar response. *BMJ*. 1992; **304**: 482.
Van Gijn J. The Babinski sign: a centenary. Utrecht: Universiteit Utrecht; 1996.
Van Munster CEP, Weinstein HC, Uitdehaag BMJ, van Gijn J. The plantar reflex: additional value of stroking the lateral border of the foot to provoke an upgoing toe sign and the influence of experience. *J Neurol*. 2012; **259**: 2424–8.
**Cross References**
Abdominal reflexes; Babinski's sign (1); Chaddock's sign; Gordon's sign; Oppenheim's sign; Reflexes; Striatal toe, Upper motor neurone (UMN) syndrome

## Platysma Sign

"Platysma sign" has been used to describe weakness of platysma, which may be seen in the context of central hemiparesis or in high cervical cord lesions, since innervation of the muscle comes from both the seventh cranial nerve and from the high cervical cord (C3).
**Reference**
Ogawa Y, Sakakibara R. Platysma sign in high cervical lesion. *J Neurol Neurosurg Psychiatry*. 2005; **76**: 735.

## Plegia

Plegia means stillness, implying a complete weakness (or paralysis in common parlance), as in monoplegia, diplegia, ophthalmoplegia, paraplegia, quadriplegia, cardioplegia. Hence plegia denotes more severe weakness than paresis.
**Cross References**
Paresis; Weakness

## Pleurothotonos

Pleurothotonos describes lateral flexion of the trunk due to spasm in paraspinal musculature. It may occur as one form of tonic spasm in tetanus.

Pisa syndrome is a truncal dystonia characterised by twisting and bending of the upper thorax, with involuntary flexion of the neck and head, to one side. Tilting symptoms occurring bilaterally may be labelled as "metronome Pisa syndrome". It may be seen in extrapyramidal disorders such as Parkinson's disease and multiple system atrophy, or as a rare extrapyramidal side effect caused by neuroleptic medications, or by cholinesterase inhibitors in Alzheimer's disease patients. Hence some form of cholinergic-dopaminergic imbalance would seem to be involved in pathogenesis. Other than discontinuing or adjusting medication, no specific treatment has been described.
**Reference**
Michel SF, Oscar AC, Correa TE, Alejandro PL, Micheli F. Pisa syndrome. *Clin Neuropharmacol*. 2015; **38**: 135–40.
**Cross References**
Dystonia; Emprosthotonos; Opisthtonos

## Plexopathy

Lesions confined to the brachial, lumbar, or sacral plexi may produce a constellation of motor and sensory signs (weakness, reflex diminution or loss, sensory loss) which cannot be ascribed to single or multiple roots (radiculopathy) or peripheral nerves (neuropathy). Lesions may involve the whole plexus (panplexopathy):

- Brachial: C5-T1
- Lumbar: L2-L4
- Sacral: L5-S3

or be partial, *e.g.* upper trunk of brachial plexus (C5-C6), producing "waiter's tip" posture (as for C5/C6 root avulsion); lower trunk of brachial plexus (C8-T1; as for C8/T1 root avulsion).

Neurophysiological studies may be helpful in distinguishing plexopathy from radiculopathy: sensory nerve action potentials (SNAPs) are reduced or absent in plexopathies because the lesion is located distal to the dorsal root ganglion (DRG), whereas SNAPs are normal in radiculopathies because the lesion is proximal to the DRG. EMG shows sparing of paraspinal muscles in a plexopathy because the lesion is, by definition, distal to the origin of the dorsal primary rami (*cf*. radiculopathy). Coexistence of radiculopathy and plexopathy may invalidate these simple rules.

- Recognised causes of brachial plexopathy include:

  Trauma: upper plexus: Dejerine-Klumpke paralysis ("waiter's tip" posture): lower plexus: Erb Duchenne paralysis (claw hand).
  Inflammation/Idiopathic: brachial neuritis, neuralgic amyotrophy.
  Malignant infiltration, *e.g.* carcinoma of lung (Pancoast), breast, +/− Horner's syndrome; pain a significant symptom.
  Post-radiation (*e.g.* after radiotherapy for malignant breast cancer with axillary spread; myokymic discharges may be seen on EMG).
  Tomaculous neuropathy.
  Hereditary neuropathy with liability to pressure palsies (HNLPP).

  Neurogenic thoracic outlet syndrome (rare): cervical rib or C7 transverse process or fibrous band compressing the lower trunk; may be surgically remediable.
- Recognised causes of lumbosacral plexopathy include:

  Compression; *e.g.* iliopsoas haematoma (anticoagulation, haemophilia), abscess (tuberculosis); abdominal aortic aneurysm; pregnancy (fetal head in the second stage of labour).
  Neoplasia (direct spread > metastasis).
  Trauma (rare; *cf*. brachial plexopathy).
  Post-radiation.
  Vasculitis (mononeuritis multiplex much commoner).

  Idiopathic.

Imaging with MRI is superior to CT for defining structural causes of plexopathy.

**References**

Chad DF. Nerve root and plexus disorders. In: Bogousslavsky J, Fisher M, editors. Textbook of neurology. Boston: Butterworth-Heinemann; 1998. p. 491–506.
Taylor BV, Kimmel DW, Krecke KN, Cascino TL. Magnetic resonance imaging in cancer-related lumbosacral plexopathy. *Mayo Clin Proc*. 1997; **72**: 823–9.

**Cross References**

Amyotrophy; Claw hand; Horner's syndrome; Myokymia; Nerve thickening; Neuropathy; Radiculopathy; "Waiter's tip" posture

## Polyganglionopathy

- see NEUROPATHY

## Polymyoclonus

- see MYOCLONUS

## Polyneuropathy

- see NEUROPATHY

## Polyopia

Polyopia, or polyopsia, or multiplication of images, is a visual illusory phenomenon in which a single target is seen as multiple images, most usually double but sometimes higher multiples (*e.g.* entomopia), persisting when looking away from the object. This may be likened to

"echoes" of the image, and eye movement may produce a trailing effect. Polyopia may be related to palinopsia.

Polyopia may occur as part of the visual aura of migraine, and has also been associated with occipital and occipito-parietal lesions, either bilateral or confined to the non-dominant hemisphere, and with drug abuse. It has also been described in disease of the retina and optic nerve, and occasionally in normal individuals.

The pathophysiology is unknown; suggestions include a defect of visual fixation or of visual integration; the latter may reflect pure occipital cortical dysfunction.

**Reference**

Pomeranz HD, Lessell S. Palinopsia and polyopia in the absence of drugs or cerebral disease. *Neurology*. 2000; **54**: 855–9.

