CME Article

Evaluation and Treatment of the Child with Tiptoe Gait

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Target Audience

This CME activity is intended for physicians, medical students and nurse practitioners. Pediatric orthopaedic specialists will find this information especially useful.

Learning Objectives

After completion of this article, the reader should be able to:

1. *create a differential diagnosis of early-onset versus late-onset tiptoe gait.*
2. *discuss methods for differentiating idiopathic toe walking from mild spastic diplegic cerebral palsy.*
3. *measure static Achilles contracture.*
4. *review various orthopaedic treatment modalities for dynamic and static Achilles contractures.*

Abstract

This review article describes for the primary care physician a simple four-step approach for initial evaluation of the child with tiptoe gait. First, the clinician must establish whether tiptoe gait is early-onset or late-onset. Tiptoe gait beginning at initial ambulation is almost always due to idiopathic toe walking or to mild spastic diplegic cerebral palsy, or less commonly to a psychological problem such as autism. Second, inquiry into family history will provide a positive response for about 40% of idiopathic toe walkers and may be positive for certain hereditary neuromuscular disorders. Third, the physician must look for abnormal neurologic findings. Late-onset tiptoe gait is virtually always due to neuromuscular disorders, such as Charcot-Marie-Tooth neuropathy, muscular dystrophy, or spinal cord anomalies. Finally, the physician must check for the presence of Achilles contractures since the orthopaedic management of tiptoe gait primarily depends upon the severity of static equinus contractures.

Key words: Tiptoe gait, etiology, spastic diplegia, idiopathic toe walking

It is not uncommon for the primary care physician to evaluate children with tiptoe gait. The physician may reassure parents that normal toddlers sometimes display intermittent tiptoe gait when they are first learning to ambulate. However, a more mature heel-toe gait pattern should become consistent by the age of two years.[1,2] If tiptoe gait persists, the physician should look for an explanation other than "immature gait pattern" of the young toddler.

When tiptoeing is *unilateral*, etiologies commonly include leg length discrepancy, spastic equinus (stroke, hemiplegia) and Achilles tendinitis. The author has cared for children with rare causes of unilateral tiptoe gait, including venous malformations in the calf musculature[3], linear scleroderma at the ankle[4], and hysterical conversion reaction.[5] In this review article, the author will discuss only *bilateral* tiptoe gait, for which common differential diagnoses are listed in Table 1. To begin the evaluation of the child with bilateral tiptoe gait, the physician must answer the following four questions:

Question 1: When did the tiptoe gait first appear?

This question narrows the differential diagnosis into two groups: "early-onset" and "late-onset" toe walking. Early-onset toe walking implies that the child began walking

on tiptoes within several months of initial independent ambulation. With few exceptions, the differential diagnosis of early-onset toe walking includes idiopathic toe walking (ITW) versus mild spastic diplegic cerebral palsy (CP). Less commonly, early toe-walking may be due to learning disabilities or psychiatric conditions, such as childhood autism. Surprisingly, it is not always a simple matter to distinguish among these three diagnostic categories since all three are commonly associated with speech or motor developmental delay and tight Achilles tendons.[6-10]

Late-onset tiptoe gait implies a well-established period of normal heel-toe gait followed by the appearance of toe walking. This is virtually always due to a neuromuscular abnormality, so prompt evaluation by a neurologist is advisable to look for spastic conditions, spinal cord anomalies, peripheral neuropathies, or muscular dystrophies.

Question 2: Is there a family history of toe walking?

For early-onset toe walkers, a positive response to this question makes a diagnosis of ITW likely. A positive family history is rare in CP cases, while roughly 40% (range, 10-70%) of children with ITW exhibit a positive family history of tiptoe gait.[6,7,11-13] For late-onset toe walkers, this question can also assist with diagnosis since about 50% of patients with Charcot-Marie-Tooth neuropathy or Duchenne muscular dystrophy will present with a positive family history.[14,15]

Question 3: Is the neurological exam abnormal?

