Babinski’s Sign (1)

Babinski’s sign is a polysynaptic cutaneous reflex consisting of an extensor movement (dorsiflexion) of the big toe on eliciting the plantar response, due to contraction of extensor hallucis longus. There may be in addition fanning (abduction) of the other toes (fan sign; signe de l’éventail) but this is neither necessary nor sufficient for Babinski’s sign to be judged present. There may be simultaneous contraction of other limb flexor muscles, consistent with the notion that Babinski’s sign forms part of a flexion synergy (withdrawal) of the leg. The use of the term ‘negative Babinski sign’ to indicate the normal finding of a down-going (flexor; plantar flexion) big toe is incorrect, ‘flexor plantar response’ being the appropriate description. The plantar response is most commonly performed by stroking the sole of the foot, although many other variants are described (e.g. Chaddock’s sign, Gordon’s sign, Oppenheim’s sign).

Babinski’s sign is a normal finding in infants with immature (unmyelinated) corticospinal tracts; persistence beyond 3 years of age, or re-emergence in adult life, is pathological. In this context, Babinski’s sign is considered a reliable (‘hard’) sign of corticospinal (pyramidal) tract dysfunction (upper motor neurone pathology) and may coexist with other signs of upper motor neurone dysfunction (e.g. weakness in a so-called pyramidal distribution, spasticity, hyperreflexia). However, if weakness of extensor hallucis longus is one of the features of upper motor neurone dysfunction, or from any other cause, Babinski’s sign may be unexpectedly absent although anticipated on clinical grounds. Other causes of Babinski’s sign include hepatic coma, postepileptic seizure, deep sleep following prolonged induced wakefulness, and cataleptic attack, hence it is not necessarily a consequence of a permanent and irreversible lesion of the pyramidal tracts.

In the presence of extrapyramidal signs, it is important to distinguish Babinski’s sign, a ‘pyramidal sign’, from a striatal toe (spontaneous up going plantar).

References

Cross References
Chaddock’s sign; Gordon’s sign; Hyperreflexia; Oppenheim’s sign; Parkinsonism; Plantar response; Spasticity; Striatal toe; Upper motor neurone (UMN) syndrome; Weakness
Babinski’s Sign (2)
Babinski (1905) described the paradoxical elevation of the eyebrow in hemifacial spasm as orbicularis oris contracts and the eye closes, a synkinesis which is not reproducible by will. This observation indicated to Babinski the peripheral (facial nerve) origin of hemifacial spasm. It may assist in differentiating hemifacial spasm from other craniofacial movement disorders.

Reference

Cross Reference
Hemifacial spasm

Babinski’s Trunk–Thigh Test
Babinski’s trunk–thigh test, also known as the ‘rising sign’, is suggested to be of use in distinguishing organic from functional paraplegia and hemiplegia (the abductor sign may also be of use in the former case, Hoover’s sign in the latter). The recumbent patient is asked to sit up with the arms folded on the front of the chest. In organic hemiplegia there is involuntary flexion of the paretic leg, which may automatically rise higher than the normal leg; in paraplegia both legs are involuntarily raised. In functional paraplegic weakness neither leg is raised, and in functional hemiplegia only the normal leg is raised.

Reference

Cross References
Abductor sign; Functional weakness and sensory disturbance; Hemiplegia; Hoover’s sign; Paraplegia

Bag of Worms
- see MYOKYMIA

Balaclava Helmet
A pattern of facial sensory loss resembling in distribution a balaclava helmet, involving the outer parts of the face but sparing the nose and mouth, may be seen with central brainstem lesions such as syringobulbia which progress upwards from the neck, such that the lowermost part of the spinal nucleus of the trigeminal nerve which serves the outer part of the face is involved whilst the upper part of the nucleus which serves the central part of the face is spared. This pattern of facial sensory impairment may also be known as onion peel or onion skin.

Cross Reference
Onion peel, Onion skin

Balint’s Syndrome
Balint’s syndrome, first described by a Hungarian neurologist in 1909, consists of:

- Simultanagnosia (q.v.; dorsal type):
  A constriction of visual attention, such that the patient is aware of only one object at a time; visual acuity is preserved, and patients can
Ballism, Ballismus

recognize single objects placed directly in front of them; they are unable to read or distinguish overlapping figures.

