Toe walking: causes, epidemiology, assessment, and treatment

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Purpose of review
The present review includes the most up-to-date literature on the causes, epidemiology, diagnosis, and treatment of toe walking.

Recent findings
The prevalence of toe walking at age 5.5 years is 2% in normally developing children, and 41% in children with a neuropsychiatric diagnosis or developmental delays. A recent systematic review concluded that there is good evidence for casting and surgery in the treatment of idiopathic toe walking, with only surgery providing long-term results beyond 1 year. Botox combined with casting does not provide better outcomes compared with casting alone. Ankle-foot-orthoses restrict toe walking when worn, but children revert to equinus gait once the orthosis is removed.

Summary
Toe walking can occur because of an underlying anatomic or neuromuscular condition, but in the majority of cases toe walking is idiopathic, without a discernable underlying cause. For some families, toe walking may simply be a cosmetic concern, whereas in other cases it can cause pain or functional issues. Treatment for toe walking is based on age, underlying cause, and the severity of tendon contracture. Described treatments include casting, chemical denervation, orthotics, physical therapy, and surgical lengthening of the gastroc–soleus–Achilles complex. A careful history, clinical exam, and selective diagnostic testing can be used to differentiate between different types of toe walking and determine the most appropriate treatment for each child.

Keywords
ankle-foot-orthoses, equinus, orthotics, serial casting, tendon lengthening, toe walking

INTRODUCTION

Toe walking entails walking on the toes or forefoot, with a lack of heel strike upon initiation of the stance phase of gait. Toe walking is considered a normal stage in gait development, but should resolve by 3–7 years of age [1,2]. Although the majority of toe walkers are otherwise healthy children, it is important to approach each case with a high index of suspicion as toe walking can be due to a structural problem such as a contracted tendon, compensation for a short limb, or a manifestation of autism spectrum disorders (ASDs), cerebral palsy, muscular dystrophies, or other neurologic or neuromuscular conditions (Table 1). Idiopathic toe walking (ITW) is a diagnosis of exclusion; the diagnosis can only be made when other potential diagnoses have been ruled out.

Toe walking (Fig. 1) is a frequent cause of presentation to the pediatrician, neurologist, podiatrist, and pediatric orthopedic surgeon, with toe walking accounting for approximately 1% of visits to the pediatric orthopedist [3]. For some families, toe walking may simply be a cosmetic concern, whereas other cases may be related to foot or ankle pain, increased energy expenditure with development of compensatory gait abnormalities, or psychological distress for the child or family. The prevalence of toe walking at age 5.5 is 2% in normally developing children, and 41% in children with neuropsychiatric diagnosis or developmental delays [4], with slight male predominance [5].

Joint mobility is typically normal early on, but limited dorsiflexion can develop over time [5],

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KEY POINTS

- Toe walking can be related to structural abnormalities such as tendon contracture or leg length discrepancy, or neurologic/neuromuscular conditions such as autism, cerebral palsy, or muscular dystrophy.
- Most cases of toe walking are idiopathic, without a discernable underlying cause.
- Physical therapy, orthotics, serial casting, chemical denervation, and surgical lengthening of the gastroc–soleus–Achilles complex are described treatment options.
- Treatment for toe walking is based on age, underlying cause, and the severity of tendon contracture.
- Recent systematic reviews show good evidence for the use of casting and surgery, with surgical interventions showing the most promising long term results.

presumably because of relative overuse of the gastroc–soleus complex and frequent positioning in plantarflexion with diminished muscle movement. ITW is often described as a habit, a position that child prefers to be in, but that when asked or reminded, the child can voluntarily achieve heel strike.

Although previously thought to be exclusively due to habit or preference, evidence for a neurologic basis in some cases exists [6]. ITW has been associated with speech/language delays, abnormalities in executive functions, social skills, learning, and memory [7], with differences in motor control, sensory processing, and vibration perception thresholds [6] when compared with adolescents with typical gait. At the cellular level there is an increased concentration of type 1 muscle fibers among toe walkers [8]. Ten to 88% of idiopathic toe walkers have a family history of toe walking [5] with an autosomal dominant inheritance with incomplete expression observed in some families [9].

