## Lagophthalmos

Lagophthalmos is an inability to close the eyelid in a peripheral facial (VII) nerve palsy, leaving the palpebral fissure partially open. Exposure of the corneal surface increases the risk of keratitis, ulceration, and potentially visual loss.

A similar phenomenon may be observed with aberrant regeneration of the oculomotor (III) nerve, thought to be due to co-contraction of the levator palpebrae superioris and superior rectus muscles during Bell's phenomenon.

### Reference

Vasquez LM, Medel R. Lagophthalmos after facial palsy: current therapeutic options. *Ophthal Res.* 2014; **52**: 165–9.

### **Cross References**

Bell's palsy; Bell's phenomenon, Bell's sign; Facial paresis, Facial weakness

### Lambert's Sign

Lambert's sign is the gradual increase in force over a few seconds when a patient with Lambert-Eaton myasthenic syndrome is asked to squeeze the examiner's hand as hard as possible, reflecting increased power with sustained exercise (post-tetanic potentiation). This may also be known as augmentation.

### Cross References

Augmentation; Facilitation

### Lasègue's Sign

Lasègue's sign is pain along the course of the sciatic nerve induced by exerting traction on the nerve, which is achieved by flexing the thigh at the hip while the leg is extended at the knee ("straight leg raising"). This is similar to the manoeuvre used in Kernig's sign (gradual extension of knee with thigh flexed at the hip). Both indicate irritation of the lower lumbosacral nerve roots and/or meninges. The test is said to have good sensitivity but poor specificity for herniated discs (0.91 and 0.26 in one meta-analysis). The test is non-specific with respect to aetiology, as it may be positive not only with disc protrusion but also with intraspinal tumour or inflammatory radiculopathy.

Various modifications of Lasègue's sign have been described. Pain may be aggravated or elicited sooner using Bragard's test, which is dorsiflexing the foot while raising the leg thus increasing sciatic nerve stretch, or Neri's test, flexing the neck to bring the head on to the chest, indicating dural irritation. Crossed straight leg raising, when the complaint of pain on the affected side occurs when raising the contralateral leg, is said to be less sensitive but highly specific. The femoral nerve stretch test, or "reverse straight leg raising", may detect L3 root or femoral nerve irritation by exerting traction on the femoral nerve.

#### References

Cook C, Cleland J, Huijbregts P. Creation and critique of studies of diagnostic accuracy: use of the STARD and QUADAS methodological quality assessment tools. *J Man Manip Ther.* 2007; **15**: 93–102 [at 96].

Dalfardi B, Mahmoudi Nezhad GS. Ernest-Charles Lasègue (1816–1883). J Neurol. 2014; 261: 2247–8.

Deville WL, van der Windt DA, Dzaferagic A, Bezemer PD, Bouter LM. The test of Lasègue: systematic review of the accuracy in diagnosing herniated discs. *Spine*. 2000; **25**: 1140–7.

#### Cross References

Femoral nerve stretch test; Kernig's sign

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# Lateral Medullary Syndrome

The lateral medullary syndrome (or Wallenberg's syndrome, after the neurologist who described it in 1895) results from damage (usually infarction) of the posterolateral medulla with or without involvement of the inferior cerebellum, producing the following clinical features:

- Nausea, vomiting, vertigo, oscillopsia (involvement of vestibular nuclei).
- Contralateral hypoalgesia, thermoanaesthesia (spinothalamic tract).
- Ipsilateral facial hypoalgesia, thermoanaesthesia, + facial pain (trigeminal spinal nucleus and tract).
- Horner's syndrome (descending sympathetic tract), ± ipsilateral hypohidrosis of the body.
- Ipsilateral ataxia of limbs (olivocerebellar/spinocerebellar fibres, inferior cerebellum).
- Dysphagia, dysphonia, impaired gag reflex.
- ± eye movement disorders, including nystagmus, abnormalities of ocular alignment (skew deviation, ocular tilt reaction, environmental tilt), smooth pursuit and gaze holding, and saccades (lateropulsion, ipsipulsion).
- ± hiccups (singultus); loss of sneezing.