**Cross References**

Entomopia; Illusion; Palinopsia

## "Popeye Arms"

In facioscapulohumeral (FSH) muscular dystrophy, the deltoid muscle is normally well preserved, whilst biceps and triceps are weak and wasted, giving rise to an appearance of the upper limbs sometimes labelled as "Popeye arms" or "chicken wings".

**Cross Reference**

Winging of the scapula

## Poriomania

Poriomania is a name sometimes given to prolonged wandering as an epileptic automatism, or a fugue state of nonconvulsive status epilepticus.

**Reference**

Mayeux R, Alexander MP, Benson DF, et al. Poriomania. *Neurology*. 1979; **29**: 1616–9.

**Cross References**

Automatism; Fugue; Seizures

## Porropsia

Porropsia, or teliopsia, is a form of metamorphopsia characterised by the misperception of objects as farther away from the observer than they really are (*cf.* peliopsia)

**Cross References**

Metamorphopsia; Peliopsia

## Positional Manoeuvres

- see HALLPIKE MANOEUVRE, HALLPIKE TEST; HEAD IMPULSE TEST; VESTIBULO-OCULAR REFLEXES

## Post-tetanic Potentiation

- see AUGMENTATION; FACILITATION

## Postural Hypotension

- see ORTHOSTATIC HYPOTENSION

## Postural Reflexes

Postures such as standing are largely reflex in origin, dependent upon involuntary muscle contraction in anti-gravity muscles. Interference with such reflex activity impairs normal standing. Postural and righting reflexes depend on the integration of labyrinthine, proprioceptive, exteroceptive, and visual stimuli, mostly in the brainstem but also involving the cerebral cortex. However, abnormalities in these reflexes are of relatively little diagnostic value except in infants.

One exception is extrapyramidal disease (parkinsonism, Huntington's disease, but not idiopathic dystonia) in which impairment or loss of postural reflexes may be observed. In the "pull test" the examiner stands behind the patient, who is standing comfortably, and pulls

briskly on the shoulders; if balance is normal, the patient takes a step back; with impaired postural reflexes, this may provoke repetitive steps backwards (retropulsion, festination) or even *en bloc* falling, due to the failure of reflex muscle contraction necessary to maintain equilibrium. Pushing the patient forward may likewise provoke propulsion or festination, but this manoeuvre is less safe since the examiner will not be placed to catch the patient should they begin to topple over.

Parkinson's disease patients have a characteristic pattern of muscle activation during the pull test which is not reversed after levodopa treatment.

**Reference**

Schestatsky P, Gomes-Araujo T, Gamarra A, Mello-Rieder C. Neurophysiological study of pull test in patients with Parkinson's disease and controls. *J Neurol*. 2011; **258**(Suppl 1): S29. (abstract O229).

**Cross References**

Dystonia; Festinant gait, Festination; Parkinsonism; Proprioception; "Rocket sign"

## Potomania

Potomania, or beer potomania, refers to excess drinking of beer (or cider) which may result in dilutional hyponatraemia if the diet is concurrently poor in salt and protein. This is a recognized association of central pontine myelinolysis.

**Reference**

Odier C, Nguyen DK, Panisset M. Central pontine and extrapontine myelinolysis: from epileptic and other manifestations to cognitive prognosis. *J Neurol*. 2010; **257**: 1176–80.

## Pourfour Du Petit Syndrome

Pourfour du Petit syndrome is characterized by mydriasis, widening of the palpebral fissure (eyelid retraction), exophthalmos, hyperhidrosis (*i.e.* inverse or reverse Horner's syndrome, sympathetic overactivity), flushing and increased intraocular pressure due to irritation of the sympathetic chain in the neck.

**Reference**

Al-Ansari A, Walters RJL. Case report: Pourfour du Petit syndrome in a patient with migraine. *J Neurol Neurosurg Psychiatry*. 2015; **86**: e4. doi:10.1136/jnnp-2015-312379.76.

**Cross Reference**

Horner's syndrome

## Pouting, Pout Reflex

The pout reflex consists of a pouting movement of the lips elicited by lightly tapping orbicularis oris with a finger or tendon hammer, or by tapping a spatula placed over the lips. This myotactic stretch reflex is indicative of a bilateral upper motor neurone lesion, which may be due to cerebrovascular small vessel ischaemic disease, motor neurone disease, or multiple sclerosis. It differs from the snout reflex, which refers to the reflex elicited by constant pressure on the philtrum. Hence the pout reflex is a phasic response, the snout reflex a tonic response.

**Reference**

Rossor M. Snouting, pouting and rooting. *Pract Neurol*. 2001; **1**: 119–21.

**Cross References**

Frontal release signs; Primitive reflexes

## "Prayer Sign"

An inability to oppose fully the palmar surfaces of the digits with the hands held in the praying position, the so-called "prayer sign", may be caused by ulnar neuropathy (*main en griffe*), Dupuytren's contracture, diabetic cheiroarthropathy, and camptodactyly.

**Cross References**

Camptodactyly; "Table top" sign

**Prehensile Thumb Sign**
- see FROMENT'S SIGN

**Presbyastasis**
Presbyastasis, or the disequilibrium of ageing, is a condition of elderly patients who present with imbalance and disequilibrium that cannot be ascribed to a particular disease state or single causative factor (*e.g.* vestibular disease, visual impairment, peripheral neuropathy). It is thought that abnormalities in sensory input, CNS sensory processing, control mechanisms for balance, and a decreased range of movement and strength may all contribute to symptoms. White matter changes on brain MRI have been associated with the condition. Vestibular rehabilitation therapy and avoidance of vestibular suppressant medications may be helpful.
**Reference**
Belal A, Glorig A. Disequilibrium of ageing (presbyastasis). *J Laryngol Otol*. 1986; **100**: 1037–41.
**Cross References**
Age-related signs; Astasia-abasia

**Presbycusis**
Presbycusis is a progressive sensorineural hearing loss, especially for high frequencies, developing with increasing age, which may reduce speech discrimination. It is thought to be due to age-related attrition of hair cells in the organ of Corti and/or spiral ganglion neurones.
**Reference**
Gates GA, Mills JH. Presbycusis. *Lancet*. 2005; **366**: 1111–20.
**Cross Reference**
Age-related signs

**Presbyopia**
Presbyopia is progressive far-sightedness which is increasingly common with increasing age, thought to be due to an age-related impairment of accommodation.
**Cross Reference**
Age-related signs

***Prèsque Vu***
- see DÉJÀ VU

**Pressure Provocation Test**
This is one of the provocative tests for carpal tunnel syndrome: it is positive if paraesthesia in the distribution of the median nerve develops when pressure is exerted on the palmar aspect of the patient's wrist at the level of the carpal tunnel for 60 s. It is sometimes referred to as the carpal compression test or Durkan's compression test.
**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**
Durkan's compression test; "Flick sign"; Phalen's sign; Tinel's sign

**Prevost's Sign**
Also known as Vulpian's sign, this refers to the acute and transient gaze palsy in a frontal lesion (*e.g.* infarct) which is towards the side of the lesion and away from the concurrent hemiparesis. The eyes can be brought to the other side with the oculocephalic manoeuvre or caloric testing. In contrast, thalamic and basal ganglia haemorrhages produce forced deviation of the eyes to the side contralateral to the lesion (wrong-way eyes).

