Role of Functional Electric Stimulation in Drop Foot

Aaron Bradley, DPM
Yusuf Opakunle, DPM

INTRODUCTION

Peripheral nerve deficits manifested in the lower extremity can have debilitating effects on a patient’s quality of life. The pathological process has an effect on both motor and sensory neurological control. This can have a lifelong impact, in some cases rendering a patient dependent on an assistive gait device. The leading cause of paralysis is stroke, followed by spinal cord injuries, and cerebral palsy (1). Other common neurological diseases such as polio and multiple sclerosis can have an effect on the lower extremity. Over the last few years novel treatments such as bio-implantable neuroprosthesis have been used to assist with gait (2). The key is to properly differentiate upper motor neuron (UMN) and lower motor neuron (LMN) involvement and determine the best treatment (3). This is especially relevant given the increasing number of patients in recent years presenting to a podiatric physician’s office with a chief complaint of drop foot.

PATHOPHYSIOLOGY

UMN is defined as the axonal pathway between the cortex and lower portion of the spinal cord. Any damage along the central nervous descending motor pathway within the internal capsule will cause immediate flaccid paralysis along the contralateral side. This initial hypotonia is referred to as spinal shock; within several days the typical signs and symptoms of UMN syndromes will begin to manifest. The most common UMN deficits seen in the field of podiatry are stroke, multiple sclerosis, and cerebral palsy (3).

LMN is defined as the lower motor cell bodies that arise from the brain stem or spinal cord anterior horn cells. Damage along the peripheral nerve tracts will affect the ipsilateral side immediately (3). Common pathologic processes seen in podiatry are spinal cord injuries, L4-L5 radiculopathy from herniated nucleus pulposus or foraminal stenosis, and peroneal peripheral neuropathy.

Although the physical presentation of a patient with the UMN and LMN can be distinguished, there are some similarities. Both UMN and LMN syndromes can present with weakness or inability to actively dorsiflex the ankle joint. This phenomenon of weakness of the anterior leg muscle group is known as foot drop. This is caused by deep peroneal nerve pathology (4). The anterior muscle group is a key component of clearing the foot during swing phase of the gait cycle.

Standard treatments for persistent foot drop involve ankle foot orthosis (AFO). This assistive device allows the foot an optimal position for proper stance phase clearance. The disadvantages of AFOs include discomfort, difficulty standing from a seated position, and contracture at the level of the ankle from lack of joint mobility. The alternative treatment consists of functional electrical stimulation (FES). The purpose of this neuroprosthesis is to stimulate the common peroneal nerve to achieve adequate dorsiflexion of the ankle joint. Most of the comparison studies have found FES therapy to have a superior effect on gait when compared to AFO therapy (5). We will discuss the most common etiological factors that lead to neurological deficits in the lower extremity and describe treatment modalities for foot drop.

CLINICAL MANIFESTATION

Many patients present with the classic symptoms demonstrating UMN or LMN syndromes. The key is to differentiate the patterns observed during the physical examination. The typical presentation for the UMN is spasticity or clonus, loss of fine motor movements, and normal sensation. These patients will demonstrate hypertonia of the deep tendon and pathologic reflexes such as Babinski, Gallant, and tonic neck. The muscle weakness is exhibited distally without atrophy. Alternatively, the LMN syndrome demonstrates hypotonic deep tendon reflexes and absent pathologic reflexes. Sensory deficits may occur and muscle weakness is exhibited proximally and/or distally. The LMN syndrome patient will display paralysis (3).

Foot Drop

LMN and UMN syndromes can both demonstrate neurological deficits in the lower extremity such as foot drop. On physical examination, the manual muscle test will be decreased during dorsiflexion at the ankle joint when compared to other planar movements. The characteristic gait patterns seen with foot drop are steppage and a foot slap due to weakness or paralysis of the anterior leg muscles. The steppage gait is appreciated due to greater knee flexion necessary to accommodate failure to dorsiflex. If the foot never leaves the ground the foot will drag and lead to tripping. The foot slap occurs during the initial contact phase allowing for the entire foot to hit the ground without any eccentric control from the anterior muscle group. There
will be a noticeable equinovarus foot deformity based on the plantar flexors over-powering the dorsiflexor muscles (2).

**DIAGNOSIS AND TESTING**

It is important to understand the entire physiological process of the disease; particular diagnostic tests and imaging such as plain films for traumatic injuries may be necessary. When appropriate, brain and spinal cord magnetic resonance images should be obtained. The use of ultrasound can be another assistive device to help determine the diagnosis. Particular laboratory values such as blood glucose, HbA1c, erythrocyte sedimentation rate, C-reactive protein level, blood urea nitrogen and creatinine levels, vitamin B12 level, and Lyme antibodies are helpful when evaluating the patient. Lastly, an electromyography and nerve conduction velocity study are important for studying the tone of the muscles (6).

