Variations in normal gait development
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Abstract
Normal development is not an exact science. Children progress in a variety of ways without falling outside the normal range. This applies to the shape of their legs and feet, as well as the onset and development of their gait pattern. Deviation from parents’ expectations may lead to considerable anxiety and medical consultation. It is essential to identify the important pathological diagnoses amongst the array of normal variants to facilitate early intervention and optimise outcomes.

Keywords gait development; leg deformity; preschool children; rickets

Introduction
The attainment of motor milestones such as standing and walking is anticipated and celebrated by parents. Variation from the perceived norm often generates anxiety and medical consultation. Whilst most of these variations fall within the normal physiological range, pathological processes and abnormal progression of development must be identified to facilitate appropriate management.

Normal variants of an average gait pattern may be associated with significant deformity but children often remain remarkably mobile. Toe walking, flatfeet, bow legs and knock knees, in-toeing and out-toeing can all be part of normal gait development, but the important point is that physiological variants resolve spontaneously. The mainstay of treatment is explanation and reassurance.

Recognising the cases which are outside normal limits or those who progress in an abnormal way over time allows the identification of neuromuscular or musculoskeletal problems that require a full assessment and/or medical treatment in conjunction with orthopaedic management.

Normal gait
Most children begin to walk independently between 12 and 14 months. They typically walk with straight knees and a wide-based gait. Initial ground contact may vary from heel-toe, whole foot or toe-heel. The adult pattern of heel-to-toe gait develops around 3 years, whilst changes to velocity and cadence continue up to age 7 (Figure 1).

Toe walking
In normal variant toe walking, the child may cruise on tip-toe but relax into a flat foot when standing still or on request. Normal adult heel-to-toe gait should prevail by age 3 but a few children continue to tip-toe walk. An underlying pathological cause for toe walking should be considered in children who develop toe walking after a period of walking on their heels and in those in whom toe walking is unilateral.

Idiopathic Toe Walking (ITW) is defined as “persistent toe walking after the age of 2 in healthy children without a neurologic or orthopaedic diagnosis”. It is thus a diagnosis of exclusion. It is thought to result from relative shortening of the soleus or gastrocnemius muscles effectively tightening the Achilles tendon. Most cases of ITW will improve spontaneously but for those that do not, a variety of treatments such as stretching techniques, serial casting, night braces and ankle-foot orthoses have been suggested. There is little evidence that outcomes from any intervention is better than the natural history. For cases where there is no major tightness of the muscle, in theory, botulinum toxin injections may be beneficial. A minority of patients

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Figure 1 Development of a mature gait. Top: 1 year old — flexed elbows, no arm swing. Middle: 3 year old — arm swing and heel strike. Bottom: 6 year old — adult-type gait. Figure 2.4 from Benson et al.
with significant tightness of the gastrocnemius—soleus complex and an inability to stand with their heels down may require operative lengthening of the Achilles tendon. It is essential that the parents understand, that surgical lengthening or indeed stretching of the muscle will only resolve the contracture and allow the heel to touch the ground: the urge/desire to be a tip toe walker often remains as it is centrally driven. The child must relearn their gait pattern to become a heel-to-toe walker.

The most common pathological causes for toe walking are a limb length difference (i.e. a short leg), a dislocated hip or a neuromuscular problem such as Muscular Dystrophy (MD), Cerebral Palsy (CP) or Charcot-Marie-Tooth disease and other Hereditary Sensori-Motor Neuropathies (HSMNs).

Muscular Dystrophy describes a group of inherited genetic disorders resulting in progressive muscle weakness and increasing disability, the most common of which is Duchenne muscular dystrophy (DMD); an X-linked recessive disorder affecting 1 in 4000 boys. Classically, in these children, toe walking starts after a period of normal heel-to-toe walking. The exact clinical presentation depends on the type of muscular dystrophy but may include a delay in independent walking. A suspected diagnosis is supported by markedly elevated Creatinine Kinase (CK) levels and confirmed by DNA testing.

Cerebral Palsy is a fixed, non-progressive brain lesion that occurs before, at or soon after birth. It is estimated to affect 1 in 400 children. Cerebral palsy may present in many ways, dependent on the extent of the brain lesion. Mild, hemiplegic CP may present as unilateral toe walking or a spastic diplegia with bilateral toe walking. Examination will reveal a child with permanent equinus during gait and perhaps whilst standing. Spasticity will often be present in the lower limb but it is important to look at the arms too as posture changes are sometimes more obvious in the upper limb than the lower limb. A careful clinical examination will distinguish between CP and ITW. Dynamic electromyography (EMG) may facilitate differentiation between CP and Idiopathic Toe-Walking.

