Abnormal Gaits/Ataxia

Normal gait

As the body moves forward, one limb provides support while the other is advanced in preparation for becoming the support limb. The gait cycle is composed of stance (initial double stance, single limb stance, terminal double limb stance) and swing phases. Duration of each aspect of stance decreases as walking velocity increases. The transition from walking to running is marked by loss of double support. A stride is the equivalent of a gait cycle. The duration of a stride is the interval between sequential initial floor contacts by the same limb.

Normal age-related changes in gait

Walking posture changes only slightly with age:

- Elderly people walk with about a 5° greater "toe out", possibly due to a loss of hip internal rotation or in a subconscious strategy to increase stability.
- Gait velocity remains stable until about 70, then falls about 15% per decade
- Double stance (the time when both feet are on the ground) increases with age – from 18% of a total gait cycle in young adults to approximately 26% in healthy elderly.
- Joint motion changes with age.

Common Causes of Ataxia/Gait disturbances

Systemic

- Intoxication with ↓alertness – EtOH, sedatives
- Intox without ↓alertness – phenytoin, carbamazepine, valproate, heavy metals
- Metabolic – HypoNa, inborn errors of metabolism, Wernicke's

Primarily Nervous System Disorders

- Cerebellar – CVA, degenerative changes, abscess, tumour, post-viral (children)
- Cortical – tumour, haemorrhage, trauma, hydrocephalus
- Subcortical – thalamic CVA, Parkinson's. normal pressure hydrocephalus
- Spinal – cervical spondylosis, posterior column disorders
- PNS – peripheral neuropath, vestibulopathy

Ataxia

Often categorized into:

Motor (or cerebellar) ataxia: cerebellar disorders usually – loss of integration of proprioceptive info. Lateral cerebellar lesions → motor ataxia of ipsilat limb. Causes wide based, unsteady irregular gait with difficulty turning/avoiding obstacles. Lesions of the vermis often → axial muscle incoordination (truncal ataxia). Romberg –ve.

Sensory ataxia: problem with peripheral nerves, dorsal columns or cerebellar input tracts that prevent afferent position sense/proprioceptive info reaching cerebellum. Causes abrupt movement of the legs with foot slapping impact with ground. Romberg +ve.

Antalgic gait

In this the patient avoids certain painful movements. It can be seen as a feature of:

- Trauma
- Osteoarthritis
- Pelvic girdle pain

Cerebellar disease

Ataxia is the most prominent manifestation of cerebellar disease. Eye movements, muscle tone, skilled movements and speech are also affected by cerebellar disease.

Aetiology

This may include trauma, toxic and metabolic causes, neoplasms, immune mechanisms and genetic diseases. It may also arise from multiple sclerosis. Children aged between two and ten may also present with sub-acute reversible ataxia stemming from a viral infection.

Characteristics

- Broad-based gait & posture
- Lurching quality
- Difficulty walking in a straight line & turning

Assessment includes walking heel to toe, finger-nose test, Rombergs (-ve), dysdiadochokinesis. Diurnal variation with morning unsteadiness which improves later in the day may suggest ↑ICP. Cerebellar ataxia of extremely acute onset accompanied by headache, vertigo, vomiting, altered consciousness and neck stiffness - ?cerebellar bleed.

Parkinsonism

The bradykinesia and slowness of postural adjustments, together with a forward-flexed posture produces the "festinating gait" typical of Parkinson's disease.

Hemiparesis

After a CVA

- The strong gluteals and quadriceps muscle groups are generally spastic.
- The hip flexors, hamstrings and dorsiflexors of the foot are generally weak.
- The hip and knee will thus be stiff and slightly flexed.
- The foot will be plantar-flexed and tending to drag along the floor.

This indicates pyramidal pathway damage - and the residual power left is dependent on non-pyramidal pathways and there being enough residual cortical function.

Neuropathies

Aetiology

These disorders can arise from:

- Diabetes
- Alcoholism
- HIV
- Toxin exposure
- Metabolic abnormalities
- Vitamin deficiency
- Adverse effects of certain drugs

However 32-70% of all peripheral neuropathies are idiopathic.

Characteristics

Unsteady gait, often high stepping.

This is virtually diagnostic of neuropathies when present.

Patients may fall over if asked to close their eyes.

Paraparesis

Damage to the descending corticospinal tract e.g. by a tumour may present initially with a generalised stiffening of the legs. The patient may find it impossible to walk quickly or run because of a stiff-legged gait. Ankle clonus may be a feature, and eventually develops into a spastic, foot dragging "shoe scuffing" gait.

Scissor gait

Aetiology

Usually seen in spastic cerebral palsy, usually diplegic and paraplegic varieties

Characteristic features

- Legs flexed slightly at the hips and knees ± contractures of the adductors, giving the appearance of crouching. with the knees and thighs hitting or crossing in a scissors-like movement.
- Often mixed with or accompanied by spastic gait, a stiff, foot-dragging walk caused by one-sided, long-term muscle contraction.
- The individual is forced to walk on tiptoe unless the dorsiflexor muscles are released by an orthopaedic surgical procedure.
- There may also be complicated assisting movements of the upper limbs when walking.

These features are typical and are usually present to some degree regardless of the mildness or severity of the cerebral palsy.

Vestibular disorders

Patients with unilateral vestibular disorders:

- Will veer to the affected side
- There is a wide-based gait and difficulties exaggerated by walking heel-to-toe.

Many patients respond well to a simple home program of vestibular rehabilitation head movement exercises. This results in reduced symptoms of imbalance during stance and gait.