This is the most difficult of the four questions to answer, but it is crucial for differentiating among the various etiologies of toe-walking. As a "diagnosis of exclusion", ITW can only be considered after determining that the neurological and behavioral examinations are normal. The physician must inquire about birth history, developmental history, and problems with learning or behavior. The child's gait must be analyzed clinically, and the shoes checked for abnormal wear. The back must be examined for spinal deformity or for midline skin markings (nevus, sinus tract, hair patch) which might signal an underlying spinal cord malformation. The upper motor neuron (central nervous system) function must be checked for asymmetrical or pathologic hyperreflexia, such as Babinski, stretch reflex, clonus, abdominal reflex, or ataxia. The peripheral nervous system is examined for sensation and for motor strength, particularly noting voluntary strength of ankle dorsiflexors. Acquired or progressive foot deformities, such as equinocavovarus, are suggestive of muscle imbalance caused by central or peripheral nervous system abnormalities.

After age 12 months, the presence of upper motor neuron pathologic reflexes (such as Babinkski) is highly suggestive of a spastic condition.[16] While cerebral palsy is the most common cause of childhood spasticity, there are many less common and progressive neuropathic conditions which may result in acquired spasticity, such as multiple sclerosis.[17] Consultation with a neurologist will usually be necessary to confirm the diagnosis. Psychiatric evaluation may also be necessary if a behavioral or psychological condition is suspected. Ideally, the orthopaedic surgeon should be consulted after the underlying cause of toe walking has been diagnosed definitively. Only by knowing the etiology and prognosis can the orthopaedic surgeon formulate optimal treatment.

Question 4: Are the Achilles tendons tight?

Kinematic studies show that approximately 10° of ankle dorsiflexion (DF) occurs during normal gait[2,18]; however, DF to at least 0° allows children to ambulate with a relatively normal heel-toe gait. Because the gastrocnemius muscle crosses the knee joint posteriorly, the proper way to measure gastrocsoleus contracture is to estimate ankle DF with the knee fully extended and with the talonavicular (midfoot) joint stabilized in anatomic position. If the midfoot is allowed to pronate, the measurement of ankle DF will be falsely increased 5-10° due to DF of the forefoot relative to the hindfoot.

Both early-onset and late-onset toe walkers may have static (tight) or dynamic (overactive) gastrocsoleus contractures. Therefore, the degree of equinus contractures does not narrow the differential diagnosis. However, in the absence of Achilles contracture, children with ITW can walk normally upon command.

Table 1 - Common Etiologies of Bilateral Tiptoe Gait

Early–Onset
- Idiopathic tiptoe walking
- Spastic diplegic cerebral palsy
- Psychological: autism, learning disorders

Late–Onset
- Spastic conditions: cerebral palsy, multiple sclerosis
- Hereditary motor sensory neuropathies: Charcot-Marie-Tooth
- Muscular dystrophies: Duchenne
- Spinal cord anomalies: tethered cord, syrinx, tumor

This is less likely for neuropathic conditions. The most important reason to measure Achilles contracture is for purposes of treatment following diagnosis. Regardless of etiology, the presence of fixed equinus contractures typically requires treatment and justifies referral to the orthopaedic surgeon. The typical goal for surgical correction of gastrocsoleus contracture is to restore DF to 5°. Orthopaedic treatment for each diagnostic category is discussed below.

Early-Onset Tiptoe Gait

Idiopathic Toe Walking

There are several retrospective studies which attempt to review ITW, its natural history, and its response to various treatment modalities.[6,7,19-23] Gait and balance are normal other than the persistence of tiptoe (equinus) position throughout the gait cycle.[24] The severe dynamic equinus is out of proportion to the mild static Achilles contractures. Children with ITW frequently stand in place with the heels on the ground.

ITW can be classified into two forms—with static Achilles contracture or without. Children in the latter group have passive ankle dorsiflexion above neutral with the knees extended. These children can walk normally for brief periods when asked to do so. For such children, there is little evidence that ITW causes long-term damage to the foot or leg. Furthermore, there is growing consensus that ITW rarely resolves with conservative treatments such as casting, stretching, or bracing.[6,7,20] Therefore, for children with ITW and with ankle DF above neutral, a reasonable treatment may be observation, with the expectation that the toe-walking will gradually diminish to the point that it becomes virtually unnoticeable.[20,21]

For parents who demand treatment, serial casting for approximately six weeks has been shown to provide transient weakening of the calf muscles with short-term improvement in tiptoe gait.[11,25,26] However, the lack of long-term improvement makes it difficult to justify the cost and risk of casting. Injection of botulinum-A toxin into the calf muscles is an unproven treatment modality for ITW, but studies are underway.

