triceps surae clonus after tibial nerve hyponeurotisation. Because partial, this hyponeurotisation allows preservation of fibularis muscles voluntary control and hindfoot stability.

Our present trend consists in systematically searching fibularis spasticity and including its treatment in therapeutic planning. We do not hesitate to propose fibularis longus hyponeurotisation in surgical program, simultaneously to other procedures against equinovarus deformity and/or toe claws.

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CO52-004-e
Lesser toes deformities in neurologic disease
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Targets.— Lesser toes deformities in neurologic disease are either the consequence of a muscular imbalance, either a mechanism of compensation. They occur in pyramidal lesion with spasticity, or in extra-pyramidal disease with dystonia. The treatment, after a clinical investigation, relies on shoe’s adaptation, toxin therapy, surgery of spasticity or soft tissues, and rehabilitation.

Clinical balance.— It needs an articular examination of the all leg, a neuromuscular balance of the involved muscles and antagonisms, an examination superficial and proprioceptive sensibilities and shoe’s inspection. This aim is to determine the muscle responsible of the toe deformity. Neuromuscular blocks can help us.

Therapeutic methods.— Shoe’s adaptation is essential. Without muscular retraction, botulin toxin is the best treatment of lesser toes deformities. With retraction, the surgical treatment relies more and more on mini invasive surgery with combination of soft tissues and bone procedures. The surgery is a personalized surgery. The solution is chosen regarding to reducible or fixed deformity, proximal or distal deformity. Tendon lengthening or selective neurotomy can be associated.

Indications.— A treatment is necessary if lesser toe deformity become aching, with shoe’s difficulties, skin lesions or walking impairment. A treatment by toxin or surgery on flexor digitorum longus involve a real evaluation of extensor digitorum. Do not take in account of them, will lead to an aggravation of deformity.

Conclusion.— Lesser toe deformities are frequently observed in neurologic diseases, but is rarely alone. The treatment must include physiotherapy, specific shoes, toxin injection and eventually surgery.

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Which is the best strategy for Extensor hallucis longus and Extensor digitorum communis dystonia?
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Keywords: Dystonia; Extensor hallucis longus; Extensor digitorum communis; Botulinum toxin; Tendon lengthening; Deep peroneal nerve hyponeurotisation Spastic hemiplegia or diplegia is often responsible for troubles with great or small toes: impingement between the dorsal aspect of toes and the shoe, difficulties in slipping the foot into the sock and/or the shoe, spontaneous pain due to spasm, because of dystonia of Extensor hallucis longus (EHL) with or without that of Extensor digitorum longus (EDL).

EHL and EDC dystonia can be considered as an attempt of the patient to recruit these muscles to compensate the inefficacy of the Tibialis anterior muscle; it then should appear only at non-bearing phase of gait. When it is permanent, or when it occurs only during weight bearing; this explanation is less acceptable. When it does not occur at each step, or when its importance varies, it is considered as a functional dystonia.

Such a dystonia can be increased by toe claw surgical treatment, either by hyponeurotisation, or by lengthening or tenotomy of great and small toes. This dystonia can be treated by repetitive injections of botulinum toxin or by surgery: lengthening of the tendon(s), EHL ± EDC hyponeurotisation. Advantages and drawbacks of each technique are discussed. Botulinum toxin use needs repetitive injections; its efficacy can decrease with time.

Tendon lengthening can be done inside the muscle belly (intramuscular lengthening) or in the tendon itself at the dorsal aspect of the foot. Its magnitude is difficult to choose, exposing the patient either to hypocorrection with less important persisting dystonia, or hypercorrection favouring toe claw. Partial division of the branches of the deep fibular nerve to the EHL ± the EDC is another interesting surgical possibility; nevertheless it necessitates a long approach of the anterior compartment of leg; it can be combined with other surgical procedures on hindfoot and/or forefoot.

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Causes of flat foot valgus original proposal central and neurological therapy
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The valgus flat foot deformity is common in patients with neurogenic hypertension center. Functional impairment is well tolerated long. Patient complaints are:

– support internal pain on the head of the talus, or submalleolar side by pinching of the peroneal tendons;
– sensation of instability of the foot support phase, and;
– the deformations of the forefoot.

We describe the different causes of valgus flat foot of neurological origin: retraction or hypertonia of the calcaneal tendon (Achilles tendon) with rocker block calcaneofibular pedal, muscle hypertonia valgisants deficit and the tibiialis posterior. Different causes can accumulate and worsen then strain. The clinical and radiological precise;

– etiology;
– the impact at the forefoot (especially the deformation of the hallux), the talocrural joint (search tibriofibular diastasis possible) and;
– the impact on the most proximal knee joint.

Therapeutic solutions related to both the treatment of the case, but also the deformation at the midfoot. Solutions to address the collapse of the medial arch are limited:

– by making an orthotic insole;
– or surgical repositioning of the calcaneofibular pedal block by performing a fusion of torque.

There is no tendon transfer possible to correct this distortion and rebalancing the forefoot. We present the procedures and therapeutic decision tree. Deformation valgus flat foot has many causes identified, which can offer a prevention through early management of hypertonia deforming in any priority that the gastrocnemius muscle.

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