## Priapism

Priapism is an unintended, sustained, and usually painful erection of the penis unrelated to sexual activity. It may occur with intramedullary spinal cord lesions (*e.g.* multiple sclerosis) which damage the lumbosacral erection centres, and has also been reported with lumbar canal spinal stenosis. There are also non-neurological causes, such as haematological conditions (sickle cell anaemia, polycythaemia rubra vera) which may cause intrapenile thromboses.

## Primitive Reflexes

Reflexes which are normally found in infancy but which disappear with brain maturation during childhood may be labelled as "primitive reflexes" if they re-emerge in adulthood as a consequence of pathological states. Many of these reflexes are seen with frontal lobe pathology (*e.g.* grasp, pout/snout, palmomental, rooting, corneomandibular) and hence may also be known as "frontal release signs". However, the term "primitive reflex" could equally apply to Babinski's sign which is not necessarily frontal in origin.

**References**

Paulson G, Gottlieb G. Developmental reflexes: the reappearance of foetal and neonatal reflexes in aged patients. *Brain*. 1968; **91**: 37–52.

Schott JM, Rossor MN. The grasp and other primitive reflexes. *J Neurol Neurosurg Psychiatry*. 2003; **74**: 558–60.

**Cross References**

Babinski's sign (1); Corneomandibular reflex; Frontal release signs; Grasp reflex; Palmomental reflex; Pout reflex; Rooting reflex

## Procerus Sign

A focal dystonia of the procerus muscle, denoted the procerus sign, has been suggested to contribute to the "astonished", "worried" or "reptile-like" facial expression typical of progressive supranuclear palsy, which may also be characterised by reduced blinking, lid retraction, and gaze palsy. All these features contrast with the hypomimia of Parkinson's disease. It has also been described in corticobasal degeneration.

**References**

Romano S, Colosimo C. Procerus sign in progressive supranuclear palsy. *Neurology*. 2001; **57**: 1928.

Shibasaki Warabi Y, Nagao M, Bandoh M, Kanda T, Hirai S. Procerus sign in corticobasal degeneration. *Intern Med*. 2002; **41**: 1217–8.

**Cross References**

Blinking; Dystonia; Hypomimia; Parkinsonism

## Pronator Drift

Pronator drift describes a gradual pronation of the forearm observed when the arms are held straight forward, palms up, with the eyes closed. It suggests a contralateral corticospinal tract lesion and may be accompanied by downward drift of the arm and flexion of the fingers and/or elbow. It reflects the relative weakness of supinators versus pronators in the arm with a pyramidal lesion, in addition to the relative weakness of extensors versus flexors. It may be an early sign of corticospinal tract dysfunction.

**Cross References**

Forearm and finger rolling, Upper motor neurone (UMN) syndrome; Weakness

## Proprioception

Proprioception sensation, or joint position sense, is knowledge about ones position in space, originating from sensory receptors in skin, muscle, and viscera. Proprioceptive information is carried within the dorsal columns of the spinal cord (more reliably so than vibration sensation, though not necessarily exclusively). Lesions affecting this part of the cord, particularly in the cervical region (*e.g.* subacute combined degeneration of the cord due to vitamin $B_{12}$ deficiency, tabes dorsalis), lead to impairments of proprioception with sparing of spinothalamic sensations (pin-prick, temperature) producing a dissociated sensory loss. Impairment

of proprioception leads to sensory ataxia which may manifest clinically with pseudoathetosis or pseudochoreoathetosis (also seen in useless hand of Oppenheim) and with a positive Romberg's sign.

**Reference**

Gilman S. Joint position sense and vibration sense. *J Neurol Neurosurg Psychiatry*. 2002; **73**: 473–7.

**Cross References**

Ataxia; Dissociated sensory loss; Myelopathy; Pseudoathetosis; Pseudochoreoathetosis; Rombergism, Romberg's sign; Useless hand of Oppenheim; Vibration

## Proptosis

Proptosis is forward displacement of the eyeball, an exaggerated degree of exophthalmos. There may be lower lid retraction. Proptosis may be assessed clinically by standing directly behind the patient and gradually tipping the head back, observing when the globe of the eyeball first comes into view; this is most useful for asymmetric proptosis. An exophthalmometer may be used to measure proptosis.

Once established, it is crucial to determine whether the proptosis is axial or non-axial. Axial proptosis reflects increased pressure within or transmitted through the cone of extraocular muscles (*e.g.* thyroid ophthalmopathy, cavernous sinus thrombosis), whereas non-axial proptosis suggests pressure from an orbital mass outside the cone of muscles (*e.g.* orbital lymphoma, pseudotumour, mucocele). Pulsatile axial proptosis may occur in caroticocavernous fistula, in which case there may be a bruit audible by auscultation over the eye. Venous angioma of the orbit may cause an intermittent proptosis associated with straining, bending, coughing or blowing the nose.

Dedicated orbital CT or MRI, the latter with fat-suppression sequences and intravenous gadolinium contrast, may be required to detect intraorbital masses.

Middle cranial fossa tumours may cause pressure on the veins of the cavernous sinus with secondary intraorbital venous congestion causing a "false-localising" proptosis.

**Cross References**

Exophthalmos; "False-localising signs"; Lid retraction

## Propulsion

- see FESTINANT GAIT, FESTINATION; POSTURAL REFLEXES

## Prosopagnosia

Prosopagnosia is a form of visual agnosia characterized by an inability to recognize human faces or equivalent stimuli. This may be developmental, or acquired loss of recogntion (hence, a retrograde defect) and inability to learn new faces (anterograde defect). As with more pervasive visual agnosia, prosopagnosia may be characterised as:

- *apperceptive*: due to faulty perceptual analysis of faces; or
- *associative*: a semantic defect in recognition.

Familiar individuals may be recognized by their voices or clothing or hair; hence, the defect may be one of visually triggered episodic memory. It is important to note that the defect is not limited solely to faces; it may encompass animals ("zooagnosia"), or cars.