- **Spatial disorientation:**
  Loss of spatial reference and memory, leaving the patient 'lost in space'.

- **Disorders of oculomotor function:**
  Specifically, visually guided eye movements (fixation, pursuit, saccades); Balint's 'psychic paralysis of gaze', or 'sticky fixation', refers to an inability to direct voluntary eye movements to visual targets, despite a full range of eye movements; this has also been characterized as a form of oculomotor apraxia. Accurate eye movements may be programmed by sound or touch. Loss of spontaneous blinking has also been reported.

- **Optic ataxia:**
  A failure to grasp or touch an object under visual guidance.

Not all elements may be present; there may also be coexisting visual field defects, hemispatial neglect, visual agnosia, or prosopagnosia.

Balint's syndrome results from bilateral lesions of the parieto-occipital junction causing a functional disconnection between higher-order visual cortical regions and the frontal eye fields, with sparing of the primary visual cortex. Brain imaging, either structural (CT, MRI) or functional (SPECT, PET), may demonstrate this bilateral damage, which is usually of vascular origin, for example, due to watershed or border zone ischaemia or top-of-the-basilar syndrome. Balint syndrome has also been reported as a migrainous phenomenon, following traumatic brain injury and in association with Alzheimer's disease, brain tumour (butterfly glioma), radiation necrosis, progressive multifocal leucoencephalopathy, Marchiafava–Bignami disease with pathology affecting the corpus callosum, and X-linked adrenoleucoencephalodystrophy.

**References**
Husein M, Stein J. Rezso Balint and his most celebrated case. *Archives of Neurology* 1988; 45: 89–93.

**Cross References**
Apraxia; Blinking; Ocular apraxia; Optic ataxia; Simultanagnosia

**Ballism, Ballismus**

Ballism or ballismus is a hyperkinetic involuntary movement disorder characterized by wild, flinging, throwing movements of a limb. These movements most usually involve one-half of the body (*hemiballismus*), although they may sometimes involve a single extremity (*monoballismus*) or both halves of the body (*paraballismus*). The movements are often continuous during wakefulness but cease during sleep. Hemiballismus may be associated with limb hypotonia. Clinical and pathophysiological studies suggest that ballism is a severe form of chorea. It is most commonly associated with lesions of the contralateral subthalamic nucleus.
Bathing Suit Sensory Loss
- see SUSPENDED SENSORY LOSS

Battle’s Sign
Battle’s sign is a haematoma overlying the mastoid process, which indicates an underlying basilar skull fracture extending into the mastoid portion of the temporal bone. It appears 48–72 h after the trauma which causes the fracture.

Beevor’s Sign
Beevor’s sign is an upward movement of the umbilicus in a supine patient attempting either to flex the head onto the chest against resistance (e.g. the examiner’s hand) or performing a sit-up. It indicates a lesion causing rectus abdominis muscle weakness below the umbilicus. This may occur with a spinal lesion (e.g. tumour, syringomyelia) between T10 and T12 causing isolated weakness of the lower part of the muscle, or myopathies affecting abdominal muscles, particularly facioscapulohumeral muscular dystrophy. Lower cutaneous abdominal reflexes are also absent, having the same localizing value.

   Downward movement of the umbilicus (‘inverted Beevor’s sign’) due to weakness of the upper part of rectus abdominis is less often seen.

References

Cross Reference
Abdominal reflexes

Belle Indifférence
La belle indifférence refers to a patient’s seeming lack of concern in the presence of serious symptoms. This was first defined in the context of ‘hysteria’, along with exaggerated emotional reactions, what might now be termed functional or somatoform illness. However, the sign is a poor discriminator against ‘organic’ illness. Some patients’ coping style is to make light of serious symptoms; they might be labelled stoical.

   Patients with neuropathological lesions may also demonstrate a lack of concern for their disabilities, either due to a disorder of body schema (anosodiaphoria) or due to incongruence of mood (typically in frontal lobe syndromes, sometimes seen in multiple sclerosis).

Reference
Bell's Palsy
Bell's palsy is an idiopathic peripheral (lower motor neurone) facial weakness (prosopoplegia). It is thought to result from viral inflammation of the facial (VII) nerve. Other causes of lower motor neurone facial paresis may need to be excluded before a diagnosis of Bell's palsy can be made.

