long abord de la loge antérieure de la jambe, qui peut être associé à d'autres gestes chirurgicaux sur l'arrière pied sur les orteils.

http://dx.doi.org/10.1016/j.rehab.2013.07.998

CO52-006-f

Causes du pied plat valgus d'origine neurologique centrale et propositions therapeutiques

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Le pied plat valgus est une déformation fréquente chez les patients présentant une hypertonie d'origine neurologique centrale. Le retentissement fonctionnel est longtemps bien toléré. Les plaintes des patients sont :
les douleurs d'appui interne sur la tête du talus, ou sous-malléolaires latérales par pincement des tendons fibulaires ;
la sensation d'instabilité du pied à la phase d'appui, et ;
les déformations de l'avant-pied.
Nous présentons les différentes causes du pied plat valgus d'origine neurologique : rétraction ou hypertonie du tendon calcanéen (tendon d'Achille) avec bascule du bloc calcanéo-pédieux, hypertonie des muscles valgisants et déficit du muscle tibial postérieur. Les différentes causes peuvent se cumuler et aggravent alors la déformation. Le bilan clinique et radiologique précise :
l'étiologie ;
le retentissement au niveau de l'avant pied (surtout la déformation de l'hallux), de l'articulation talo-crurale (recherche d'un éventuel diastasis tibio-fibulaire) ;
et l'impact proximal surtout sur l'articulation fémoro-tibiale.
Les solutions thérapeutiques concernent à la fois le traitement de la cause, mais aussi celui de la déformation au niveau du médio-pied. Les solutions pour corriger l'effondrement de l'arche médiale sont limitées :
orthétique par confection d'une semelle orthopédique ;
ou chirurgicale par repositionnement du bloc calcanéo-pédieux en réalisant une arthrodèse du couple de torsion.
Il n'y a pas de transfert tendineux possible pour corriger cette déformation et rééquilibrer l'avant pied. Nous présentons les modalités et un arbre de décision thérapeutique.
La déformation en pied plat valgus a des causes bien identifiées, qui permettent de proposer une prévention par une gestion précoce des hypertonies déformantes, en toute priorité celle du muscle gastrocnémien.

http://dx.doi.org/10.1016/j.rehab.2013.07.999

Oral communications

English version

CO52-001-e

Correction of equinus foot of the patient with cerebral palsy

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Unknown abstract.

http://dx.doi.org/10.1016/j.rehab.2013.07.1000

CO52-002-e

Restoration of the dorsal flexion of the foot in equinovarus deformity

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Keywords: Equinovarus deformity; Foot dorsal flexion restoration; Tendon transfer

Restoration of the dorsal flexion of the foot provides good functional results in neurologic equinovarus foot.
Dynamic varus depends on the imbalance between weak eversor muscles and spastic inversor muscles, such as tibialis anterior and tibialis posterior, whose respective roles are sometimes difficult to clinically appreciate.
Different surgical procedures can be used; they need beforehand a large enough passive dorsal flexion, which can be restored by tendinous lengthening during the same procedure, and absence of fixed varus or its correction by triple arthrodesis.
After giving explanation to the patient about the interest of this kind of functional surgery, the choice of the procedure depends on several factors: availability of tendon for transfer, their functional status, quality of motor control, potential spasticity of the transferred muscle which becomes useful after the transfer, quality of bony and/or tendinous fixation and, in last, surgeon's experience!
Three transfers are usually performed:
– the tibialis anterior tendon can be transferred on the lateral cuneiform bone. It is possible to split the tibialis anterior tendon and to fix the lateral half tendon onto the cuboid or the fibularis brevis tendon;
– the tibialis posterior tendon can be transferred through the interosseous membrane to the lateral cuneiform bone. This kind of transfer is rarely used in central neurologic patient. Its efficacy is partial for the gait improvement;
– the fibularis brevis tendon can be cut proximally and sutured onto the tibialis anterior tendon as proposed by Bardot.
As bone fixation is often not strong enough in osteoporotic patient with a important risk of loosening, it is then better to use tendon-to-tendon suture.
A postoperative cast is needed during three to five weeks according to teams and is followed by rehabilitation in center or at home

http://dx.doi.org/10.1016/j.rehab.2013.07.1001

CO52-003-e

Fibularis muscles spasticity: Is it troublesome? How can one manage it?

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Keywords: Fibularis longus; Fibularis brevis; Spasticity; Clonus; Functional surgery; Neurotomy; Hyponeurotisation; Botulinum toxin

Spasticity of the lateral compartment of leg is not well known: no reference in PubMed with the following.
It is responsible for clonus of fibularis brevis and/or fibularis longus. The first one works in the transverse plane, it is not always troublesome for the patient; the second works in the sagittal plane, it can be confused with a triceps surae clonus, even after tibial hyponeurotisation. Fibularis muscle hypertonia plays also a role in valgus deformity in neurologic patients.
When troublesome, this spasticity can be treated by repetitive botulinum toxin injections or superficial peroneal nerve hyponeurotisation.
The anatomical bases of this hyponeurotisation have been studied by dissection of five formaline preserved legs. The most proximal branches to the fibularis longus arose from the common peroneal nerve.
Operating technique is described: posterior lateral approach, detachment of posterior head of fibularis longus or opening of its belly, to follow the superficial peroneal nerve up to the fibularis brevis. A neurostimulator is mandatory to correctly identify the branches of the common peroneal nerve to the fibularis longus.
Early results are described apropos of four cases (2M, 2F, follow-up: 6–28 months): disappearance of preoperative clonus in three patients, but reappearance in one because of intense sprouting confirmed by giant action potentials at electromyography, in a patient who also experienced recurrence of

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.

http://dx.doi.org/10.1016/j.rehab.2013.07.1002

CO52-004-e

Lesser toes deformities in neurologic desease

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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.

http://dx.doi.org/10.1016/j.rehab.2013.07.1003

CO52-005-e

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.

http://dx.doi.org/10.1016/j.rehab.2013.07.1004

CO52-006-e

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 tibialis 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 tibiofibular 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.

http://dx.doi.org/10.1016/j.rehab.2013.07.1005