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An Orthosis to Aid in Reduction of Lower Limb Spasticity

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INTRODUCTION

Since February, 1979 at the West Texas Rehabilitation Center Orthotics Department, lower limb spasticity has been treated with an orthosis that uses the same principles as some of the tone-inhibiting cast techniques[3,4] (Figures 1 and 2). Twenty-one patients, all of whom were ambulatory with a plantar flexed gait, either bilaterally or unilaterally, have been treated with 37 orthoses. The goals of treatment were to improve ambulation and correct any present deformity. While the majority of the 21 patients were diagnosed as having cerebral palsy, three suffered from head injuries, three were idiopathic toe walkers, one had a psychomotor retardation, and one had a developmental delay following premature birth. Two, the cerebral palsy and psychomotor retardation patients, did not meet the goals of treatment.

CASE STUDIES

The first patient with an orthosis was a white male, age two years and five months, with cerebral palsy. When first seen, the patient had plantar flexion contractures and moderately pronated forefeet. The prescription called for bilateral conventional AFOs with 90° plantar flexion stops and metal sole plates. Prior to this, he had been wearing Rood shoes.†

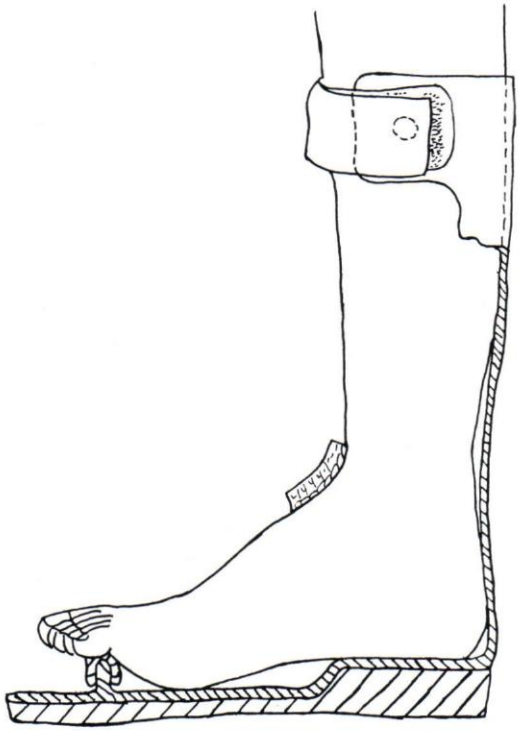

Figure 1. A line drawing of the tone inhibiting orthosis.

† Rood, M.S. Neurophysiological mechanisms utilized in the treatment of neuromuscular dysfunction. *American Journal of Occupational Therapy*, 1956, Vol. 19, pp. 220–224.

After three months, the metal sole plates had to be removed due to wear on the soles of the shoes and toe area. The patient was still walking on his toes and was having cramps in the calfs of his legs, accompanied by pes planus. The physician then recommended arch pads and medial heel wedges. Four months later, without previous orthotic success, plastic AFOs with spastic inhibitor bars were discussed with the physician and the parents.

Upon the initial fitting and wearing schedule of 24 hours per day for the first two weeks and then to a normal wearing schedule of eight hours per day, the patient began walking with a heel-toe gait. In three months, the range of motion at the ankle had greatly improved—he had from two to three hours of carry over and was able to walk heel-toe without the orthoses. Many modifications and orthoses were tried on this patient to find the most effective combination. One year after fitting, he could actively dorsiflex to 10° and only wore the orthoses one-half of each day. After two years and one month, he can actively dorsiflex to 20°, and walk 24 feet on his heels in full dorsiflexion, and he has developed a normal arch. We are still following this patient. Presently, he is wearing foot orthoses to reinforce normal arches and weight-bearing surfaces on the foot.