Prosopagnosia is often found in association with a visual field defect, most often a left superior quadrantanopia or even hemianopia, although for the diagnosis of prosopagnosia to be made this should not be sufficient to produce a perceptual deficit. Alexia and achromatopsia may also be present, depending on the exact extent of the underlying lesion.

Anatomically, prosopagnosia occurs most often in association with bilateral occipitotemporal lesions involving the inferior and mesial visual association cortices in the lingual and fusiform gyri, sometimes with subjacent white matter. Unilateral non-dominant (right) hemisphere lesions have occasionally been associated with prosopagnosia, and a syndrome of

progressive prosopagnosia associated with selective focal atrophy of the right temporal lobe has been reported. Involvement of the periventricular region on the left side may explain accompanying alexia, and disconnection of the inferior visual association cortex (area V4) may explain achromatopsia.

Pathological causes of prosopagnosia include:

- Cerebrovascular disease: by far the most common cause.
- Tumour, *e.g.* glioma, extending from one hemisphere to the other via the splenium of the corpus callosum.
- Epilepsy (paroxysmal prosopagnosia), due to bilateral foci or spread from one occipital focus to the contralateral hemisphere.
- Focal right temporal lobe atrophy: possibly a non-dominant hemisphere form of semantic dementia.
- Herpes simplex encephalitis, usually as part of an extensive amnesic syndrome (although memory impairment may put this outwith the operational criteria for an agnosia).

A developmental (or "congenital") form of prosopagnosia suggests that facial recognition is a separate neuropsychological function, since acquired pathologies do not respect functional boundaries.

**References**

Evans JJ, Heggs AJ, Antoun N, Hodges JR. Progressive prosopagnosia associated with selective right temporal lobe atrophy. A new syndrome? *Brain*. 1995; **118**: 1–13.
Farah MJ. Visual agnosia: disorders of object recognition and what they tell us about normal vision. Cambridge: MIT Press; 1995.
Larner AJ. Lewis Carroll's Humpty Dumpty: an early report of prosopagnosia? *J Neurol Neurosurg Psychiatry*. 2004; **75**: 1063.
Nunn JA, Postma P, Pearson R. Developmental prosopagnosia: should it be taken at face value? *Neurocase*. 2001; **7**: 15–27.

**Cross References**

Achromatopsia; Agnosia; Alexia; Hemianopia; Phonagnosia; Quadrantanopia; Visual agnosia; Zooagnosia

## Prosopoplegia

- see BELL'S PALSY; FACIAL PARESIS, FACIAL WEAKNESS

## Proust Phenomenon

The Proust phenomenon, named after the author Marcel Proust (1871–1922), is the observation that particular odours may trigger reminders of autobiographical memories. There is some experimental evidence that olfactory stimuli can cue autobiographical memories more effectively than cues from other sensory modalities. The "petite madeleines phenomenon" has been used to describe sudden triggering of memories in individuals with amnesia due to thalamic infarction.

**References**

Lucchelli F, Muggia S, Spinnler H. The "Petites Madeleines" phenomenon in two amnesic patients: sudden recovery of forgotten memories. *Brain*. 1995; **118**: 167–83.
Toffolo MB, Smeets MA, van den Hout MA. Proust revisited: odours as triggers of aversive memories. *Cogn Emot*. 2012; **26**: 83–92.

**Cross Reference**

Amnesia

## Proximal Limb Weakness

Weakness affecting predominantly the proximal limb musculature (shoulder abductors and hip flexors) is a pattern frequently observed in myopathic and dystrophic muscle disorders and neuromuscular junction transmission disorders, much more so than predominantly

distal weakness (the differential diagnosis of which encompasses myotonic dystrophy, distal myopathy of Miyoshi type, desmin myopathy, and, rarely, myasthenia gravis). Some neuropathic disorders may also cause a predominantly proximal weakness (*e.g.* Guillain-Barré syndrome). Age of onset and other clinical features may help to narrow the differential diagnosis:

- painful muscles may suggest an inflammatory cause (polymyositis, dermatomyositis);
- fatiguability may suggest myasthenia gravis (although lesser degrees of fatigue may be seen in myopathic disorders);
- weakness elsewhere may suggest a specific diagnosis (*e.g.* face in facioscapulohumeral muscular dystrophy, diaphragm in acid-maltase deficiency);
- cachexia points to underlying malignant disease;
- calf pseudohypertrophy suggests Duchenne or Becker muscular dystrophy;
- autonomic features and post-tetanic potentiation of reflexes occur in Lambert-Eaton myasthenic syndrome.

Investigations including blood creatine kinase, neurophysiology, and muscle biopsy may be required to determine exact diagnosis. Differential diagnosis includes:

- Myopathies:
  - Inflammatory: polymyositis, dermatomyositis.
  - Progressive muscular dystrophies: Duchenne, Becker, limb-girdle, facioscapulohumeral (FSH).
  - Metabolic: acid maltase deficiency; thyroid dysfunction, Cushing's syndrome.
  - Non-metastatic feature of malignant disease.
  - Drug-induced: alcohol, steroids.
- Neuromuscular junction transmission disorders:
  - Myasthenia gravis.
  - Lambert-Eaton myasthenic syndrome.
- Neuropathy:
  - Guillain-Barré syndrome.

**Cross References**
Facilitation; Fatigue

## Pruritus

Pruritus or itch may be a consequence of dermatologocial disorders or may be neuropathic in origin. Itch may share similar neural pathways as pain. Recognized neurological causes of pruritus include some peripheral neuropathies, multiple sclerosis, neuromyelitis optica, and Creutzfeldt-Jakob disease.

**References**

Cohen OS, Chapman J, Lee H, et al. Pruritus in familial Creutzfeldt-Jakob disease: a common symptom associated with central nervous system pathology. *J Neurol*. 2011; **258**: 89–95.

Davidson S, Giesler GJ. The multiple pathways for itch and their interactions with pain. *Trends Neurosci*. 2010; **33**: 550–8.

Elsone L, Townsend T, Mutch K, et al. Neuropathic pruritus (itch) in neuromyelitis optica. *Mult Scler*. 2013; **19**: 475–9.

## Pseudo-Abducens Palsy

- see ABDUCENS (VI) NERVE PALSY

## Pseudoachromatopsia

Pseudoachromatopsia is failure on tests of colour vision (*e.g.* pseudoisochromatic plates) which is not due to central or peripheral achromatopsia, for example due to visual neglect.

