Spastic Foot and Ankle Deformities: Evaluation and Treatment

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KEYWORDS

• Spastic foot and ankle deformities • SPLATT • Equinovarus

KEY POINTS

- Spastic equinovarus is the most common foot and ankle deformity following cerebral vascular accidents.
- Dynamic electromyogram is the most important preoperative tool in planning surgical correction.
- Surgery should be considered when the patient is at a plateau of neurologic improvement with goals of producing a balanced, functional foot, minimize brace wear, pain relief, callus/ulcer prevention, facilitating hygiene, and/or positioning in a wheelchair.
- Operative intervention has been shown to improve ambulatory status and decrease necessity of brace wear.

INTRODUCTION

Traumatic brain injuries (TBIs) and strokes, or cerebral vascular accidents (CVAs), can have profound effects on both the individual and society as a whole. The most recent American Heart Association statistics report the annual adult incidence of CVAs in the United States to be nearly 800,000, of which more than half survive,[1–3] thereby making it the leading cause of serious long-term disability in the United States.[1,4] TBIs are even more common, with an incidence of 1.5 million per year.[5] In addition to frequent cognitive deficits, residual musculoskeletal disabilities are common, with 30% unable to ambulate without assistance.[1] TBIs and CVAs are therefore the leading causes of adult spastic foot and ankle deformities.[4,6] Similar deformities are also common in cerebral palsy with similar treatment strategies, although most are addressed in childhood.

Disclosures: None (B.W. King and D.J. Ruta); I and/or my spouse/significant partner/immediate family member have the following financial relationships: Smith and Nephew – consultant, advisor; Saunders/Mosby-Elsevier – financial support from publisher (T.A. Irwin).
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Foot Ankle Clin N Am 19 (2014) 97–111
http://dx.doi.org/10.1016/j.fcl.2013.10.007 **foot.theclinics.com**
1083-7515/14/$ – see front matter

CAUSE

Spastic foot deformities are caused by selectively increased muscle tone that disturbs the physiologic agonist-antagonist balance of the lower extremity musculature. Head or spinal cord trauma, ischemia, hemorrhage, demyelinating disease, infection, and inflammatory diseases are all potential causes.[4,6,7] Spasticity stems from an upper motor neuron disruption along the descending corticospinal tract, with variability in both specific location and cause.[4,8] For instance, a CVA in the distribution of the anterior cerebral artery results in hemiparesis or hemiplegia, and there is even greater severity with brainstem involvement.[4,6] The result is a loss of the normal inhibitory signal from interneurons, producing hyperexcitability of motor neurons. Exaggeration of the muscle-tendon stretch reflex occurs, causing a velocity-dependent increase in muscular tone.[4] In the lower extremity, the extensors are typically more affected, in contrast to greater flexor involvement in the upper extremity.[4] The spasticity can also be affected by temperature and emotion.[4,9]

Cerebral palsy is a static encephalopathy secondary to a nonprogressive insult to the developing brain. The resultant musculoskeletal deformities, however, are progressive, in that the growth of bones outpaces that of associated tendons.[4] With increasing age and growth, the tendons are no longer able to compensate, and permanent structural deformities develop. The previously supple deformities are no longer correctable with manipulation and become static deformities.[9] Typically, the clinical picture of spastic cerebral palsy that most commonly affects the foot and ankle is overactive ankle plantar flexors and ineffective dorsiflexors, although exact presentation varies.[4,9]

CLINICAL COURSE

After an insult to the central nervous system, it can take days to weeks for spasticity to develop.[4,6] Following an acute injury, there is often a brief period of flaccid paralysis and hypotonia. Deep tendon reflexes are also diminished during this time, which can last from a few hours to weeks.[6,8] Elevated muscle tone and hyperreflexia then develop, although this is followed by a long period of spontaneous recovery. During this time, spasticity can improve with the return of strength, coordination, and sensation. Cognitive function also improves throughout this period, as a new baseline is established. Following CVA, recovery continues for 6 to 9 months, whereas TBIs continue to improve for about 18 months.[2,6] Recognition of the duration and spontaneous recovery potential is important for evaluation and surgical planning.

Initially, spastic-type cerebral palsy also presents with hypotonia and weakness, which progresses to hypertonia and spasticity.[4] Patients have pain with ambulation, shoe wear, and use of orthotic devices and demonstrate combinations of tripping, in-toeing, and out-toeing.[9]

CLINICAL CONDITIONS

Spastic equinovarus is the most common foot and ankle deformity both following CVA[2,3] and also in cerebral palsy (**Fig. 1**).[10] It usually results from spasticity of the plantarflexor and invertor muscles, combined with a deficiency of their associated antagonists.[2] Specifically, spasticity can be seen in a combination of several different muscles including gastrocnemius, soleus, anterior tibialis, posterior tibialis, flexor hallucis longus (FHL), and flexor digitorum longus (FDL), while associated weakness can be seen in the peroneals.[4,5,8,9,11–13] In TBI and CVA patients, varus is typically caused by spasticity of tibialis anterior, whereas tibialis posterior often produces the deformity