In the majority of patients with Bell's palsy (idiopathic facial paresis), spontaneous recovery occurs over 3 weeks to 2 months. Poorer prognosis is associated with older age (over 40 years) and if no recovery is seen within 4 weeks of onset. Meta-analyses suggest that steroids are associated with better outcome than no treatment, but that acyclovir alone has no benefit.

References

Bell's Phenomenon, Bell's Sign
Bell's phenomenon or sign is reflex upward, and slightly outward, deviation of the eyes in response to forced closure, or attempted closure, of the eyelids. This is a synkinesis of central origin involving superior rectus and inferior oblique muscles. It may be very evident in a patient with Bell's palsy (idiopathic facial nerve paralysis) attempting to close the paretic eyelid. The reflex indicates intact nuclear and infranuclear mechanisms of upward gaze, and hence that any defect of upgaze is supranuclear. However, in making this interpretation it should be remembered that perhaps 10–15% of the normal population do not show a Bell’s phenomenon.

Bell's phenomenon is usually absent in progressive supranuclear palsy and is only sometimes spared in Parinaud’s syndrome.

Reference

Cross References
Bell's palsy; Gaze palsy; Parinaud’s syndrome; Supranuclear gaze palsy; Synkinesia, Synkinesis

Benediction Hand
Median nerve lesions in the axilla or upper arm cause weakness in all median nerve innervated muscles, including flexor digitorum profundus. Thus
on attempting to make a fist, impaired flexion of the index and middle fingers, complete and partial, respectively, but with normal ring and little finger flexion (ulnar nerve mediated) results in a hand posture likened to that of a priest saying benediction (also sometimes known as Benedictine hand or orator’s hand).

**Cross References**
Claw hand; Simian hand

**Bent Spine Syndrome**
- see CAMPTOCORMIA

**Bielschowsky’s Sign, Bielschowsky’s Test**
Bielschowsky’s sign is head tilt towards the shoulder, typically towards the side contralateral to a trochlear (IV) nerve palsy. The intorsion of the unaffected eye brought about by the head tilt compensates for the double vision caused by the unopposed extorsion of the affected eye. Very occasionally, head tilt is paradoxical, i.e. towards the involved side: presumably the greater separation of images thus produced allows one of them to be ignored.

Bielschowsky’s (head tilt) test consists of the examiner tipping the patient’s head from shoulder to shoulder to see if this improves or exacerbates double vision, as will be the case when the head is, respectively, tilted away from or towards the affected side in a unilateral trochlear (IV) nerve lesion. The test is usually negative in a skew deviation causing vertical divergence of the eyes. This test may also be used as part of the assessment of vertical diplopia to see whether hypertropia changes with head tilt to left or right; increased hypertropia on left head tilt suggests a weak intortor of the left eye (superior rectus); increased hypertropia on right head tilt suggests a weak intortor of the right eye (superior oblique).

**Cross References**
Diplopia; Hypertropia; Skew deviation

**Binasal Hemianopia**
Of the hemianopic defects, binasal hemianopia, suggesting lateral compression of the chiasm, is less common than bitemporal hemianopia. Various causes are recorded including syphilis, glaucoma, drusen, and chronically raised intracranial pressure.

**Reference**

**Cross Reference**
Hemianopia

**Bitemporal Hemianopia**
Bitemporal hemianopia due to chiasmal compression, for example, by a pituitary lesion or craniopharyngioma, is probably the most common cause of a heteronymous hemianopia. Conditions mimicking bitemporal hemianopia include congenitally tilted discs, nasal sector retinitis pigmentosa, and papilloedema with greatly enlarged blind spots.
Cross References
Hemianopia; Visual field defects

Blepharoptosis
- see PTOSIS

Blepharospasm
Blepharospasm is a focal dystonia of the orbicularis oculi resulting in repeated involuntary forced eyelid closure, with failure of voluntary eye opening. Usually bilateral in origin, it may be sufficiently severe to result in functional blindness. The condition typically begins in the sixth decade of life and is more common in women than in men. Blepharospasm may occur in isolation (‘benign essential blepharospasm’), or in combination with other involuntary movements which may be dystonic (orobuccolingual dystonia or Meige syndrome; limb dystonia) or dyspraxic (eyelid apraxia), or in association with another neurological disorder such as Parkinson’s disease. Other examples of ‘secondary blepharospasm’ include drug therapy (neuroleptics, levodopa) and lesions of the brainstem and more rarely cerebellum and striatum. Like other forms of dystonia, blepharospasm may be relieved by sensory tricks (geste antagoniste), such as talking, yawning, singing, humming, or touching the eyelid. This feature is helpful in diagnosis. Blepharospasm may be aggravated by reading, watching television, and exposure to wind or bright light.