Infarction due to vertebral artery occlusion (occasionally posterior inferior cerebellar artery) or dissection is the most common cause of lateral medullary syndrome, although tumour, demyelination, and trauma are also recognised causes.

## References

Fisher CM, Karnes W, Kubik C. Lateral medullary infarction: the pattern of vascular occlusion. *J Neuropathol Exp Neurol.* 1961; **20**: 103–13.

Sacco RL, Freddo L, Bello JA, Odel JG, Onesti ST, Mohr JP. Wallenberg's lateral medullary syndrome. Clinical-magnetic resonance imaging correlations. *Arch Neurol.* 1993; **50**: 609–14. **Cross References** 

Anaesthesia; Dysphagia; Dysphonia; Environmental tilt; Gag reflex; Hemiataxia; Hiccups; Horner's syndrome; Hypoalgesia; Hypohidrosis; Lateropulsion; Medial medullary syndrome; Nystagmus; Ocular tilt reaction; Oscillopsia; Saccades; Skew deviation; Sneezing; Vertigo

# Lateral Rectus Palsy

- see ABDUCENS (VI) NERVE PALSY

# Laterocollis

Laterocollis is a lateral head tilt; this may be seen in 10–15% of patients with torticollis. Cross Reference

Torticollis

# Lateropulsion

Lateropulsion or ipsipulsion is literally pulling to one side. The term may be used to describe ipsilateral axial lateropulsion after cerebellar infarcts preventing patients from standing upright, causing them to lean to towards the opposite side. Lateral medullary syndrome may be associated with lateropulsion of the eye toward the involved medulla, and there may also be lateropulsion of voluntary saccadic eye movements (hypermetric to the side of the lesion, hypometric towards the opposite side).

# Laughter

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- see AUTOMATISM; PATHOLOGICAL CRYING, PATHOLOGICAL LAUGHTER
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# Lazarus Sign

Various spontaneous and reflex movements are described in brain death, the most dramatic of which has been labelled the Lazarus sign, after Lazarus, raised from the dead by Christ (John 11:1–44). This spinal reflex manifests as flexion of the arms at the elbow, adduction of the shoulders, lifting of the arms, dystonic posturing of the hands and crossing of the hands.

#### Reference

Bueri JA, Saposnik G, Mauriño J, Saizar R, Garretto NS. Lazarus' sign in brain death. *Mov Disord*. 2000; **15**: 583–6. **Cross Reference** Spinal mass reflex

Leadpipe Rigidity - see RIGIDITY

#### Leucocoria

Leucocoria is a white pupillary reflex, in contrast to the normal red reflex. Causes include retinoblastoma, retinal detachment, toxocara infection, congenital cataract, and benign retinal hypopigmentation.

#### Levator Inhibition

- see EYELID APRAXIA

## Levitation

Spontaneous levitation may be displayed by an alien limb, more usually an arm than a leg, indicative of parietal lobe pathology. It is most often seen in corticobasal (ganglionic) degeneration, but a few cases with pathologically confirmed progressive supranuclear palsy have been reported. This has sometimes been called the posterior variant of alien limb.

# References

Barclay CL, Bergeron C, Lang AE. Arm levitation in progressive supranuclear palsy. *Neurology*. 1999; **52**: 879–82.

Brunt ER, van Weerden TW, Pruim J, Lakke JW. Unique myoclonic pattern in corticobasal degeneration. *Mov Disord*. 1995; **10**: 132–42.