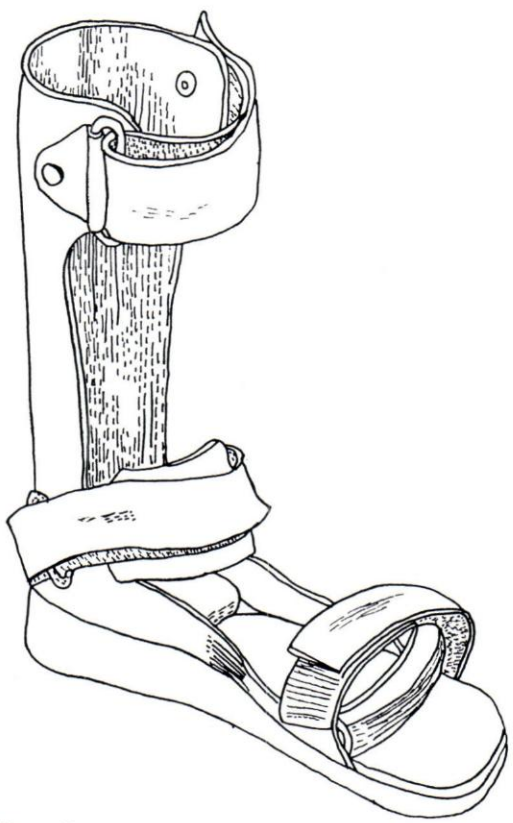

Figure 2.

Another patient, a 20 year old male automobile accident victim with a closed head injury, also improved successfully. This patient had been comatose for two and one-half months, with resulting bilateral plantar flexion contractures. After extensive physical therapy, he was referred to the orthotics department. He was ambulatory with two physical therapists assisting him, and his range of motion at the ankle was limited in dorsiflexion to neutral, with moderate extensor spasticity. He was fit with bilateral AFOs with spastic inhibitor bars and was instructed to wear them 24 hours per day initially and to ambulate in therapy only. When checked ten days later, he could be passively stretched to 5° of dorsiflexion, and stand and ambulate with minimal assistance from one person. The full day treatment was continued for two more weeks. Four weeks after fitting, he could be dorsiflexed 10° to 15° with minimal force and nighttime wear of the orthoses was discontinued.

The spastic inhibition bars and internal heels were removed two months after the initial fitting. On his next visit one month later, he exhibited a full range of motion at the ankle with active dorsiflexion to neutral. He was then instructed to wear the orthosis four hours per day. Three months and three weeks post-fitting, he could actively dorsiflex the right ankle 5° and the left ankle 15°. He was using a walker unassisted to ambulate without the AFOs in the evenings. After four months and three weeks of orthotic treatment, this patient is now wearing tennis shoes and using a quad cane.

FABRICATION OF THE ORTHOSIS

An impression is taken for an AFO without the heel board and at neutral if possible. Modification of the impression is done by wedging if the neutral position is not attained. The impression is extended anteriorly from the toes, maintaining the width of the metatarsal heads (Figure 3). This extension is approximately 2" and is important for the placement of the spastic inhibition bar and to increase leverage. The build-ups are similar to that of a normal AFO. Be sure the sole is flat. Fill in any present arch and do not lose the metatarsal head reference marks. The malleoli are extended proximally 6mm (¼") (Figure 4). Three-sixteenths inch polypropylene is used to vacuum form the AFO. The trimlines are like any AFO (i.e. proximally 20mm distal to the neck of the fibula; distally it is trimmed proximal to the metatarsal heads with the sole extended past the toes). There are three straps on the orthosis: a toe strap across the metatarsal head apices, a sable strap with padding, and the proximal calf strap (Figure 2). The spastic inhibition bar may be fabricated out of many types of materials. In the original design, a plastic welding rod was used and padded. More recently, neoprene crepe or plantation crepe has been hot glued in place.

The height of the bar varies according to the size of the patient. The important factor is to dorsiflex the toes, but not hyperextend them. A ⅝" crepe sole or plantation crepe sole is then hot glued to the sole of the AFO to add rigidity and serve as a wearing surface, since no shoe is worn over the orthosis. In some cases the orthosis functions as described; however, the majority of the patients seen require a ¼" crepe heel placed inside the orthosis. This height may vary with size and age of the patient (Figure 5). In the original design, this relief was done on the cast, making cast modifications more difficult and time consuming.

The placement of the spastic inhibition bar has proven to be critical. It must dorsiflex the toes, but not press on the metatarsals. This is best achieved in the following way. Have the patient don the orthosis before the spastic inhibition bar is in place and mark the sole for placement with the patient standing.