Flatfeet

The term ‘flatfeet’ is used to label feet with a depressed medial longitudinal arch. Flatfeet occur as part of normal development: babies have no detectable medial arch as it is obscured by a fat pad until the onset of walking. The medial arch typically appears between 2 and 3 years of age. In a proportion of children failure to develop this arch occurs as part of a pathological process. Flatfeet can be divided into three subgroups: physiological flexible flatfeet, pathological flexible flatfeet often with a short Achilles and rigid flatfeet.

Flexible flatfeet are part of normal development. They are present from birth, run in families and are more frequent in both obese children and certain races. They are associated with familial joint laxity. Examination reveals a foot with good mobility of the subtalar joint. The arch is seen when the child is non-weight bearing and is reproducible in standing using the Jack’s Test, where the great toe is extended by the examiner or visible when the child stands on tip-toe. See Figure 2.

Flexible flatfeet may be associated with activity related pain and nocturnal ache. Traditionally these patients have been managed with exercises and orthoses. There is no evidence this improves outcomes, but it may relieve some symptoms and increase longevity of footwear by altering the wear pattern of shoes.

Flexible flatfeet with a short Achilles tendon occur in older children and may account for 25% of flatfeet in adults. Diagnosis is confirmed when less than 10 degrees of dorsiflexion can be achieved with the knee fully extended and the hindfoot valgus corrected. The majority of patients improve with simple stretching exercises, although a few symptomatic patients may require surgical lengthening procedures or osteotomies. Flexible flatfeet associated with soft tissue laxity conditions can be very difficult to treat.

Rigid flatfeet are pathological and a cause should be identified. Examination reveals a stiff subtalar joint and persistence of flatfeet despite great toe extension. Most result from tarsal conditions, such as calcaneonavicular or talocalcaneal coalition. These patients present with pain at the time that the cartilaginous coalition begins to ossify; between 8 and 12 years in calcaneonavicular coalition or between 12 and 16 years in talocalcaneal coalition. The coalitions may be imaged with CT or MR scans. The pain may be relieved by application of a below-knee walking cast, which should be applied for 4–6 weeks. Orthotic supports can be tried but are often unsuccessful as they can not correct the alignment of a rigid hindfoot. If symptoms persist despite initial treatment, surgical options include resection of the coalition, osteotomy or, in older patients with advanced arthritis, triple arthodesis.
**Bow legs and knock knees**

Both bow legs and knock knees are normal variants at certain stages in development. Babies are born bow-legged and continue to be so until 2 years of age. Children develop knock knees between 3 and 6 years before straightening out to normal physiological valgus of approximately 6 degrees.

Deviation from the normal pattern of varus or valgus by more than two standard deviations requires investigation. Unilateral deformities or those associated with leg length discrepancy or causing gait disturbance and pain should also be explored. Initial investigation should include full length radiographs of legs, along with lab studies to assess bone profile, thyroid function and chromosomal analysis. Height and weight charts should also be kept and the child’s height related to parental heights: short stature is an important clue to a skeletal dysplasia.

Legs are determined as bowed if there is separation between the knees when standing upright with the ankles touching. The inter-condylar distance can be measured to allow quantitative data to assess progression or improvement. Bow legs often occur in conjunction with outward femoral torsion and inward tibial torsion. There are several potential pathological causes for bow legs, including Blount’s disease, skeletal genetic dysplasias and growth plate injuries. Rickets is, however, the most important diagnosis to exclude.

Children with rickets often have a typical history, with at least one risk factor (Table 1). They are unlikely to have been taking Vitamin D supplements or drinking Vitamin D fortified milk. Most cases of Rickets are a result of Vitamin D deficiency, of which there may be a family history. Blood tests will show an alkaline phosphatase greater than twice the upper limit of normal, a low vitamin D level (usually less than 25 nmol/litre) and a raised parathyroid hormone. Serum calcium and phosphate may also be reduced. X-rays of a child with rickets will show fraying, cupping and splaying of the metaphyses around the knees, ankles and wrists with widening of the growth plate, bone demineralization and a widened joint space.

Risk factors for Vitamin D deficient rickets

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Description</th>
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<tbody>
<tr>
<td>Lack of sun exposure</td>
<td>Dark skin, dark pigmentation, living far from the equator</td>
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<tr>
<td>Diet</td>
<td>Prolonged breast feeding, poor dietary intake of vitamin D</td>
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<tr>
<td>Drugs</td>
<td>Anti-convulsant therapies, P450 inducing drugs, anti-TB therapy</td>
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<tr>
<td>Chronic diseases</td>
<td>Malabsorption syndromes e.g. Coeliac, chronic liver disease, chronic renal</td>
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**Table 1**

Because it is not being calcified adequately. See Figure 4. Management of these patients focuses on correction of the underlying cause. Bracing is rarely used.