## **Cross Reference**

Alien hand, Alien limb

## Lhermitte's Sign

Lhermitte's sign, or the "barber's chair syndrome", is a painless but unpleasant tingling or electric shock-like sensation in the back and spreading instantaneously down the arms and legs following neck flexion (active or passive). It is associated with pathology within the cervical spinal cord. Although most commonly encountered (and originally described in) multiple sclerosis, it is not pathognomonic of demyelination, and has been described with other local pathologies such as:

- subacute combined degeneration of the cord (vitamin  $B_{12}$  deficiency); nitrous oxide (N<sub>2</sub>O) exposure.
- traumatic or compressive cervical myelopathy (*e.g.* cervical spondylotic myelopathy).
- epidural/subdural/intraparenchymal tumour.
- radiation myelitis.
- pyridoxine toxicity.
- inflammation, e.g. systemic lupus erythematosus, Behçet's disease.
- cervical herpes zoster myelitis.
- cavernous angioma of the cervical cord.

Pathophysiologically, this movement-induced symptom may reflect the exquisite mechanosensitivity of axons which are demyelinated, or damaged in some other way.

A "motor equivalent" of Lhermitte's sign, McArdle's sign, has been described, as has "reverse Lhermitte's sign", a label applied either to the aformentioned symptoms occurring on neck extension, or in which neck flexion induces electrical shock-like sensation travelling from the feet upward.

# References

Lhermitte J, Bollack J, Nicolas M. Les douleurs à type de décharge electrique consécutives à la flexion céphalique dans la sclérose en plaques: un case de forme sensitive de la sclérose multiple. *Rev Neurol (Paris)*. 1924; **39**: 56–62.

Pearce JMS. Lhermitte's sign. In: Fragments of neurological history. London: Imperial College Press; 2003. p. 367–9.

Smith KJ. Conduction properties of central demyelinated axons: the generation of symptoms in demyelinating disease. In: Bostock H, Kirkwood PA, Pullen AH, editors. The neurobiology of disease: contributions from neuroscience to clinical neurology. Cambridge: Cambridge University Press; 1996. p. 95–117.

# Cross References

McArdle's sign; Myelopathy

# Lid Lag

Lid lag is present if a band of sclera is visible between the upper eyelid and the corneal limbus on attempted downgaze (*cf.* lid retraction), seen for example in thyroid eye disease (von Graefe's sign), progressive supranuclear palsy (Steele-Richardson-Olszewski syndrome), and Guillain-Barré syndrome.

# Cross References

Lid retraction; Von Graefe's sign

# Lid Retraction

Lid (eyelid) retraction is present if a band of sclera is visible between the upper eyelid and the corneal limbus in the primary position (*cf.* lid lag). This should be distinguished from contralateral ptosis. Recognised causes of lid retraction include:

- Overactivity of levator palpebrae superioris:
  - Dorsal mesencephalic lesion (Collier's sign).
  - Opposite to unilateral ptosis, *e.g.* in myasthenia gravis; retracted lid may fall when ptotic lid raised (enhanced ptosis); frontalis overactivity usually evident.
  - Paradoxical lid retraction with jaw movement (jaw winking, Marcus Gunn phenomenon).
- Overactivity of Müller's muscle:
  - irritative oculosympathetic lesions (Claude-Bernard syndrome).
- Contracture of the levator muscle:
  - Hyperthyroidism, Graves' ophthalmopathy (Dalrymple's sign): may be associated lid lag.
  - Myotonic syndromes.
  - Aberrant oculomotor (III) nerve regeneration (pseudo-von Graefe's sign).
- Central lesion:
  - progressive supranuclear palsy.
  - dementia with Lewy bodies.
  - Parkinson's disease.
- Cicatricial retraction of the lid, *e.g.* following trauma.
- Hepatic disease (Summerskill's sign).
- Guillain-Barré syndrome.

Lower lid retraction may be congenital, or a sign of proptosis. Ectropion may also be seen with lower lid tumour or chalazion, trauma with scarring, and ageing.

# Reference

Onofrj M, Monaco D, Bonanni L, et al. Eyelid retraction in dementia with Lewy bodies and Parkinson's disease. *J Neurol*. 2011; **258**: 1542–4.

