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Trauma Tips & Tricks: Peroneal Nerve Palsy

Part II

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Non-Operative Treatment

The goal of non-operative treatment of peroneal nerve palsy is to provide an ankle position in swing that is compatible with heel-toe gait, that reproduces the first and second rocker to the extent necessary to provide deceleration of the forefoot during early stance to prevent a “foot slap”. Additionally, because the ankle evertors are also paralyzed, a successful intervention should provide sufficient ankle stability to prevent inversion ankle injuries and sufficiently control the tendency of the hindfoot to tilt into varus when it is loaded. Finally, non-operative treatment should prevent contracture of the triceps surae, as they are un-checked by denervated pre-tibial muscles.

As such, non-operative therapy will consist of a stretching program of the triceps surae, and an ankle foot orthosis (AFO). For the stretching program, we recommend daily stretching (5 times 30 seconds) of both the gastrocnemius and soleus muscles. For an orthosis, we generally utilize a low resistance carbon fiber AFO which allows some deformation into plantarflexion, but has sufficient elastic recoil which allows the foot to spring back into neutral dorsi/plantarflexion. This brace treatment depends on the stretching program to prevent contracture formation which positions the foot in equinus. Occasionally, patients will find this brace to be too flexible to sufficiently control the tendency of the foot to invert. If this is the case, either a brace with a more rigid material, or high top shoes or boots is recommended.

Many patients with peroneal nerve palsy are quite pleased with non-operative therapy. It is our practice to treat this set of patients as such indefinitely assuming the process is not due to a reversible process in which delay would compromise viability of the nerve.

Surgical treatment

Surgical treatment of peroneal nerve palsy consists of four basic types of operations: 1. Nerve decompression, 2. Nerve reconstruction, 3. Tendon transfers, and 4. Ankle fusion. The choice of an operation will depend on the duration of the injury, the type of pathology involved, the patient’s lifestyle, and other factors.

Nerve Decompression

Nerve decompression is possible when the nerve palsy is early in the course of pathology, assuming the mechanism of injury is compressive. If a compressive lesion (tumor, hematoma, or surgically induced swelling) has occurred, and the process is relatively new, it can be postulated that the clinical symptoms are due to a compression neuropraxia. If a compression neuropraxia is suspected, then treatment should not be delayed. The nerve should be decompressed; the most common site of compression is as the nerve courses around the fibular neck. However, intra operative EMG/nerve stimulation should be utilized to determine the site of compression and decompress the nerve proximal to this.

Nerve reconstruction

Nerve reconstruction is favored as a next possible procedure if EMG studies at 3-6 months show no recovery. Stretch neuropraxias from knee dislocations, or arthroplasty may be generated. In these cases, no specific compression may be present. Intraoperative EMG and nerve conduction studies may be performed. If action potentials are noted across the nerve, and the nerve is continuous, decompression alone may be performed⁴. In cases in which the nerve is non-conducting, but continuous the decision is more complex. The nerve may either be decompressed and observed, or resected and cable grafted. If cable grafting is selected, most often a sural nerve graft is selected. Recovery rate is strongly associated with graft length. A large series showed poor outcomes with less than 50% recovery rate with grafts longer than 6 cm^{4,5}. If the nerve is lacerated or discontinuous, the decision becomes simpler, the injured nerve ends are excised, and the nerve then cable grafted with a sural nerve autograft and nerve tube conduit.

Nerve reconstruction if successful offers a patient the greatest chance of an anatomically normal or near normal result with minimal residua (ankle numbness resultant from sural nerve harvest). However, for many pathologies in which the mechanism of injury is stretch, grafting is less successful owing to the large zone of injury.

Tendon transfers

Tendon transfers are a salvage procedure available to patients in whom decompression is not ideal due to the pathology involved in their injury, and nerve grafting is either not compatible with success due to the mechanism of injury or the duration of symptoms (ie the effector muscles are too atrophied to work even if they had a nerve supply). Many tendon transfers have been described for peroneal nerve palsy. All transfers use the “back to front” idea of transferring a flexor or flexor/ inverter to the dorsum of the foot for replacement of dorsiflexion function at the expense of one or more of the functions of the tendon to flex or invert joints of the foot. As such, tendon transfers are not possible in the case of a full sciatic palsy. EMG can distinguish between a common peroneal palsy and a sciatic nerve palsy.