Table 1. Causes and diagnoses associated with toe walking [31]

Neurologic/neuromuscular	Other
Cerebral palsy	Idiopathic
Muscular dystrophy	Limb length discrepancy
Charcot-Marie-Tooth disease	Clubfoot
Spinal cord or brain injury	Tendon or Joint Contracture
Tethered cord	Ankylosing spondylitis
Spina bifida	Unilateral hip dislocation
Autism spectrum disorder	Plantar foot injuries
Schizophrenia	



FIGURE 1. A child toe walking.

Toe walkers most commonly present for medical evaluation in the toddler years [10]. Typically, the caregiver notices an abnormality in gait compared with siblings or peers. Poor balance, frequent falls, pain, and fatigue are other potential concerns [5]. The majority of patients with ITW walk at a normal age, and can stand plantigrade [5]. Spontaneous heel strike occurs from 15–92% of the time [11] and sometimes is under voluntary control whereas in most nonidiopathic cases the child cannot voluntarily produce heel strike.

Typically, ITW is first observed when children first learn to walk, whereas in neuromuscular conditions such as cerebral palsy it may present at initial walking or develop over time. There is limited information on the natural history of ITW. In a cohort of children with ITW, observed for an average of 3 years [10], only 12% had completely ceased to toe walk at final follow up. Non-ITW typically does not resolve spontaneously.

Toe walkers may be prone to ankle sprains [12] and have less efficient gait, with decreased step length and increased cadence leading to increased energy expenditure [13]. Overuse of the gastroc–soleus may result in muscle cramps. Over time the increased pressure on the metatarsal heads can lead to foot pain, callouses, stress fractures, and difficulty with shoe wear [12]. A careful history, physical exam, and in some cases diagnostic testing can help to classify the type of toe walking, and determine the optimal treatment.

HISTORY

Ascertain the family's main concerns, and whether the child is having difficulty with function (tripping, difficulty keeping up with peers), pain, social

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problems, or cosmetic concerns. Obtain a detailed birth and developmental history, with attention to both motor and communication milestones. Determine if the toe walking was first noted when the child learned to walk, or if it has developed over time. Note the proportion of time the child toe walks and whether the child can stand and walk with heel contact, if cued. Past medical, surgical, and family history should be obtained, specifically inquiring about toe walking or neuromuscular conditions in other family members. Inquire into improvement over time, and previous treatments and their efficacies.

EXAMINATION

If possible, observe as the child arrives at the examination room. Children are more likely to show their natural gait before they are aware of being watched. Instruct the child to walk naturally, and to then attempt heel strike both barefoot and in shoes. Watch the child walking toward and away, and also observe from the side. Starting at the foot, observe during the stance phase (segment of gait cycle when the foot contacts the floor) for heel strike. Estimate the degree of equinus in stance phase. During swing (nonweight bearing segment of the gait cycle) assess whether the ankle appropriately dorsiflexes. Assess knee and hip motion during gait. Assess laterality of toe walking as unilateral toe walking can be a sign of underlying diseases such as hemiplegia or unilateral hip dislocation. Assess posturing of the upper extremities while walking/running as this may be indicative of cerebral palsy.

In a neutral standing position assess whether the heels contact the ground. Leg lengths can be assessed in this position by assessing for pelvic obliquity, and if obliquity is identified, placing blocks beneath the shorter leg until a level pelvis has been achieved. Assess for muscle atrophy, commonly seen in clubfoot, spinal cord injuries and cerebral palsy, or calf hypertrophy which may be seen in muscular dystrophy.