## **Cross References**

Collier's sign; Contracture; Dalrymple's sign; Jaw winking; Lid lag; Proptosis; Pseudo-von Graefe's sign; Ptosis; Stellwag's sign; Sunset sign

## Light-Near Pupillary Dissociation

Light-near pupillary dissociation refers to the loss of pupillary light reflexes, whilst the convergence-accommodation reaction is preserved (see Pupillary Reflexes). This dissociation may be seen in a variety of clinical circumstances:

- Argyll Robertson pupil: small irregular pupils with reduced reaction to light, typically seen in neurosyphilis; the absence of miosis and/or pupillary irregularity has been referred to as pseudo-Argyll Robertson pupil, which may occur in neurosarcoidosis, diabetes mellitus, and aberrant regeneration of the oculomotor (III) nerve.
- Holmes-Adie pupil: dilated pupil showing strong but slow reaction to accommodation but minimal reaction to light (tonic > phasic).
- Parinaud's syndrome (dorsal rostral midbrain syndrome): due to a lesion at the level of the posterior commissure, and characterized by vertical gaze palsy, lid retraction (Collier's sign) or ptosis, and large regular pupils responding to accommodation but not light.

### 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; Collier's sign; Holmes-Adie pupil, Holmes-Adie syndrome; Lid retraction; Parinaud's syndrome; Pseudo-Argyll Robertson pupil; Pupillary reflexes

### Light Reflex

- see PUPILLARY REFLEXES

## Locked-in Syndrome

The locked-in syndrome results from de-efferentiation, such that a patient is awake, self-ventilating and alert, but unable to speak or move; vertical eye movements and blinking are usually preserved, affording a channel for simple (yes/no) communication.

The most common cause of the locked-in syndrome is basilar artery thrombosis causing ventral pontine infarction (both pathological laughter and pathological crying have on occasion been reported to herald this event). Other pathologies include pontine haemorrhage and central pontine myelinolysis. Bilateral ventral midbrain and internal capsule infarcts can produce a similar picture. Generally this is irreversible, although recovery has on occasion been recorded.

The locked-in syndrome may be mistaken for abulia, akinetic mutism, coma, and catatonia.

## References

Bauby J-D. The diving-bell and the butterfly. London: Fourth Estate; 1997.

Laureys S, Pellas F, van Eeckhout P, et al. The locked-in syndrome: what is it like to be conscious but paralyzed and voiceless? *Prog Brain Res.* 2005; **150**: 495–511.

Smith E, Delargy M. Locked-in syndrome. BMJ. 2005; 330: 406–9.

#### **Cross References**

Abulia; Akinetic mutism; Blinking; Catatonia; Coma; Pathological crying, Pathological laughter; Vegetative states

## Lockjaw

- see TRISMUS

L

### Logoclonia

Logoclonia is the tendency for a patient to repeat the final syllable of a word when speaking; hence it is one of the reiterative speech disorders (*cf.* echolalia, palilalia). Liepmann apparently coined this term in 1905 to indicate "continuous perseveration". It may be described as the festinating repetition of individual phonemes.

Logoclonia is an indicator of bilateral brain injury, usually involving subcortical structures, and may be seen in the late stages of dementia of Alzheimer type (but not in semantic dementia).

### Reference

Kertesz A. Language in Alzheimer's disease. In: Morris R, Becker JT, editors. Cognitive neuropsychology of Alzheimer's disease. 2nd ed. Oxford: Oxford University Press; 2004. p. 197–218 [at 202].

## Cross References

Echolalia; Festination, Festinant gait; Palilalia; Perseveration

### Logopenia

Logopenia denotes a language disorder characterised by word finding pauses, but with relatively preserved phrase length and syntactically complete language, but with impaired repetition of phrases and sentences. A logopenic variant of primary progressive aphasia has been delineated in recent years, with Alzheimer-type pathology as the most common neuropathological substrate, hence this is one of the acknowledged variant forms of Alzheimer's disease.