Posterior tibial tendon transfer through the intraosseous membrane was developed over 100 years ago by Codvilla in 1899 and Putti in 1914⁵. Several authors have subsequently modified the technique to address some of the technical difficulties associated with the procedure. Namely the posterior tibial tendon is of insufficient length which can result in calcaneus position of the foot and difficulties with fixation on the dorsum of the foot. Though these issues with the procedure can be largely addressed with either tendon-tendon interface, interference screw, or indwelling suture button fixation, other issues exist with the technique also. Some authors have observed an acquired flat foot deformity resultant from the harvest of the posterior tibial tendon without replacement of its function³. This has led some authors to recommend subtalar fusion at the time of tendon transfer to prevent iatrogenic pes planovalgus. This procedure can be associated with over 80% return to brace free gait. However, it is important to note that the strength is 30% less than the contralateral limb.

Many authors believe that the replacement of function of the pre-tibial muscles is due to a tenodesis effect. Transfer of other posterior muscles such as the flexor digitorum longus and flexor hallucis longus has been discussed and described. These tendon transfers are intrinsically appealing because they remove a deforming force (clawing of the toes after paralysis of the extensor muscles) and potentially restore a lost function (dorsiflexion). These transfers have been described as the so called “Hiroshima” procedure for treatment of spastic equinovarus in cerebral palsy, and recently for flaccid peroneal nerve palsy⁵.

Additionally, if tendon transfer is to be considered, the patient must be assessed for an equinus contracture. If an equinus contracture exists, it is our practice to address the equinus contracture during the same surgical episode. We generally perform a silverskold test, in which the equinus contracture is assessed with knee flexion and knee extension. If the examination shows that the contracture is only present during knee flexion but disappears during extension, the gastrocnemius alone is tight and we perform a Strayer type zone III gastrocnemius recession. If the equinus contracture is

present in both flexion and extension of the knee, both muscles are tight and we perform a Zone II Baker type recession if the contracture is mild (10-20 degrees), and a Hoke percutaneous zone I tendon lengthening if the contracture is greater than 20 degrees with the knee in extension.

The patient is maintained in a cast for six weeks and a solid brace for an additional six weeks after tendon transfer. If a subtalar fusion is included a non-weight bearing cast is utilized for six weeks followed by a weight bearing cast for six weeks. Following this, retraining of the tendon transfer can take place with physical therapy. Rehabilitation is an integral part of the procedure, and any tendon transfer will fail to produce the desired results without a good therapist. We recommend stretching the triceps surae and imagery based retraining of the tendon transfer, as the patient must retrain the tendon to work in a way it is not accustomed to working. Functional electrical stimulation can be helpful in this regard.

Ankle Fusion

Ankle fusion is reserved for patients who are unable to tolerate bracing, and have either failed all or are ineligible for other modalities (tendon transfer, nerve graft, decompression). The goal of the procedure is to effectively fix the foot in a ninety degree position to the tibia. This may be accomplished through ankle (tibiotalar) fusion or pan talar fusion depending on other pathology involved.

Ankle fusion is a last resort because it takes all motion from the ankle joint, has possible complications of non-union as well as hardware related complications, and adjacent segment arthritis. Additionally, forefoot cavus may still be present and will be poorly compensated by a stiff ankle. A negative heel and a metatarsal rocker may then be necessary to restore the rockers of the foot.

Most peroneal nerve palsy can be treated either operatively or non-operatively without resorting to ankle fusion.

Conclusion

Peroneal nerve palsy is a common lower extremity injury after trauma. It can be successfully managed by non-operative or operative means, but requires a thoughtful approach by the treating clinician.

References

1. Qian Dong, Jon A. Jacobson. Entrapment Neuropathies in the Upper and Lower Limbs: Anatomy and MRI Features. *Radiology Research and Practice*. Volume 2012 (2012), Article ID 230679, 12 pages
2. Jenkins DB. The Leg. *Hollinhead's functional anatomy of the limbs and back*. 8th ed. Philadelphia: WB Saunders; 2002.
3. Rockwood CA, Green DP, Bucholz RW, Heckman JD. Rockwood, Green, and Wilkins' Handbook of Fractures. 6th ed. *Philadelphia, Pa.*: Lippincott Williams & Wilkins; 2006.
4. Chapman MW, Campbell WC. Chapman's Orthopaedic Surgery. 3rd ed. *Philadelphia, Pa.*: Lippincott Williams & Wilkins; 2001.
5. Mary Keenan, Scott H. Kozin, Anthony C. Berlet. *Manual of Orthopaedic Surgery for Spasticity*. Raven Press, 1993.