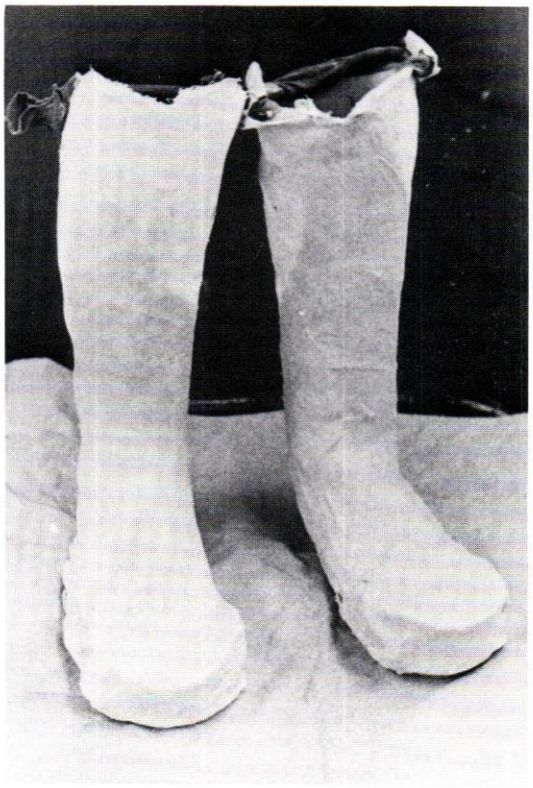

Figure 3. Impression extended anteriorly from the toes, maintaining width of metatarsal heads.

THEORY

Since there have been no kinematic studies explaining the mechanisms of the orthosis, we can only theorize at this point as to why the orthosis works. To do this, one must understand the basic reflexes affected in the foot. The majority of information is found in the cerebral palsy literature. Of key interest is the toe-grasping reflex of the foot and the associated pattern of extension. The ultimate adequate stimulus for these reflexes is not understood. In the newborn, the lightest touch

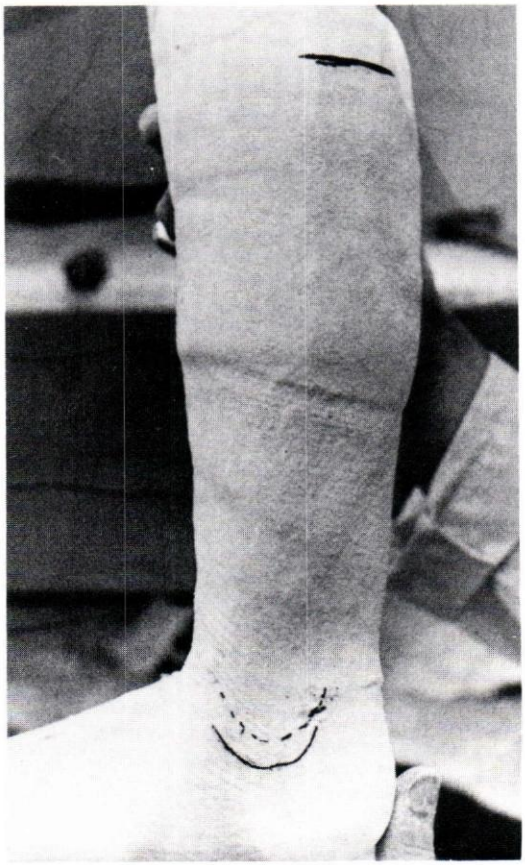

Figure 4. Malleoli extended proximally 6mm (¼").

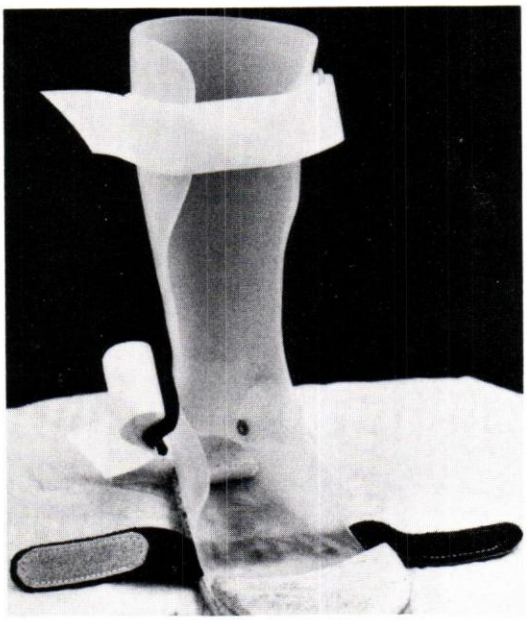

Figure 5. The ¼" heel is placed inside the orthosis.

on the ball of the foot is sufficient to produce the toe-grasping reflex. As the child matures, this sensitivity gradually diminishes as does the reflex.