### References

Dubois B, Feldman HH, Jacova C et al. Advancing research diagnostic criteria for Alzheimer's disease: the IWG-2 criteria. *Lancet Neurol.* 2014; **13**: 614–29 [Erratum *Lancet Neurol.* 2014; **13**: 757].

Gorno-Tempini ML, Hillis AE, Weintraub S, et al. Classification of primary progressive aphasia and its variants. *Neurology*. 2011; **76**: 1006–14.

#### **Cross Reference**

Aphasia

## Logorrhoea

Logorrhoea is literally a flow of speech, or pressure of speech, denoting an excessive verbal output, an abnormal number of words produced during each utterance. Content is often irrelevant, disconnected and difficult to interpret. The term may be used to describe the output in the Wernicke/posterior/sensory type of aphasia, or an output which superficially resembles Wernicke aphasia but in which syntax and morphology are intact, rhythm and articulation are usually normal, and paraphasias and neologisms are few. Moreover comprehension is better than anticipated in the Wernicke type of aphasia. Patients may be unaware of their impaired output (anosognosia) due to a failure of self-monitoring.

Logorrhoea may be observed in subcortical (thalamic) aphasia, usually following recovery from lesions (usually haemorrhage) to the anterolateral nuclei. Similar speech output may be observed in psychiatric disorders such as mania and schizophrenia (schizophasia).

## Reference

Damasio AR. Aphasia. N Engl J Med. 1992; **326**: 531–9.

# Cross References

Aphasia; Delirium; Echolalia; Jargon aphasia; Schizophasia; Wernicke's aphasia

## Lombard Effect

The Lombard effect is the reflex increase in spoken sound volume in a noisy environment, named after Etienne Lombard, a French ENT surgeon who first described the phenomenon in the early 1900s. Something similar may be noted when attempting conversation with those wearing headphones.

# Reference Zollinger SA, Brumm H. The Lombard effect. *Curr Biol*. 2011; **21**: R614–5.

Long Tract Signs - see UPPER MOTOR NEURONE (UMN) SYNDROME

## "Looking Glass Syndrome"

- see MIRROR AGNOSIA

# Lower Motor Neurone (LMN) Syndrome

A lower motor neurone (LMN) syndrome constitutes a constellation of motor signs resulting from damage to lower motor neurone pathways, *i.e.* from anterior horn cell distally, encompassing the motor roots, nerve plexuses, peripheral nerves, and neuromuscular junction. Following the standard order of neurological examination of the motor system, the signs include:

• Appearance:

muscle wasting; fasciculations (or "fibrillations") may be observed or induced, particularly if the pathology is at the level of the anterior horn cell.

- Tone:
  - reduced tone (flaccidity, hypotonus), although this may simply reflect weakness.
- Power:

weakness, often marked; depending on the precise pathological process, weakness often affects both flexor and extensor muscles equally (although this is not always the case).

• Co-ordination:

depending on the degree of weakness, it may not be possible to comment on the integrity or otherwise of co-ordination in LMN syndromes; in a pure LMN syndrome co-ordination will be normal.

Reflexes:

depressed (hyporeflexia) or absent (areflexia); plantar responses are flexor.

It is often possible to draw a clinical distinction between motor symptoms resulting from lower or upper motor neurone pathology and hence to formulate a differential diagnosis and direct investigations accordingly. Sensory features may also be present in LMN syndromes if the pathology affects sensory as well as motor roots, or both motor and sensory fibres in peripheral nerves.

# Cross References

Areflexia; Fasciculation; Fibrillation; Flaccidity; Hyporeflexia; Hypotonia, Hypotonus; Neuropathy; Reflexes; Upper motor neurone (UMN) syndrome; Weakness

## Luria Test

- see FIST-EDGE-PALM TEST