The range of motion at the hips, knees, ankles, and subtalar joints should be assessed with the child in a supine position. Ankle dorsiflexion should be performed with the foot in a subtalar neutral position (i.e. neither inverted nor everted), ensuring the measured dorsiflexion is coming from the ankle and not exaggerated due to motion at the subtalar joint. The Silverskiold test (Fig. 2) can differentiate whether limited dorsiflexion is solely gastrocnemius tightness or gastrocnemius and soleus combined. Normal ankle dorsiflexion in 2–8 years olds with the knees flexed has been reported at 25° and 23° for females and males respectively, with these numbers decreasing with age; young adults having 14° and 13° dorsiflexion, respectively [14]. A goniometer helps to accurately estimate dorsiflexion, and track response to treatment.

Assess femoral anteversion through hip internal/external rotation and tibial torsion through thigh foot angle with the patient prone. [15]. Not only are rotational abnormalities common in neuromuscular conditions such as cerebral palsy, but increased external tibial torsion is frequently seen in toe walking. Compensatory external rotation is hypothesized to develop over time as a means of increasing gait efficiency [16]. Palpate for areas of bony or soft tissue tenderness. Assess ligamentous laxity, skin for callouses or erythema, and patient's shoes for wear pattern.

A full neurologic exam should be completed, including assessment for sensation, strength, deep tendon reflexes, tone, clonus, Babinski, and special tests including Gower's test. A positive Gower's

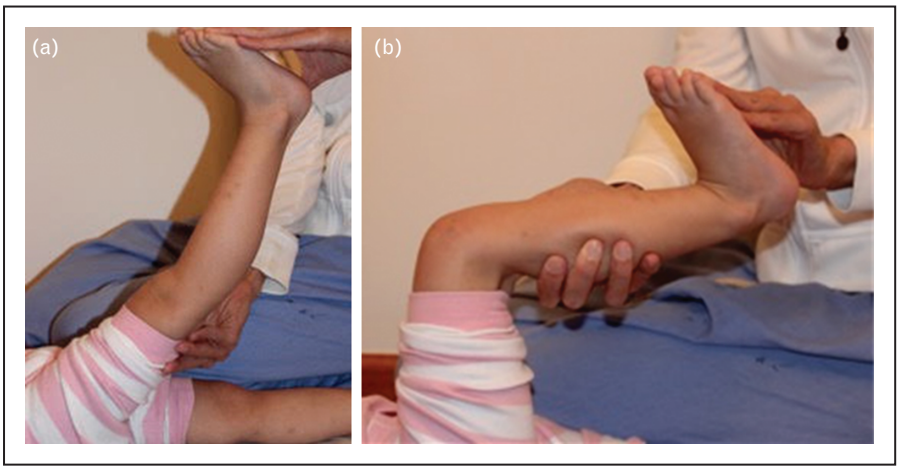


FIGURE 2. The Silverskiold test. (a) Ankle dorsiflexion with the knee extended. (b) Ankle dorsiflexion with the knee flexed, eliminating the contribution of the gastrocnemius.

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can be seen in muscular dystrophy and other neuromuscular conditions. Even with a normal neurologic exam, one may consider referring toe walking patients to a pediatric neurologist [7] for a formal neurologic assessment, especially when it persists beyond 3–5 years, given the frequent association with underlying neurologic conditions and potentially subtle findings on clinical exam.

DIAGNOSTIC TESTING

Laboratory studies and imaging may be helpful in differentiating between pathologic and idiopathic toe walking. Consider creatine phosphokinase (CPK) as a simple screening test, and muscle biopsy for more detailed testing if muscular dystrophy or myopathy are suspected. Radiographs of the foot and ankle are rarely indicated, but could be obtained in the setting of a rigid equinus deformity. Formal gait analysis can help differentiate between ITW and neuromuscular disorders. Cerebral palsy typically shows increased knee flexion, ITW shows more variable kinematics at the ankle [17], and CMT typically shows delayed peak dorsiflexion in stance phase [17]. Electromyography (EMG) can help differentiate between ITW and mild diplegic cerebral palsy. Although early firing of the gastroc–soleus occurs in both conditions, coactivation of the gastrocnemius during knee extension is much more common in diplegic cerebral palsy than ITW, and is rare in normal children [18]. Brain and spinal MRI may be helpful in the setting of an abnormal neurologic exam and can assess for brain or spinal cord abnormalities.