**Cross References**

Achromatopsia; Neglect

## Pseudoagnosia

- see AGNOSIA

## Pseudo-Argyll Robertson Pupil

A pseudo-Argyll Robertson pupil shows light-near dissociation of pupillary reactions but, unlike the "true" Argyll Robertson pupil, there is no miosis or pupil irregularity. Indeed the pupil may be dilated (mydriasis) and resemble a Holmes-Adie pupil. The latter may be differentiated on the basis of its response to dilute (0.2%) pilocarpine: Holmes-Adie pupil results from a peripheral lesion and shows denervation supersensitivity, constricting with dilute pilocarpine, whereas the pseudo-Argyll Robertson pupil results from a central lesion and does not respond. Pseudo-Argyll Robertson pupil has been reported in:

- diabetes mellitus.
- multiple sclerosis.
- Wernicke's encephalopathy (thiamine deficiency).
- neurosarcoidosis.
- tumour.
- haemorrhage.
- aberrant oculomotor (III) nerve regeneration.
- spinocerebellar ataxia type 1 (SCA1).

**Cross References**

Argyll Robertson pupil; Holmes-Adie pupil, Holmes-Adie syndrome; Miosis; Mydriasis

## Pseudoathetosis

Pseudoathetosis is the name given to athetoid-like movements, most usually of the outstretched fingers ("piano-playing fingers") and hands, resulting from sensory ataxia (impaired proprioception), worse with the eyes closed. There may also be chorea-like movements, hence pseudochoreoathetosis. Causes include any interruption to the anatomical pathway mediating proprioception, most often lesions in the dorsal cervical cord (*e.g.* multiple sclerosis, subacute combined degeneration of the cord due to vitamin $B_{12}$ deficiency or nitrous oxide overuse), but also lesions of the large (myelinated) peripheral nerve fibres, and of the parietal lobe.

**References**

Ghika J, Bogousslavsky J. Spinal pseudoathetosis: a rare, forgotten syndrome, with a review of old and recent descriptions. *Neurology*. 1997; **49**: 432–7.

Lo YL, See S. Images in clinical medicine. Pseudoathetosis. *N Engl J Med*. 2010; **363**: e29.

Spitz M, Costa Machado AA, Carvalho Rdo C, et al. Pseudoathetosis: report of three patients. *Mov Disord*. 2006; **21**: 1520–2.

**Cross References**

Athetosis; Chorea, Choreoathetosis; Proprioception; Pseudochoreoathetosis

## Pseudo-Babinski Sign

Pseudo-Babinski sign is the name given to dystonic extension of the great toe on stroking the sole of the foot, as when trying to elicit Babinski's sign, with which this may be confused, although pseudo-Babinski responses persist for longer, and spontaenous extension of the toe, striatal toe, may also be present. Pseudo-Babinski signs may normalise after dopaminergic treatment in dopa-responsive dystonia.

**Reference**
Horstink MWIM, Haaxma C, Bloem BR. Babinski, pseudo-Babinski, and dystonia. *Arch Neurol*. 2007; **64**: 1207–9.
**Cross References**
Babinski sign (1); Striatal toe

## Pseudobitemporal Hemianopia
- see HEMIANOPIA

## Pseudobulbar Affect
- see EMOTIONALISM, EMOTIONAL LABILITY; PATHOLOGICAL CRYING, PATHOLOGICAL LAUGHTER; PSEUDOBULBAR PALSY

## Pseudobulbar Palsy
Pseudobulbar palsy, or spastic bulbar palsy, describes bilateral upper motor neurone lesions affecting fibres passing to the cranial nerve nuclei (*cf*. bulbar palsy). This leads to a variety of clinical features, including:

- difficulty with speech: spastic dysarthria, dysphonia.
- difficulty with swallowing: dysphagia.
- brisk jaw jerk and pout reflex; there may be trismus.
- gag reflex may be depressed or exaggerated.
- slow, spastic, tongue movements.

There may be associated emotional lability, or pathological laughter and crying ("pseudobulbar affect"), and a gait disorder with *marche à petit pas*. There are otherwise few signs in the limbs, aside from brisk reflexes and upgoing plantar responses (Babinski's sign).

Recognised causes of pseudobulbar palsy include:

- Motor neurone disease (in which there may be coincident bulbar palsy).
- Multiple sclerosis.
- Bilateral internal capsule lacunar infarctions, widespread small vessel ischaemic disease (Binswanger's disease).
- Progressive supranuclear palsy: pseudobulbar palsy was part of the initial descripition of this condition by Steele, Richardson, and Olszewski.
- Congenital childhood suprabulbar palsy (Worster-Drought syndrome; perisylvian syndrome).

Pseudobulbar affect may respond to serotonin reuptake inhibitors.

**Cross References**
Babinski's sign (1); Bulbar palsy; Dysarthria; Dysphagia; Dysphonia; Emotionalism, Emotional lability; Gag reflex; Jaw jerk; *Marche à petit pas*; Pathological crying, Pathological laughter; Trismus; Upper motor neurone (UMN) syndrome

## Pseudochoreoathetosis
Pseudochoreoathetosis is a name which has been given to choreoathetoid type involuntary movements, including dystonic movements, which result from a loss or impairment of proprioception. These may be observed with lesions anywhere along the proprioceptive pathways, including parietal cortex, thalamus (there may be associated ataxic hemiparesis and hemihypoaesthesia), spinal cord, dorsal root ganglia (neuronopathy), and mononeuropathy.

**References**
Dineen JM, Greenberg SA. Pseudochoreoathetosis in sensory ataxic variant of Guillain-Barré syndrome. *Muscle Nerve*. 2014; **50**: 300–1.

Kim JW, Kim SH, Cha JK. Pseudochoreoathetosis in four patients with hypesthetic ataxic hemiparesis in a thalamic lesion. *J Neurol*. 1999; **246**: 1075–9.
Sharp FR, Rando TA, Greenberg SA, Brown L, Sagar SM. Pseudochoreoathetosis. Movements associated with loss of proprioception. *Arch Neurol*. 1994; **51**: 1103–9.
**Cross References**
Ataxic hemiparesis; Chorea, Choreoathetosis; Dystonia; Proprioception; Pseudoathetosis; Useless hand of Oppenheim

## Pseudodementia

The term pseudodementia has been used by some authors to describe cognitive impairments which result from affective disorders, most commonly anxiety and depression. The terms "dementia syndrome of depression" and "depression-related cognitive dysfunction" have also been used. The pattern of cognitive deficits in individuals with depression most closely resembles that seen in so-called subcortical dementia, with bradyphrenia, attentional and executive deficits. In addition there may be evident lack of effort and application, frequent "No" or "Don't know" answers, approximate answers (Ganser phenomenon, *vorbereiden*), and evidence of mood disturbance (tearfulness). Memory loss for recent and distant events may be equally severe (*cf*. temporal gradient of memory loss in dementia, *e.g.* due to Alzheimer's disease). A 22-item checklist to help differentiate pseudodementia from Alzheimer's disease has been described, based on clinical history, behaviour and mental status.