Blount’s disease is a knee deformity of unknown aetiology. It is more common in overweight African Americans and is due to an abnormality of the proximal medial tibial physis exacerbated perhaps by excessive weight. It may be associated with an early onset of walking. It is often bilateral and associated with tibial torsion. Radiographs typically show varus deformity in the proximal tibia and metaphyseal beaking of the medial tibial condyle. Weight should be controlled if possible, but surgery may also be required: often repeated during growth. A combination of treatment method may be required including growth plate fusions, osteotomies, external fixator correction of limb deformity and length with the aim of obtaining straight limbs of equal length at skeletal maturity.

Knock knee deformity is measured by the intermalleolar distance when the patient is standing upright with the knees touching. A distance of more than 10–15 cm is considered pathological particularly if it is progressive. Children may walk with their knees rubbing and their ankles pronated. They may have associated inward femoral torsion and outward tibial torsion. Causes are varied; from skeletal dysplasia to fractures, infection or benign tumours and can include metabolic bone diseases such as rickets. Most cases resolve spontaneously if they are within the limits of the normal curve (Figure 3). Bracing has been found to be ineffective. Surgical management, reserved for those with persistent, significant deformity, is again with hemiepiphysiodesis or osteotomy.

**In-toeing and out-toeing**

The foot progression angle (FPA) represents the angular difference between the child’s foot and the direction in which it is walking: it may be negative when the feet point in and positive when the feet point out. The source of the rotation may be anywhere in the leg.

In-toeing is common and usually part of normal variation. Only if the deformity is more than two standard deviations from...
the norm is in-toeing considered a torsional deformity. Deformity may result from medial femoral torsion (anteversion), medial tibial torsion or forefoot (metatarsus) adductus. The child’s rotational profile, determined by examination, can identify the site of the problem. Most cases will resolve spontaneously. Rigid forefoot adductus benefits from stretching and casting but severe in-toeing persisting into the teens may require a corrective osteotomy.

Medial tibial torsion (MTT) is common in toddlers and tends to improve with growth. There is limited evidence for the use of night splints and corrective osteotomy is rarely necessary.

All infants have femoral neck anteversion, in most it has improved by walking age but if it does not it can be a cause of an in-toeing gait. It is also often described as internal or medial femoral torsion. Medial femoral torsion runs in families and is more common in girls. It leads to in-toeing in combination with a medially facing patella and an ability to ‘W’ sit easily. Femoral neck anteversion improves with time and the child should learn to control their foot progression angle. If the deformity persists or causes problems with gait, it may require corrective osteotomy.

Out-toeing describes a lateral rotation of the foot relative to the direction of walking. It is common in infants when they first start walking but usually resolves within a year. In the older child, when out-toeing is secondary to lateral tibial torsion (LTT) it may be progressive and require surgical correction with an osteotomy. Lateral femoral torsion (LFT) (or relative femoral neck retroversion) is rarely severe enough to require treatment. There is, however, some evidence that it increases the risk of osteoarthritis of the hip and may be a risk factor in slipped capital femoral epiphyses.

The foot progression angle (FPA) is a result of the torsional profile of the whole lower limb: in Torsional Malalignment Syndrome (TMS) this angle might be normal despite significant torsional deformity. TMS is usually a combination of medial femoral torsion and lateral tibial torsion, leading to a medially rotated knee despite a normal FPA. Patients may present as teenagers with patellofemoral pain, symptoms of chondromalacia patellae or patella subluxation or dislocation. Management is focused on behavioural modification. If surgical intervention is required then double osteotomy may be necessary to correct both the femoral and tibial deformities.

**Summary**

The massive range in both appearance and dynamic motion seen in children during gait development is remarkable. Whilst variations are only considered deformities when they lie outside two standard deviations from the norm, parents are often concerned, prompting medical consultation for children within these limits. As medical professionals it is imperative that we address parental concerns, investigate when appropriate and identify those children who warrant further management. In most cases, we need do nothing more than provide reassurance for parents that variations will correct with time, and remind them that each child is unique.

**FURTHER READING**

4. Normal development of the tibio-femoral angle in children: [http://www.boneandjoint.org.uk/content/fo...](http://www.boneandjoint.org.uk/content/fo...).