TREATMENT – IDIOPATHIC TOE WALKING

Long-term natural history studies are limited; it is unknown whether treatment for toe walking is mandatory, or at what age intervention should be initiated. Many treatment options have been described, including no treatment, stretching of the gastroc–soleus–Achilles with physical therapy or casting, surgical lengthening of the gastroc– soleus–Achilles, blocking plantar flexion as with an ankle-foot-orthosis, or some combination of these.

Cueing the child to walk with a heel strike has not been shown to be effective [19], likely attributable to a child's short attention span. Children will often walk with a heel strike for a few steps and then revert back to toe walking. Verbal cueing to walk with a heel strike can be frustrating for both the child and family. Unfortunately, there is no evidence that cueing leads to sustainable long term normal gait.

There is little evidence that physical therapy and home stretching provide long-term change in ankle range of motion or toe walking in ITW. In a mixed group of patients that received either physical therapy or were simply observed, only 12% attained normal gait [10]. Botox has been tested in conjunction with a stretching program and at 1 year, toe walking resolved in 1/3, was less frequent in 1/3 and persisted entirely in 1/3 [20]. Unfortunately, this was a case series without a comparative group, and it is unclear whether the improvements documented would have been greater than observation alone. Because of insufficient evidence, this modality in isolation is not the authors' preference in children without spasticity.

Rigid inserts, frequently made of carbon fiber [21▪] theoretically work by sensory feedback or perhaps by limiting foot flexibility, thereby making it difficult to toe walk. AFOs with a plantarflexion block limit plantarflexion beyond neutral. In one of the few prospective studies on the treatment of toe walking, children were randomized to AFO versus rigid shoe insert. The AFO was 100% effective at ensuring heel strike, whereas heel strike only occurred 87% of the time in the foot orthotic group. When orthotics were discontinued at 6 weeks, the improvement seen in the AFO group was not sustained. Toe walking resumed to some degree in both groups. Long-term follow up was not assessed [21▪]. AFOs are bulky from the hinged ankle, and visible unless under pants. There can be difficulty finding accommodating shoes, and families often dislike their appearance. Rigid foot orthoses and AFOs can be uncomfortable because of their rigidity, and expensive as they are typically custom made. Finally, AFOs effectively trade one gait deviation for another by promoting a heel strike, but consequently limiting push-off. AFOs may be reasonable postoperatively for some patients after surgical lengthening, but in general are not the authors' preference for first line treatment of ITW.

Casting involves the application of short leg casts in maximal dorsiflexion for 4–6 weeks. A waterproof lining allows children to bathe, and a rubber sole provides traction. Casting theoretically works by two mechanisms. First, the child has no choice but to walk with heel contact for the time they are in casts, which is full time, and this may break the 'habit' of toe walking. Second, casting provides a continuous stretch to the gastroc–soleus–Achilles complex. For children with limited dorsiflexion, casts are replaced every 1 to 2 weeks, gaining additional dorsiflexion with every cast change. For children without significant contractures, the cast could be molded in maximum dorsiflexion and cast changes are not required over

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the period of casting. At the completion of casting, a course of physical therapy may help regain strength and coordination as mild temporary weakness can develop. A randomized trial compared 4 weeks of casting versus Botox and casting [22]. In both groups ankle dorsiflexion improved by 5 degrees. Range of motion and gait improvements were sustained at 12 months, although the majority of children still intermittently toe walked at study completion. There was no benefit from the addition of Botox to the casting regimen. A number of series have shown improvement with cast treatment for both children with ITW and cerebral palsy [3,23], although these series were without comparative group and it is possible that improvements were no greater than observation alone.