Blepharospasm is usually idiopathic but may be associated with lesions (usually infarction) of the rostral brainstem, diencephalon, and striatum; it has been occasionally reported with thalamic lesions. The pathophysiological mechanisms underlying blepharospasm are not understood, but may reflect dopaminergic pathway disruption causing disinhibition of brainstem reflexes.

Local injections of botulinum toxin into orbicularis oculi are the treatment of choice, the majority of patients deriving benefit and requesting further injection. Failure to respond to botulinum toxin may be due to concurrent eyelid apraxia or dopaminergic therapy with levodopa.

References

Cross References
Blinking; Dystonia; Eyelid apraxia; Gaping; Geste antagoniste; Yawning

Blindsight
Blindsight describes a rare phenomenon in which patients with bilateral occipital lobe damage affecting the primary visual cortex are nonetheless able to discriminate certain visual events within their ‘blind’ fields, but are not aware of their ability to do so.

Reference
Cross Reference
Scotoma

Blind Spot
The blind spot is defined anatomically as the point on the retina at which axons from the retinal ganglion cells enter the optic nerve; since this area is devoid of photoreceptors there is a physiological blind spot. This area may be mapped clinically by confrontation with the examiner’s blind spot or mechanically. Minor enlargement of the blind spot is difficult to identify clinically, formal perimetry is needed in this situation.

Enlargement of the blind spot (peripapillary scotoma) is observed with raised intracranial pressure causing papilloedema: this may be helpful in differentiating papilloedema from other causes of disc swelling such as optic neuritis, in which a central scotoma is the most common field defect. Enlargement of the blind spot may also be a feature of peripapillary retinal disorders including big blind spot syndrome.

Cross References
Disc swelling; Papilloedema; Scotoma

Blinking
Involuntary blinking rate is decreased in idiopathic Parkinson’s disease (and may be improved by dopaminergic therapy) and in progressive supranuclear palsy (Steele–Richardson–Olszewski syndrome) where the rate may be <5/min. In contrast, blink rate is normal in multiple system atrophy and dopa-responsive dystonia, and increased in schizophrenia and postencephalitic parkinsonism. These disparate observations are not easily reconciled with the suggestion that blinking might be a marker of central dopaminergic activity.

Loss of spontaneous blinking has been reported in Balint’s syndrome. In patients with impaired consciousness, the presence of involuntary blinking implies an intact pontine reticular formation; absence suggests structural or metabolic dysfunction of the reticular formation. Blinking decreases in coma. Functional disorders may be accompanied by an increase in blinking.

Reference

Cross References
Balint’s syndrome; Blink reflex; Coma; Corneal reflex; Parkinsonism; Sighing; Yawning

Blink Reflex
The blink reflex consists of bilateral reflex contraction of the orbicularis oculi muscles. This may be induced by:

- **Mechanical stimulus:**
  - Examples include percussion over the supraorbital ridge (glabellar tap reflex, Myerson’s sign, nasopalpebral reflex): this quickly habituates with repetitive stimulation in normal individuals; touching the cornea (corneal reflex); stroking the eyelashes in unconscious patients with closed eyes (‘eyelash reflex’).
- Visual stimulus:
  Sudden visual stimulus approaching the eyes (menace reflex, threat reflex, visuopalpebral reflex): the stimulus should be unexpected since the reflex can be voluntarily suppressed; failure to respond to a stimulus moving into the temporal field of vision may indicate a hemianopic field defect in patients unable to comply with standard confrontation visual field testing. Care should be taken to avoid generating air currents with the hand movement as this may stimulate the corneal reflex which may simulate the visuopalpebral reflex. It is probable that this reflex requires cortical processing; it is lost in persistent vegetative states. Loss of this reflex may occur in Balint’s syndrome, ascribed to inability to recognize the nearness of the threatening object.

- Acoustic stimulus:
  Sudden loud sounds (acousticopalpebral reflex).