The recognition of pseudodementia is important since the deficits are often at least partially reversible with appropriate treatment with antidepressant medications. However, it should be borne in mind that depression is sometimes the presenting symptom of an underlying neurodegenerative dementing disorder such as Alzheimer's disease. Psychomotor retardation in dementia syndromes may also be mistaken for depression. Longitudinal assessment may be required to differentiate between these diagnostic possibilities.
**References**
Andrews C. Pseudodementia responding to antidepressant therapy. *Prog Neurol Psychiatry*. 2002; **6**(1): 26.
Kiloh L. Pseudodementia. *Acta Psychiatr Scand*. 1961; **37**: 336–51.
Roose SP, Devanand DP. The interface between dementia and depression. London: Martin Dunitz; 1999.
Wells CE. Pseudodementia. *Am J Psychiatry*. 1979; **136**: 895–900.
**Cross References**
Attention; Bradyphrenia; Dementia; Ganser phenomenon; Psychomotor retardation

## Pseudodiplopia

- see PALINOPSIA

## Pseudo-Foster Kennedy Syndrome

- see FOSTER KENNEDY SYNDROME

## Pseudohallucination

The term pseudohallucination has been used in different ways. In the European psychopathological tradition, it may refer simply to vivid visual imagery, whereas in the American arena it may refer to hallucinations that are recognised for what they are, *i.e.* the patient has insight into their non-real nature. The term "non-psychotic hallucinations" has also been proposed for these phenomena. Some patients with dementia with Lewy bodies certainly realise that their visual hallucinations do not correspond to external reality, and similar experiences may occur with dopamine agonist treatment. Pseudohallucinations may fall along a continuum of perceptual disorders.
**References**
van der Zwaard R, Polak M. Pseudohallucinations: a pseudoconcept? A review of the validity of the concept, related to associate symptomatology. *Compr Psychiatry*. 2001; **42**: 42–50.

Wearne D, Genetti A. Pseudohallucinations versus hallucinations: wherein lies the difference? *Australas Psychiatry*. 2015; **23**: 254–7.
**Cross References**
Charles Bonnet syndrome; Hallucination

## Pseudohypertrophy
- see CALF HYPERTROPHY; MUSCLE HYPERTROPHY

## Pseudo-Internuclear Ophthalmoplegia
Pseudo-internuclear ophthalmoplegia (pseudo-INO) describes a disorder of eye movements with impaired adduction in one eye and horizontal nystagmus in the abducting eye (*i.e.* signs as seen in an internuclear ophthalmoplegia) but without an intrinsic brainstem lesion. This sign may be seen in:

- Myasthenia gravis (a diagnosis which is always worthy of consideration in a patient with an "isolated INO") due to extraocular muscle weakness.
- Brainstem compression due to subdural haematoma with transtentorial herniation.
- Cerebellar mass lesion.
- Guillain-Barré syndrome, Miller Fisher syndrome.
- Thyroid ophthalmopathy.
- Orbital pseudotumour.

The preservation of rapid saccades despite restriction of eye movements in myasthenia gravis may result from selective sparing of pale global muscle fibres which generate high-speed movements.
**References**
Glaser JS. Myasthenic pseudo-internuclear ophthalmoplegia. *Arch Ophthalmol*. 1966; **75**: 363–6.
Khanna S, Liao K, Kaminski HJ, Tomsak RL, Joshi A, Leigh RJ. Ocular myasthenia revisited: insights from pseudo-internuclear ophthalmoplegia. *J Neurol*. 2007; **254**: 1569–74.
**Cross References**
Internuclear ophthalmoplegia (INO); One-and-a-half syndrome

## Pseudomyotonia
The term pseudomyotonia has been used in various ways:

- It may be used to describe the clinical appearance of myotonia (slow muscular relaxation after contraction) in the absence of myotonic discharges on electromyography. Pseudomyotonia is most commonly observed as the slow-relaxing or "hung-up" tendon reflexes (Woltman's sign) of hypothyroidism, although other causes are described.
- Pseudomyotonia has also been used to describe difficulty opening the hand in cervical osteoarthritis, although muscle relaxation is normal; finger flexion on attempted extension has been explained as due to aberrant axonal regeneration of the C7 root.
- The term pseudomyotonia has also been used to describe neuromyotonia and myokymia (as, for example, in Isaacs syndrome), to distinguish it from myotonia.

**References**
Coers C, Teleman-Toppet N, Durdu J. Neurogenic benign fasciculations, pseudomyotonia, and pseudotetany. A disease in search of a name. *Arch Neurol*. 1981; **38**: 282–7.
Satoyoshi E, Doi Y, Kinoshita M. Pseudomyotonia in cervial root lesions with myelopathy. A sign of the misdirection of regenerating nerve. *Arch Neurol*. 1972; **27**: 307–13.
**Cross References**
Myotonia; Neuromyotonia; Woltman's sign

## Pseudo-One-and-a-Half Syndrome

Pseudo-one-and-a-half syndrome is the eye movement disorder of one-and-a-half syndrome without a brainstem lesion. Myasthenia gravis and Guillain-Barré syndrome are recognised causes.

**Reference**

Davis TL, Lavin PJ. Pseudo one-and-a-half syndrome with ocular myasthenia. *Neurology*. 1989; **39**: 1553.

**Cross Reference**

One-and-a-half syndrome

## Pseudopapilloedema

Pseudopapilloedema is the name given to elevation of the optic disc that is not due to oedema (*i.e.* intracranial pressure is not raised). There may or may not be visible drusen (hyaline bodies). In distinction to oedematous disc swelling, the nerve fibre layer is not hazy and the underlying vessels are not obscured; however, spontaneous retinal venous pulsation is usually absent, and haemorrhages may be seen, so these are not reliable distinguishing features. Visual acuity is usually normal, but visual field defects (most commonly in the inferior nasal field) may be found.

**Cross References**

Disc swelling; Papilloedema; Retinal venous pulsation

## Pseudoparesis

- see PARESIS; WEAKNESS

## Pseudoptosis

Ptosis, drooping of the eyelid, may need to be differentiated from pseudoptosis or functional ptosis. This may result simply from a redundant tarsal skin fold, especially in older patients, or be a functional condition. Frontalis underactivity may be a clinical indicator of the latter diagnosis (*cf*. compensatory overactivity of frontalis with other causes of ptosis, *e.g.* myasthenia gravis).

The term pseudoptosis has also been used in the context of hypotropia; when the non-hypotropic eye fixates, the upper lid follows the hypotropic eye and appears ptotic, disappearing when fixation is with the hypotropic eye.