**MANAGEMENT**

The purpose of current treatment is to keep the foot at a neutral 90 degrees during the gait cycle. If the foot remains in plantar flexion during the phases of gait it can lead to instability, falls, cadence issues, and energy expenditure (7). Different conservative treatments include AFO and functional electrical stimulation (FES). Surgical treatments include tendon transfers and ankle arthrodesis.

**AFO**

The most common therapy for patients demonstrating foot drop due to any type of neurological pathology is AFO. These devices have an immediate effect, which allow the foot to be in a neutral position during the gait cycle and reduce the patient’s fall risk. The comparison studies have found improvement in cadence and gait velocity (8). Although there is a large amount of research in the literature, it is difficult to extrapolate specifics due to a lack of randomized controlled trials. Most of the problems with AFO treatments include muscle atrophy, increased exertion, difficulty adapting during ambulation, difficulty standing from a seated position, aesthetic concerns, and comfort (9). It is important to investigate these factors to determine if a patient is a proper candidate for AFO therapy.

**FES**

FES neuroprosthesis use electrodes to stimulate the common peroneal nerve during the swing phase of the gait cycle. Types of FES include implantable, external, and brain controlled. The most common FES therapy in the literature is the noninvasive external device, which has been shown to have an instant effect on cadence during short and long walking performance tests. Problems associated with this prosthesis include skin irritation, pain, adaptability, and limitation to lower motor neuron pathologies. The overall goal is to allow for the patient to become independent of the prosthesis (7).

The implantable and brain computer interface devices can be used for both upper and lower motor neuron pathologies. Theoretically, electroencephalography picks up signals in the cerebral cortex of the CNS, which are then wirelessly transferred to the FES device to stimulate the motor neuron at the appropriate time of gait. The phase 1 clinical trials are showing promising results with improvements to the biomechanics and walking distances. Most complications, such as infection, pain, and prominences, arise when applying the implant during surgery. The major issues include the extensive duration of physical therapy needed to adapt to the devices (10). More research is necessary in order to implement use of this modality in clinical practice.

**WalkAide**

Many FES devices for management of drop foot are currently available in the market today. One device, the WalkAide, is revolutionary in improving the lifestyle of individuals living with drop foot. WalkAide was designed at the University by Alberta by a neuroscientist Dr. Richard Stein, who is the founder of BioMotion Ltd (a subsidiary of Hanger Orthopedic Group) (11). WalkAide was approved by the Food and Drug Administration (FDA) in 2005 for patients with drop foot caused by upper motor neuron injuries such as stroke, cerebral palsy, and multiple sclerosis (12). It can also be used for drop foot caused by peripheral or lower motor neuron damage such as spinal cord injury and leg trauma.

The FDA approved WalkAide as a class II device, meaning it is subject to special control such as mandatory performance standards. This medical device is unique as it uses a tilt sensor technology built into the stimulator. This analyzes movement by tracking the speed and angle of the leg during gait, which allows a safer and smoother gait, allowing patients to look and feel more natural during ambulation (12,13). The device also enhances blood circulation, reduces joint stiffness by maintaining ankle joint range of motion, and reduces muscle atrophy. The device is similar to a blood pressure cuff placed on the leg with electrodes attached to the skin (13) (Figures 1, 2).

The electrode will stimulate the appropriate nerve such as the peroneal nerve, which in turn will send signals to the muscle, which elicits a response. The device must be fitted by a professional. It is available through Hangers’s certified orthotist. The orthotist will fit the electrode and place the cuff properly around the leg. The WalkAide setting is programmed using the walkanalyst software. A gait examination is then performed and the settings can be reprogrammed to suit each individual patient (13). Furthermore, the device is able to analyze each step during gait and adjust the stimulation as needed. It can give our drop foot patient population an improved quality of life.
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WalkAide is contraindicated in pregnant patients, patients with a history of seizures, and patients with implantable devices. Overall, WalkAide is easy to use and can be concealed with long clothing. It is convenient, not bulky, and can easily be taken off and reapplied. It can also be used in combination with other assistive devices such as a cane, walker, and knee scooter.

When determining the proper treatment for a patient it is important to first determine which motor neuron pathology is exhibited. Younger patients are generally able to adapt to the FES therapy better than the elderly patient population. It is important to evaluate each patient’s individual goals and activity level to come up with an appropriate treatment plan. Although FES therapy had a better effect on exertion and walking cadence when compared to the AFO, most of the studies reported the subjects were having trouble adapting to the FES. Some studies used a combination therapy, which showed promising effects on subjects, allowing them to walk longer distances. Overall, additional randomized controlled trials must be performed and analyzed for regular and successful use of FES therapy.

REFERENCES

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