Surgical lengthening of the gastroc–soleus–Achilles is an option in patients with limited dorsiflexion (Fig. 3). Following surgical lengthening, a postoperative cast in a dorsiflexed position is typically required for 4–6 weeks to facilitate healing in a lengthened state. Depending on the type of lengthening performed, children may be permitted to weight bear in the casts. Operative complications are reported at 7% [25▪] and include excessive lengthening with associated weakness, sural nerve injury, painful scars, and wound infections.

Studies have consistently demonstrated improvement with surgical lengthening. However, formal gait analysis has shown mild persistent anterior pelvic tilt, slightly limited dorsiflexion in stance and swing, increased external rotation through the femur and tibia [16], and abnormal ankle powers [26]. A series of toe walkers with Achilles tendon contractures [27] underwent tendon lengthening and postoperative casting. At mean follow-up of 3 years all patients had normal dorsiflexion. A small proportion still toe walked intermittently. Unfortunately, this was a retrospective case series without comparative controls.

A retrospective study of toe walkers compared observation/physical therapy to casting/AFOs and surgical lengthening [28]. Prior to treatment, mean dorsiflexion for each group was 10°, 5° and -10° degrees respectively. Satisfaction and improved dorsiflexion were greatest in the surgical group, whereas a portion of patients treated with observation, stretching, and AFOs actually developed more restricted dorsiflexion over time. The study had a 22% attrition rate, and groups were heterogeneous without separate groups differentiating between AFOs and casts, or between observation and physical therapy, or Achilles lengthening versus gastroc recession.

Following observation, casting, and surgical lengthening, at 2- to 22-year follow up, normal gait occurred only in 12, 22, and 37%, respectively [10]. The authors concluded that the majority of children do not actually outgrow toe walking with observation alone. Surgery showed the most promising