The final common (efferent) pathway for these responses is the facial nerve nucleus and facial (VII) nerve, the afferent limbs being the trigeminal (V), optic (II), and auditory (VIII) nerves, respectively.

Electrophysiological study of the blink reflex may demonstrate peripheral or central lesions of the trigeminal (V) nerve or facial (VII) nerve (afferent and efferent pathways, respectively). It has been reported that in the evaluation of sensory neuronopathy the finding of an abnormal blink reflex favours a non-paraneoplastic aetiology, since the blink reflex is normal in paraneoplastic sensory neuronopathies.

References

Cross References
Balint’s syndrome; Blinking; Corneal reflex; Glabellar tap reflex

Body Part as Object
In this phenomenon, apraxic patients use a body part when asked to pantomime certain actions, such as using the palm when asked to demonstrate the use of a hair brush or comb, or fingers when asked to demonstrate use of scissors or a toothbrush.

References

Cross References
Apraxia; Parapraxia, Parapraxis
**Bon-Bon Sign**

Involuntary pushing of the tongue against the inside of the cheek, the ‘bon-bon sign’, is said to be typical of the stereotypic orolingual movements of tardive dyskinesia, along with chewing and smacking of the mouth and lips, and rolling of the tongue in the mouth. These signs may help to distinguish tardive dyskinesia from chorea, although periodic protrusion of the tongue (flycatcher, trombone tongue) is common to both.

**Cross References**

Buccolingual syndrome; Chorea, Choreaathetosis; Trombone tongue

**Bouche de Tapir**

Patients with facioscapulohumeral (FSH) muscular dystrophy have a peculiar and characteristic facies, with puckering of the lips when attempting to whistle. The pouting quality of the mouth, unlike that seen with other types of bilateral (neurogenic) facial weakness, has been likened to the face of the tapir (*Tapirus* sp.).

**Cross Reference**

Facial paresis

**Bovine Cough**

A bovine cough lacks the explosive character of a normal voluntary cough. It may result from injury to the distal part of the vagus nerve, particularly the recurrent laryngeal branches which innervate all the muscles of the larynx (with the exception of cricothyroid) with resultant vocal cord paresis. Because of its longer intrathoracic course, the left recurrent laryngeal nerve is more often involved. A bovine cough may be heard in patients with tumours of the upper lobes of the lung (Pancoast tumour) due to recurrent laryngeal nerve palsy. Bovine cough may also result from any cause of bulbar weakness, such as motor neurone disease, Guillain–Barré syndrome, and bulbar myopathies.

**Reference**


**Cross References**

Bulbar palsy; Diplophonia; *Signe de rideau*

**Bradykinesia**

Bradykinesia is a slowness in the initiation and performance of voluntary movements in the absence of weakness and is one of the typical signs of parkinsonian syndromes, in which situation it is often accompanied by difficulty in the initiation of movement (akinesia, hypokinesia) and reduced amplitude of movement (hypometria) which may increase with rapid repetitive movements (fatigue). It may be overcome by reflexive movements or in moments of intense emotion (*kinesis paradoxica*).

Bradykinesia in parkinsonian syndromes reflects dopamine depletion in the basal ganglia. It may be improved by levodopa and dopaminergic agonists, less so by anticholinergic agents.
Slowness of voluntary movement may also be seen with psychomotor retardation, frontal lobe lesions producing abulia, and in the condition of obsessive slowness.

**Reference**

**Cross References**
Abulia; Akinesia; Fatigue; Hypokinesia; Hypometria; *Kinesis paradoxica*; Parkinsonism; Psychomotor retardation

**Bradylalia**
Bradylalia is slowness of speech, typically seen in the frontal–subcortical types of cognitive impairment, with or without extrapyramidal features, or in depression.

**Cross References**
Palilalia; Tachylalia

**Bradyphrenia**
Bradyphrenia is a slowness of thought, typically seen in the frontal–subcortical types of cognitive impairment, e.g. progressive supranuclear palsy, vascular dementia, and Huntington’s disease. Such patients typically answer questions correctly but with long response times.