If a reflex remains active for several years, which frequently occurs in children with cerebral palsy, there is often an unexplained spread of the reflexogenous area, with the result that spasticity occasionally includes the entire foot (Figure 6).

Tests were done to determine whether the four reflexes illustrated in Figure 6 were mediated through skin receptors, deep proprioceptive receptors, or both. Electromyographic results seem conclusive that these reflexes are mediated through skin receptors. All four reflexes were intensified by the placing of the subject in the upright position, by the startle reaction, and by the waves of increased ten-

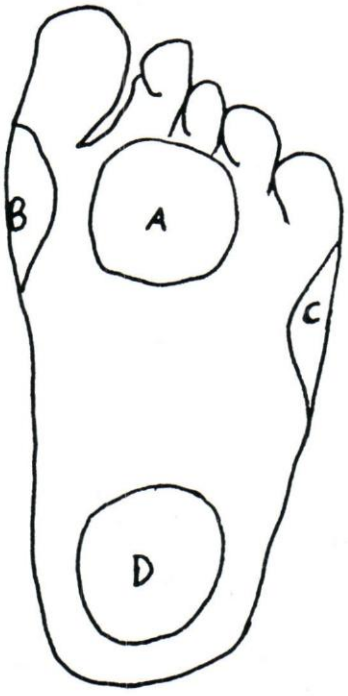

Figure 6. The typical reflexogenous areas (a) toe-grasping reflex, (b) inversion reflex, (c) eversion reflex, and (d) dorsiflexion reflex.

sion in the muscles of patients with athetosis. The reflexes were unobtainable during sleep and second-stage anesthesia. Because the toe grasp was the last to disappear, we concluded it was the strongest, most persistent reflex action pattern.[1]

In the reflex-induced deformities, dominant reflexes are pathologically hyperactive. This situation is usually encountered in cerebral palsy. One or more of the reflexes may persist throughout childhood and instead of gradually disappearing, become more active with time.

The primitive reflex of the extensor thrust, of which plantar flexion and inversion are strong components, is mediated by the lower third of the pons. This reflex is quite often found in the normal infant up to two months of age. The Positive Supporting Reaction, of which plantar flexion and inversion are also strong components, is mediated from the brain stem level and is found in normal children up to six months old.[2]

J.D. French, in a recent article in *Scientific American* (196:30), called "The Reticular Formation," sums up the brain stem functions this way, "It awakens the brain to consciousness and keeps it alert; it directs the traffic of messages in the nervous system; it monitors the myriads of stimuli that beat upon our senses, accepting what we need to perceive and rejecting what is irrelevant; it tempers and refines our muscular activity and bodily movements."

Since the lower third of the pons and the brain stem control the aforementioned primitive reflexes, it can be theorized that the closed head injury patients treated with this orthosis had sustained damage to the lower pons, or to structures which modulate the functioning of the lower pons.

The spastic inhibition bar on the AFO is theorized to reduce the stimulus to the reflexogenous area of the toe-grasp reflex. The heel placed inside the AFO is to increase the ground reaction forces in the reflexogenous area of the dorsiflexion reflex. The anterior extension, in conjunction with the AFO, increases the leverage, causing a downward moment at the calcaneous. The combination of the increased dorsiflexion and the inhibited toe-grasp reflex has proven useful itself in the treatment of plantar flexion spasticity of the lower limb.

A major consideration in application of the AFO's with spastic inhibition bars is the cooperation of the physician, the parents, and the therapists. The physician is instrumental in evaluating the radiologic structure of the feet, in determining whether the child might benefit from the AFOs, and determining whether additional support for the long term maintenance is indicated. The caretakers have the responsibility of placing the AFOs on the patient's feet in the morning and keeping them on until bedtime. The AFOs are not attractive and do cause comments and questions when the patient is in public. Many caretakers find this extremely difficult. It is also the caretakers' responsibility to bring the patient to therapy. Therefore, without full support, the effectiveness of the AFOs is compromised.

Several parents have reported that their child would ask for his AFOs if the parent forgot to put them on. This was regarded as an indirect indication that the child felt more secure in the AFOs and was thought to be a positive outcome by the therapist and the parents.

SUMMARY

This article presents two case studies involving the use of tone reducing orthoses. The orthoses utilize spastic inhibition bars to dorsiflex the toes which help break the spasticity patterns. There is also a discussion of the origins of spasticity and theory behind the effectiveness of the orthoses.

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