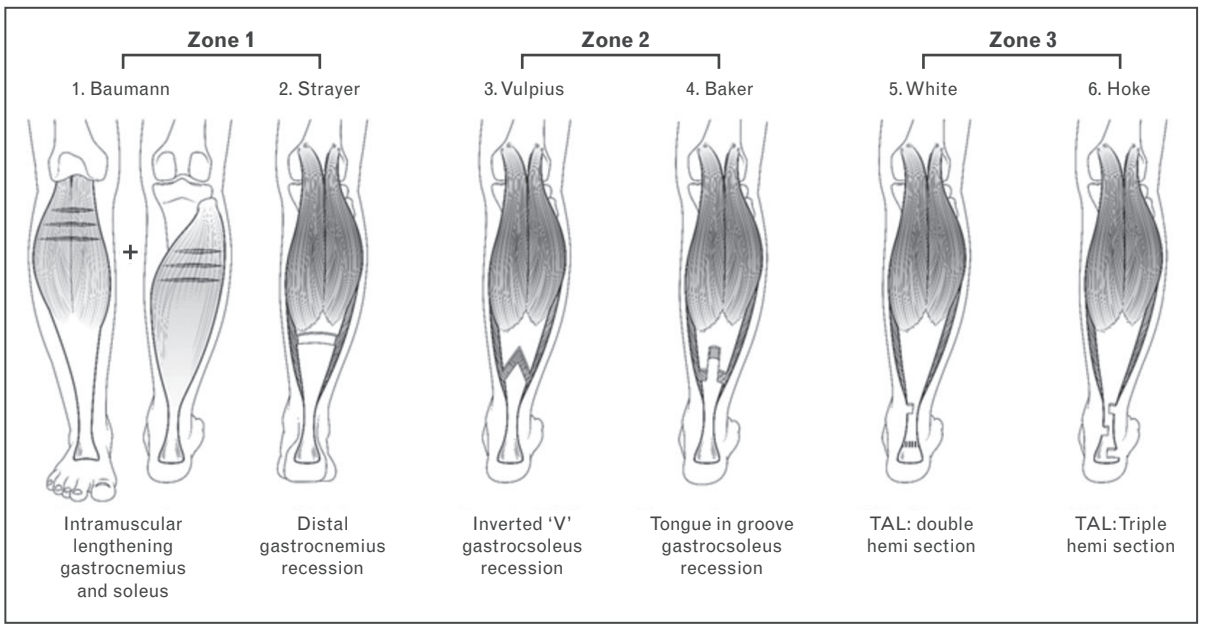


FIGURE 3. Surgical gastroc–soleus–Achilles complex lengthenings. Zone 1 procedures (i.e. Baumann and Strayer) lengthen only the gastrocnemius muscle proximally. Zone 2 procedures (i.e. Vulpius and Baker) lengthen the gastrocnemius muscle at its distal tendinous portion. Zone 3 procedures (i.e. White and Hoke) lengthen the Achilles tendon. Permission JBJS [24].

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results, despite the fact that this group was on average older and had more severe dorsiflexion limitations at the time of treatment.

Three systematic reviews have been published recently. The first assessed outcomes following casting and surgery; surgery improved dorsiflexion range by 10° degree more than casting, but there was no significant difference in proportion toe walking at follow up [25▪]. Williams *et al.* [29▪] assessed 21 manuscripts and concluded that there was evidence for casting, surgery and Botox, but little evidence for footwear and orthotic interventions. Van Kuijk *et al.* [30▪] assessed 19 studies and concluded there was evidence for casting and surgery, that Botox does not improve the effects of casting, and that only surgery has shown long-term results. These systematic reviews included a mix of studies containing patients with varying degrees of ankle range of motion. Based on the evidence presented, it is not possible to determine whether these interventions apply only to children with limited dorsiflexion (contracture), or if toe walking children with normal ankle range of motion could also benefit from the recommended treatments.

Optimal treatment for ITW has not been fully elucidated. Reassuring parents that toe walking does not cause back pain, hip pain, or arthritis is often useful. The authors preferred primary treatment for children under 5 years, with dorsiflexion beyond neutral, is to recommend observation, as the majority of toe walking will resolve by this age [4]. For children 5 years and older, the authors' preferred treatment algorithm includes: for children with ankle dorsiflexion greater than 10°, continued observation is an option, or a cast in maximum dorsiflexion may be applied for 6 weeks; for children with ankle dorsiflexion that is limited, maximum dorsiflexion casts can be applied and changed every 1–2 weeks with additional dorsiflexion attained with each cast change until sufficient dorsiflexion is achieved or the correction has plateaued. Casting is generally not effective for children older than 7. For children that fail casting, or have severely limited motion and cannot achieve dorsiflexion to neutral, surgical lengthening can be considered. A short course of physical therapy after casting or surgery is reasonable to recover strength. In children who achieve good dorsiflexion, but the 'habit' of toe walking persists, an AFO may be considered.

TREATMENT – AUTISM SPECTRUM DISORDERS

The treatment algorithm for toe walking in autism is similar to that of ITW. However, because of the potential for cognitive impairment, as well as the high sensory drive in many children with ASD, toe walking may be resistant to treatment and if treated, more likely to recur. Children with ASD will often not tolerate casts or orthotics and may have difficulty participating in physiotherapy. If the toe walking child with ASD is not having pain or serious functional issues related to toe walking, it may also be reasonable to observe only and avoid treatments that may be painful and potentially futile. For those in this group that require surgical lengthening because of contractures, the authors routinely use postoperative bracing with AFOs in an attempt to decrease the risk for recurrence.

CONCLUSION

Toe walking can be because of an underlying pathology, but most cases are idiopathic. A careful history, clinical exam, and selective diagnostic testing can be used to determine the cause of toe walking. There is limited comparative research to guide selection of treatment methods, but serial casting and surgical lengthening appear to have the most evidence. Further prospective comparative studies are required to better define optimal treatment for toe walkers.

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Conflicts of interest

There are no conflicts of interest.

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- ▪ of special interest
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