**Cross References**
Abulia; Dementia

**Bragard’s Test**
- see LASÈGUE’S SIGN

**Broca’s Aphasia**
Broca’s aphasia is the classic ‘expressive aphasia’, in distinction to the ‘receptive aphasia’ of Wernicke; however, there are problems with this simple classification, since Broca’s aphasics may show comprehension problems with complex material, particularly in relation to syntax. Considering each of the features suggested for the clinical classification of aphasias (see Aphasia), Broca’s aphasia is characterized by:

- **Fluency**: slow, laboured, effortful speech (non-fluent) with phonemic paraphasias, agrammatism, and aprosody; the patient knows what s/he wants to say and usually recognizes the paraphasic errors (i.e. patients can ‘self-monitor’);
- **Comprehension**: comprehension for simple material is preserved, but there may be problems with more complex syntax;
- **Repetition**: impaired;
- **Naming**: impaired (anomia, dysnomia); may be aided by phonemic or contextual cueing (cf. Wernicke’s aphasia);
- **Reading**: alexia with laboured oral reading, especially of function words and verb inflections. Silent reading may also be impaired (deep dyslexia) as reflected by poor text comprehension;
- **Writing**: similarly affected.
Aphemia was the name originally given by Broca to the language disorder subsequently named ‘Broca’s aphasia’. The term alalia was also once used. The terms ‘small Broca’s aphasia’, ‘mini-Broca’s aphasia’, and ‘Broca’s area aphasia’ have been reserved for a more circumscribed clinical and neuroanatomical deficit than Broca’s aphasia, wherein the damage is restricted to Broca’s area or its subjacent white matter. There is a mild and transient aphasia or anomia which may share some of the characteristics of aphemia/phonetic disintegration (i.e. a motor disorder of speech production with preserved comprehension of spoken and written language).

The syndrome of Broca’s aphasia may emerge during recovery from a global aphasia. Broca’s aphasia is sometimes associated with a right hemiparesis, especially affecting the arm and face; there may also be bucco-lingual-facial dyspraxia. Depression may be a concurrent feature.

Classically Broca’s aphasia is associated with a vascular lesion of the third frontal gyrus in the inferior frontal lobe (Broca’s area), but in practice such a circumscribed lesion is seldom seen. More commonly there is infarction in the perisylvian region affecting the insula and operculum (Brodmann areas 44 and 45), which may include underlying white matter and the basal ganglia (territory of the superior branch of the middle cerebral artery).

References


Cross References
Agrammatism; Agraphia; Alalia; Alexia; Aphasia; Aphemia; Aprosodia, Aprosody; Paraphasia; Recurrent utterances; Wernicke’s aphasia

**Brown-Séquard Syndrome**
The Brown-Séquard syndrome is the consequence of anatomical or, more usually, functional hemisection of the spinal cord (spinal hemisection syndrome), producing the following pattern of clinical findings:

- **Motor:**
  - Ipsilaterial spastic weakness, due to corticospinal tract involvement;
  - Segmental lower motor neurone signs at the level of the lesion, due to root and/or anterior horn cell involvement.

- **Sensory:**
  - A dissociated sensory loss, i.e.:
  - Ipsilateral loss of proprioception, due to dorsal column involvement;
  - Contralateral loss of pain and temperature sensation, due to crossed spinothalamic tract involvement.

  Spinal cord lesions producing this syndrome may be either extramedullary (e.g. prolapsed cervical intervertebral disc, extrinsic spinal cord tumour) or intramedullary (e.g. multiple sclerosis, intrinsic spinal cord tumour, myelitis, radiation-induced myelopathy); the former group is said to be the more common cause.
References

Cross References
Dissociated sensory loss; Myelopathy; Proprioception; Spasticity; Weakness

Brudzinski’s (Neck) Sign
Brudzinski described a number of signs, but the one most often used in clinical practice is the neck sign, which is sometimes evident in cases of meningeal irritation, for example, due to meningitis. Passive flexion of the neck to bring the head onto the chest is accompanied by flexion of the thighs and legs. As with nuchal rigidity and Kernig’s sign, Brudzinski’s sign may be absent in elderly or immunosuppressed patients with meningeal irritation.

Reference

Cross References
Kernig’s sign; Meningism; Nuchal rigidity

Brueghel’s Syndrome
Brueghel’s syndrome [NB some texts give ‘Breughel’s’ syndrome] is the name given to a dystonia of the motor trigeminal nerve causing gaping or involuntary opening of the mouth, so named after Brueghel’s painting De Gaper of 1558, thought to illustrate a typical case. Additional features may include paroxysmal hyperpnoea and upbeating nystagmus. Brueghel’s syndrome should be distinguished from other syndromes of cranial dystonia featuring blepharospasm and oromandibular dystonia, better termed Meige’s syndrome.