Children with ITW may present with, or gradually develop, significant Achilles contractures with loss of ability to DF the ankles to neutral.[7,20] Idiopathic toe walkers with Achilles contractures are probably best treated surgically. This typically involves bilateral gastrocnemius recession lengthening to achieve 5° ankle DF. Lengthening of the Achilles tendon may be necessary if the plantarflexion contracture exceeds 15°.

Gastrocnemius recession is generally considered safer, due to lower risk of overlengthening. Postoperatively, patients ambulate in short leg walking casts for six weeks, followed by gradual return to sports. Even with appropriate surgical treatment, at least 30% of children with ITW will develop partial recurrence of tiptoe walking.[6,7]

Spastic Diplegic Cerebral Palsy

With spastic diplegia and developmental delay, the arms have abnormal posture, and there is excessive crouch (knee flexion during stance). The ankles are in minimal plantarflexion, and much of the tiptoeing is related to knee flexion.[24,27]

While this appearance may be typical, the physician should be aware that some children with mild spastic diplegia ambulate with exactly the same posture as the ITW child.

In spastic CP, toe walking is usually early-onset, but may occur later as static contractures develop. Family history of CP is usually negative unless it is related to complications of multiparity.

Examination shows abnormal upper motor neuron reflexes such as clonus, Babinski, or stretch reflex. Electromyographic methods to distinguish ITW from CP are of limited usefulness, but can sometimes be helpful.[13,26-29]

Dynamic EMG's may show abnormal co-contractures of antagonist muscles during gait or during voluntary knee extension.[30]

As in children with ITW, children with spastic CP may have static or dynamic Achilles contractures. Dynamic contractures can be controlled with ankle-foot orthoses (AFO's) worn during daytime hours[31,32].

For patients who cannot tolerate braces due to severe hypertonicity, the dynamic gastrocsoleus contracture can be temporarily reduced with botulinum toxin injections into the calf muscles.[33,34] Static equinus contractures may require gastrocnemius or Achilles lengthening to achieve neutral ankle DF.[35-37]

Postoperative correction can then be maintained with AFO's. Surgical lengthening of hip flexors or hamstrings may be necessary to decrease toe walking caused by crouching.[35]

Psychological Conditions

Although less common than in ITW or CP, tiptoe walking is sometimes associated with learning disabilities, mental retardation, and psychiatric conditions such as childhood autism.[8,9,38-40]

Weber[10] theorized that autistic toe-walking is due to an arrested stage of walking motor development.

Children with learning and behavioral disorders usually have early-onset tiptoe gait even though they may be late to achieve the milestone of independent ambulation. Psychiatric conditions may have multifactorial inheritance, so family history is occasionally positive.[41] Examination may show cognitive and speech delay, behavioral outbursts, poor social interaction, and attention-deficit. Upper motor neuron signs are usually normal or equivocal. Patients may require physical, occupational, or speech therapy as well as psychiatric care and medications to improve attention or behavior. The orthopaedic surgeon may find that behavioral issues make bracing, casting, and surgical treatment difficult and unpredictable.[10]

Late-Onset Tiptoe Gait

Charcot-Marie-Tooth (CMT) Neuropathy

The hereditary motor sensory neuropathies, which include CMT, may result in late-onset toe walking, usually after age six years. CMT (HMSN type-1) is usually inherited as an autosomal dominant trait, so family history will be positive for about 50% of patients.[14] Neurological examination shows weakness of ankle dorsiflexor and peroneal muscles with normal strength in the calf muscles and posterior tibialis. Patients gradually develop symmetrical cavus feet, followed by heel inversion and clawing of the toes.[42,43] The tiptoe gait is due more to forefoot equinus than ankle equinus. A definitive diagnosis can usually be established with nerve conduction testing or nerve biopsy.