**References**

Hop JW, Frijns CJ, van Gijn J. Psychogenic pseudoptosis. *J Neurol*. 1997; **244**: 623–4.
Stone J. Pseudo-ptosis. *Pract Neurol*. 2002; **2**: 364–5.

**Cross Reference**

Ptosis

## Pseudoradicular Syndrome

Thalamic lesions may sometimes cause contralateral sensory symptoms in an apparent radicular (*e.g.* C8) distribution. If associated with perioral sensory symptoms this may be known as the cheiro-oral syndrome.

**Reference**

Kim JS. Restricted acral sensory syndrome following minor stroke: further observations with special reference to differential severity of symptoms among individual digits. *Stroke*. 1994; **25**: 2497–502.

**Cross Reference**

"False-localising signs"

## Pseudo-Von Graefe's Sign

Pseudo-von Graefe's sign is involuntary retraction or elevation of the upper eyelid (*cf*. von Graefe's sign), medial rotation of the eye, and pupillary constriction seen on attempted downgaze or adduction of the eye. This constellation of findings is said to be a lid-gaze synkinesis

following aberrant axonal regeneration after an oculomotor (III) nerve palsy, usually of traumatic or chronic compressive rather than ischaemic origin.

**Cross References**

Lid retraction; Synkinesia, Synkinesis; Von Graefe's sign

**Psychic Akinesia**

- see ATHYMHORMIA

**Psychic Blindness**

- see VISUAL AGNOSIA

**Psychic Paralysis of Gaze**

- see BALINT'S SYNDROME; OCULAR APRAXIA

**Psychomotor Retardation**

Psychomotor retardation is a slowness of thought (bradyphrenia) and movement (bradykinesia) seen in psychiatric disorders, particularly depression, or as a consequence of drug use (*e.g.* multiple analgesic, sedative, hypnotic medications). It may be confused with the akinesia of parkinsonism and with states of abulia or catatonia. Psychomotor retardation may also be a feature of the "subcortical" type of dementia, or of impairments of arousal (obtundation).

**Cross References**

Abulia; Akinesia; Catatonia; Dementia; Obtundation; Parkinsonism

**Psychomotor Signs**

- see FRONTAL RELEASE SIGNS

**Ptarmus**

- see SNEEZING

**Ptosis**

Ptosis, or blepharoptosis, is the name given to drooping of the eyelid. This may be due to mechanical causes such as aponeurosis dehiscence, or neurological disease, in which case it may be congenital or acquired, partial or complete, unilateral or bilateral, fixed or variable, isolated or accompanied by other signs, *e.g.* miosis in a Horner's syndrome; diplopia in myasthenia gravis; mydriasis and downward and outward deviation of the eye in an oculomotor (III) nerve palsy.

Ptosis may result from pathology in a variety of locations: brainstem disease involving the oculomotor (III) nerve; anywhere along the oculosympathetic autonomic pathway causing a Horner's syndrome; or cortical disease (*e.g.* infarction) reflecting hemispheric control of the eyelid (probably bilaterally represented).

When considering the cause of ptosis, the differential diagnosis is broad. Recognised neurological causes include:

- Congenital:
  - Cranial nerve dysinnervation disorder.
  - Congenital Horner's syndrome.
  - Oculomotor-trigeminal (or trigeminal-levator) synkinesis: Marcus Gunn jaw-winking phenomenon, or inverse Marcus Gunn phenomenon (ptosis on jaw opening).
- Neurogenic:
  - Supranuclear lesion:
    - Hemiparesis: due to cortical infarct; ptosis usually ipsilateral, incomplete.

Duane syndrome: ptosis on eye adduction, due to supranuclear levator inhibition; usually with family history.

Oculomotor (III) nerve:

Hypertension, diabetes mellitus: ptosis often complete; in a superior divisional third nerve palsy partial ptosis is associated with superior rectus weakness only.

Compressive lesion (*e.g.* posterior communicating artery aneurysm): ptosis usually incomplete; ptosis may be present with subarachnoid haemorrhage.

Guillain-Barré syndrome.

Facial paresis.

- Neuromuscular junction:

Myasthenia gravis: ptosis variable, bilateral or unilateral.

Excessive botulinum toxin, *e.g.* given for treatment of blepharospasm.

- Myogenic: ptosis usually bilateral:

Mitochondrial disease (CPEO).
Myotonic dystrophy.

Oculopharyngeal muscular dystrophy (OPMD).

Local, ophthalmological causes should also be considered, such as age-related aponeurosis dehiscence, trauma, thyroid eye disease, lid inflammation (chalazion), and lymphoma. Pseudoptosis (*q.v.*) enters the differential diagnosis.

Enhanced ptosis, worsening of ptosis on one side when the other eyelid is held elevated in a fixed position, may be demonstrated in myasthenia gravis and Lambert Eaton myasthenic syndrome.

**References**

Ahmad K, Wright M, Lueck CJ. Ptosis. *Pract Neurol*. 2011; **11**: 332–40.
Caplan LR. Ptosis. *J Neurol Neurosurg Psychiatry*. 1974; **37**: 1–7.

**Cross References**

Blepharospasm; Curtaining; Diplopia; Divisional palsy; Enhanced ptosis; Ewart phenomenon; Horner's syndrome; Ice pack test; Jaw winking; Miosis; Mydriasis; Pseudoptosis; Pupil sparing; Synkinesia, Synkinesis

## Ptyalism

- see SIALORRHOEA

## Pulfrich Phenomenon

The Pulfrich phenomenon is the observation that a pendulum swinging from side to side appears to traverse a curved trajectory. This is a stereo-illusion resulting from latency disparities in the visual pathways, most commonly seen as a consequence of slowed conduction in a demyelinated optic nerve following unilateral optic neuritis. A tinted coloured lens in front of the good eye can alleviate the symptom (or induce it in the normally sighted).

**References**

McGowan G, Ahmed TY, Heron G, Diaper C. The Pulfrich phenomenon; clumsiness and collisions which can be ameliorated. *Pract Neurol*. 2011; **11**: 173–6.
Rushton D. Use of the Pulfrich pendulum for detecting abnormal delay in the visual pathways in multiple sclerosis. *Brain*. 1975; **98**: 283–96.

**Cross References**

Phosphene; Relative afferent pupillary defect (RAPD)

## "Pull Test"

- see POSTURAL REFLEXES

## Punding

Punding describes repetitive pointless behaviours, with a compulsive flavour to them, carried on for long periods of time to the exclusion of other activities. It is frequently related to previous occupation or hobbies but is seldom pleasurable (writing a book might be an example?). It occurs in Parkinson's disease but the incidence is low (1.4% in one study). It is thought to be related to dopaminergic stimulation, and may be associated with impulse control disorders such as pathological gambling and hypersexuality.