Reference

Cross References
Blepharospasm; Dystonia

Bruit
Bruits arise from turbulent blood flow causing arterial wall vibrations which are audible at the body surface with the unassisted ear or with a stethoscope (diaphragm rather than bell, better for detecting higher frequency sounds). They are associated with stenotic vessels or with fistulae where there is arteriovenous shunting of blood. Dependent on the clinical indication, various sites may be auscultated: eye for orbital bruit in carotico-cavernous fistula; head for bruit of AV fistula; but probably the most frequently auscultated region is the carotid bifurcation, high up under the angle of the jaw, in individuals thought to have had a transient ischaemic attack or ischaemic stroke. Examination for carotid bruits in asymptomatic individuals is probably best avoided, other than in the clinical trial
setting, since the optimal management of asymptomatic carotid artery stenosis has yet to be fully defined.

Reference

**Brushfield Spots**
Brushfield spots are small grey-white specks of depigmentation that can be seen in the irides of some (90%) patients with Down’s syndrome; they may also occur in normal individuals.

**Bruxism**
Bruxism is forcible grinding or gnashing of the teeth. This is common in children and is said to occur in 5–20% of the population during non-REM sleep (a parasomnia). Masseter hypertrophy may become apparent in persistent grinders. Bruxism may also occur in encephalopathic disorders (e.g. hepatic encephalopathy) and occasionally in disorders of the basal ganglia (multiple system atrophy, basal ganglia infarcts). Dysfunction of efferent and/or afferent thalamic and striatopallidal tracts has been suggested as the neural substrate. If necessary, a rubber gum shield or bite may be worn in the mouth to protect the teeth. Botulinum toxin injections have also been tried.

Reference

**Cross References**
Encephalopathy; Masseter hypertrophy

**Buccofacial Dyspraxia**
- see ORO-FACIAL DYSPRAXIA

**Buccolingual Syndrome**
This is a form of tardive dyskinesia that involves involuntary movements of the facial muscles and protrusion of the tongue.

**Cross References**
‘Bon-bon sign’; Dyskinesia

**Bulbar Palsy**
Bulbar palsy is weakness of bulbar musculature of lower motor neurone origin. This may be differentiated clinically from bulbar weakness of upper motor neurone origin (pseudobulbar palsy).

Clinical features of bulbar palsy include

- Dysarthria of flaccid/nasal type;
- Dysphonia;
- Dysphagia, often with nasal regurgitation;
- Weak (‘bovine’) cough; risk of aspiration;
- +/- wasted, fasciculating tongue;
- +/- absent jaw jerk;
- +/- absent gag reflex.
Bulbar palsy is usually neurogenic. Recognized causes include

- **Brainstem disorders affecting cranial nerve motor nuclei (intrinsic):**
  - Motor neurone disease (which may also cause a pseudobulbar palsy);
  - Poliomyelitis;
  - Glioma;
  - Syringobulbia.

- **Cranial nerve lesions outside the brainstem (there may be associated sensory signs):**
  - Infiltration by carcinoma, granuloma.

- **Neuromuscular junction transmission defect:**
  - Myasthenia gravis.

A myogenic bulbar palsy may be seen in oculopharyngeal muscular dystrophy, inclusion body myositis, and polymyositis.

**Cross References**
Bovine cough; Dysarthria; Dysphagia; Dysphonia; Fasciculation; Gag reflex; Jaw jerk; Lower motor neurone (LMN) syndrome; Pseudobulbar palsy; Upper motor neurone (UMN) syndrome

**Bulbocavernosus Reflex**
A test of the integrity of the S2, S3, and S4 spinal roots, looking for contraction of the anal sphincter (may be felt with a gloved finger in the rectum) when squeezing the glans penis or clitoris. The reflex may be abolished in lesions of the cauda equina.

**Cross References**
Cauda equina syndrome; Reflexes

**Buphthalmos**
Buphthalmos, literally ox-eye, consists of a large and bulging eye caused by raised intraocular pressure due to congenital or secondary glaucoma. This is one of the ophthalmological features of Sturge–Weber syndrome.

**Butt-First Manoeuvre**
- see GOWERS’ SIGN
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