Orthopaedic treatment depends upon the severity of clawfoot deformity. Splinting with AFO's may initially slow the development of contractures. Eventually, many patients require surgical treatment, such as transfer of the posterior tibialis tendon to the dorsum of the foot, plantar fasciotomy, and transfer of the peroneus longus to the peroneus brevis.[42,43] Rigid cavovarus foot deformity may require osteotomy of the calcaneus and metatarsals.

Duchenne Muscular Dystrophy

Children with Duchenne muscular dystrophy, the most common form, have late-onset toe-walking which may begin by age 3-5 years.[44] Duchenne dystrophy is caused by an X-linked recessive mutation in males, so the family history may be positive for about 50% of patients.[15] Examination shows that the child is clumsy with proximal muscle weakness (positive Gower's sign) and normal Achilles reflexes. Fibrofatty and inflammatory infiltration of the gastrocsoleus muscles results in marked calf pseudohypertrophy with progressive static Achilles contractures. Left untreated, the feet will usually develop severe rigid equinocavovarus deformities. The diagnosis is confirmed with elevated serum CPK, EMG findings, and muscle biopsy.

Fulltime AFO bracing is initially utilized to slow progression of foot contractures. As equinovarus deformity progresses, surgical treatment may become necessary to enhance brace tolerance. Surgery typically consists of bilateral gastrocsoleus lengthening with transfer of the posterior tibialis tendon to the dorsum of the foot.[45-47] Surgery is more safely performed prior to development of severe cardiac and pulmonary dysfunction. Even with appropriate management of Duchenne dystrophy, walking ability is usually lost by early teenage years. In the milder Becker and limb-girdle dystrophies, nonsurgical management of contractures is usually possible until adulthood.

Spinal Cord Anomalies

Spinal cord anomalies may lead to late-onset tiptoe walking due to foot deformity, muscle imbalance, or gastrocsoleus spasticity. In tethered spinal cord syndrome, the cord becomes stretched during growth, typically due to a tight filum terminale or due to a split cord malformation (diastematomyelia).[48,49]

In syringomyelia, myelopathy occurs due to the development of a cyst within the cord.[50] These conditions are rarely hereditary. Most patients complain of back pain, leg weakness, or sensory loss. At least 50% of patients with tethered cord will demonstrate a hair patch, sinus tract, or nevus in the midline back. Cord anomalies may result in scoliosis and abnormal reflexes.[51] Progressive asymmetrical cavus foot deformities commonly develop during childhood. The spinal MRI best demonstrates the tip of the conus located caudal to its normal position at the L1-2 interspace.[52]

MRI is also excellent for establishing a diagnosis of spinal cord tumor or syrinx. Orthopaedic treatment of toe walking must be individualized depending upon the degree of foot deformity and the preservation of motor function following treatment of the underlying condition.

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Stricker, S *Int Pediatr* 2006;21(2)91-96.

QUESTIONS

1. Which of the following conditions is most likely to cause *early-onset* tiptoe gait?

A. idiopathic toe walking
B. multiple sclerosis
C. Duchenne muscular dystrophy
D. Charcot-Marie-Tooth disease (HMSN-1)
E. tethered spinal cord

2. Which of the following best distinguishes cerebral palsy from idiopathic toe walking?

A. premature birth
B. developmental delay
C. negative family history
D. positive Babinski
E. mild Achilles contracture

3. Which feature accompanies toe-walking in children with Duchenne muscular dystrophy?

A. ankle clonus
B. calf pseudohypertrophy
C. leg muscle co-contractures on dynamic EMG
D. scoliosis
E. passive ankle dorsiflexion >10°

4. Which of the following is characteristic of idiopathic toe walking?

A. poor balance
B. crouched gait with tight hamstrings
C. severe dynamic equinus contractures
D. leg muscle co-contractures on dynamic EMG
E. permanent resolution following brace or cast treatment

5. Which of the following causes of toe-walking is *most* likely to have a positive family history?

A. autism
B. idiopathic toe walking
C. spastic diplegic cerebral palsy
D. tethered spinal cord
E. syringomyelia

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