**References**

O'Sullivan SS, Evans AH, Lees AJ. Punding in Parkinson's disease. *Pract Neurol*. 2007; **7**: 397–9.

Spencer AH, Rickards H, Fasano A, Cavanna AE. The prevalence and clinical characteristics of punding in Parkinson's disease. *Mov Disord*. 2011; **26**: 578–86.

**Cross References**

Gambling; Hypersexuality

## Pupillary Reflexes

Two pupillary reflexes, first described by Robert Whytt (1714–1766) and sometimes known as Whytt's reflex, are routinely examined in clinical practice:

- *Light reflex*:

  The eye is illuminated directly and the reaction (constriction) observed; the consensual light reflex is observed by illuminating the contralateral eye. In an eye with poor visual acuity, a relative afferent pupillary defect may be observed using the "swinging flashlight test". The afferent pathway subserving the light reflex is optic nerve to thalamus, brainstem, and Edinger-Westphal nucleus, with the efferent limb (pupillomotor parasympathetic fibres) in the oculomotor (III) nerve. The contralateral (consensual) response results from fibres crossing the midline in the optic chiasm and in the posterior commissure at the level of the rostral brainstem.

  Paradoxical constriction of the pupil in darkness (Flynn phenomenon) has been described.

- *Accommodation reflex*:

  This is most conveniently examined by asking the patient to look into the distance, then focus on a near object (sufficiently close to necessitate convergence of the visual axes) when pupil constriction should occur (accommodation-convergence synkinesis). The afferent pathways subserving this response are less certain than for the light reflex, and may involve the occipital cortex, although the final (efferent) pathway via Edinger-Westphal nucleus and oculomotor (III) nerve is common to both accommodation and light reflexes.

In comatose patients, fixed dilated pupils may be observed with central diencephalic herniation, whereas midbrain lesions produce fixed midposition pupils.

A dissociation between the light and accommodation reactions (light-near pupillary dissociation, *q.v.*) may be observed.

**Reference**

Kawasaki A. Approach to the patient with abnormal pupils. In: Biller J, editor. Practical neurology. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 2002. p. 135–46.

**Cross References**

Argyll Robertson pupil; Ciliospinal response; Cortical blindness; Flynn phenomenon; Light-near pupillary dissociation; Miosis; Mydriasis; Relative afferent pupillary defect (RAPD); Swinging flashlight sign

## Pupil Sparing

Oculomotor (III) nerve lesions may be pupil sparing (normal response to light) or pupil-involving (mydriasis, loss of light reflex). The latter situation usually implies a "surgical" cause of oculomotor nerve palsy (*e.g.* posterior communicating artery aneurysm), especially if extraocular muscle function is relatively preserved. Pupil sparing suggests a "medical" cause (*e.g.* diabetes mellitus, hypertension) especially if the palsy is otherwise complete (complete ptosis, eye deviated downwards and outwards). This disparity arises because pupillomotor fibres run on the outside of the oculomotor nerve, and are relatively spared by ischaemia but are vulnerable to external compression. However, the distinction is not absolute; imaging for an aneurysm (by means of spiral CT, MRA, or catheter angiography) may be necessary if the clinical scenario leaves room for doubt.

**Cross References**

Oculomotor (III) nerve palsy; Ophthalmoparesis, Ophthalmoplegia; Ptosis; Pupillary reflexes

## Pure Word Blindness

- see ALEXIA

## Pure Word Deafness

The term word deafness was first used by Henry Charlton Bastian in 1869 (as was word blindness). Pure word deafness is a rare condition characterized by an inability to comprehend and discriminate spoken language, despite adequate hearing as measured by audiometry, and with preserved spontaneous speech, reading, reading comprehension, and writing (*i.e.* no aphasia, alexia, or agraphia). Lip reading may assist in the understanding of others who sometimes seem to the patient as though they are speaking in a foreign language. Patients can copy and write spontaneously, follow written commands, but cannot write to dictation. Word repetition tasks are impaired. There may be associated amusia, depending on the precise location of cerebral damage.

Pure word deafness has been variously conceptualised as a form of auditory agnosia or a subcortical sensory aphasia.

Pure word deafness is most commonly associated with bilateral lesions of the temporal cortex or subcortical lesions whose anatomical effect is to damage the primary auditory cortex or isolate it (*e.g.* from Wernicke's area) through lesions of the auditory radiation; unilateral lesions producing this syndrome have been reported. Very rarely pure word deafness has been associated with bilateral brainstem lesions at the level of the inferior colliculi.

**References**

Meyer B, Kral T, Zentner J. Pure word deafness after resection of a tectal plate glioma with preservation of wave V of brain stem auditory evoked potentials. *J Neurol Neurosurg Psychiatry*. 1996; **61**: 423–4.

Roberts M, Sandercock P, Ghadiali E. Pure word deafness and unilateral right temporoparietal lesions: a case report. *J Neurol Neurosurg Psychiatry*. 1987; **50**: 1708–9.

Tanaka Y, Yamadori A, Mori E. Pure word deafness following bilateral lesions. A psychophysical analysis. *Brain*. 1987; **110**: 381–403.

**Cross References**

Agnosia; Amusia; Aphasia; Auditory agnosia

## Pursuit

Pursuit, or smooth pursuit, eye movements hold the image of a moving target on the fovea, or during linear self motion, *i.e.* they stabilize the gaze. This is dependent upon vestibulo-ocular reflexes and visually-mediated reflexes. Impaired ("broken") pursuit may result from occipital lobe lesions, and may be abolished by bilateral lesions, and may co-exist with some forms of congenital nystagmus.

**References**

Gaymard B, Pierrot-Deseilligny C. Neurology of saccades and smooth pursuit. *Curr Opin Neurol*. 1999; **12**: 13–9.

Leigh RJ, Zee DS. The neurology of eye movements. 4th ed. New York: Oxford University Press; 2006. p. 188–240.

**Cross References**

Nystamgus; Saccades; Saccadic intrusion, Saccadic pursuit

**Pyramidal Decussation Syndrome**

Pyramidal decussation syndrome is a rare crossed hemiplegia syndrome, with weakness of one arm and the contralateral leg (hemiplegia cruciata) without involvement of the face, due to a lesion within the pyramid below the decussation of corticiospinal fibres destined for the arm but above that for fibres destined for the leg.

**Cross Reference**

Hemiplegia cruciata

**Pyramidal Signs, Pyramidal Syndrome, Pyramidal Weakness**

- see HEMIPARESIS; UPPER MOTOR NEURONE (UMN) SYNDROME; WEAKNESS