CHAPTER 5

Pediatric Spastic Equinus Deformity

Stephen J. Miller, D.P.M.

CEREBRAL PALSY

The term "cerebral palsy," coined by William Osler in 1889 and also known as "static encephalopathy," refers to the symptom complex produced by a non-progressive central nervous system (CNS) lesion occurring during gestation or at birth, and resulting in some motor deficit. Cerebral palsy affects approximately 0.1-0.2% of all children, however, there is no familial or genetic inheritance pattern. There are approximately 2,500 new cerebral palsy patients born each year, with an approximate total population of 1 million patients in the United States at any one time.

The degree of functional impairment depends on the part of the brain which is damaged. There may be pyramidal (associated with the motor cortex) or extrapyramidal involvement. Spasticity occurs when the pre-motor cortex is damaged, whereas flaccidity or atonia is present when the motor cortex is damaged. Extrapyramidal basal ganglia damage results in athetosis, while damage to the cerebellum results in ataxia. When there is diffuse brain damage, it is manifested by various amounts of rigidity.

This paper will focus on patients diagnosed with spastic cerebral palsy. Spasticity refers to a pattern of muscle hyperreflexia, hypertonicity, and clonus. The cause is brain damage which results in a reduction of inhibitory control from the CNS. This causes dysfunction of the gamma motor system with a subsequent increase in lower motor neuron impulse. Hence, spastic cerebral palsy is characterized by an increased stretch reflex mediated through the gamma motor neuron system. As a result, attempted movement of a muscle results in instant and various resistance.

Since we are dealing with ambulatory spastic cerebral palsy, it is important to understand the anatomic classification of this disorder, which depends on the limbs involved. Spastic monoplegia, a rare presentation, indicates one limb involvement only. Spastic paraplegia refers to involvement of the lower limbs only, whereas spastic diplegia indicates the lower limbs are involved to a greater degree than the upper extremities. Spastic quadriplegia means that all four limbs are involved, whereas spastic hemiplegia involves the limbs on one side of the body only.

SPASTIC EQUINUS

The most common spastic deformity in cerebral palsy is equinus, which involves contracture of the gastrocnemius or the gastrocnemius-soleus muscle tendon complex (triceps surae). It may be combined with an equinovalgus deformity of the foot when there is associated pronation due to spastic peroneal muscles, or equinovarus deformity when associated with over-activity or spasm of the tibialis posterior muscle.

There are two types of equinus deformity. "Dynamic contracture" is due to marked hypertonicity of the triceps surae mechanism. Attempted passive stretch results in immediate resistance due to increased muscle tone. The muscle will ultimately yield to a sustained stretching force. "Myostatic contracture" occurs when the muscle becomes intrinsically, irreversibly shortened. No amount of stretching will produce any additional length without tissue damage.

CLINICAL ASSESSMENT OF CEREBRAL PALSY

The initial assessment begins with an observation of the patient's daily activities. This will give a general idea of the intelligence level and physical capabilities of the patient. Pathologic reflexes seen in cerbral palsy include: Babinski's reflex, hyperactive tendon reflexes, and the neck-righting reflex. If these persist for greater than 20 weeks of age, the

prognosis for unsupportive walking is poor.

Examination of seven postural reflexes, as outlined by Bleck, can help determine a patient's potential for unassisted gait. These include:

1. Asymmetric tonic neck reflexes (ASTNR)
2. Neck-righting reflex
3. Moro reflex
4. Symmetric tonic neck reflex (TNR)
5. Parachute reaction
6. Foot placement reaction
7. Extensor thrust

If any one of these is still present at twelve months or older, then the ambulatory prognosis is guarded. If two abnormal reflexes are present, the prognosis is poor. It is also poor if there is a positive parachute reaction (absence of protective response).

DIAGNOSIS OF SPASTIC EQUINUS

Equinus is present when dorsiflexion of the foot at the ankle is equal to or less than neutral (90 degrees), when the subtalar joint is held in a neutral position.

Initial evaluation of the equinus deformity should determine whether it is "primary" due to muscle imbalance (usually spasticity of the triceps surae or its components), or "secondary" to another deformity such as hip or knee flexion, or a result of "overflow." Overflow refers to the involuntary activity of one muscle group caused by the voluntary activity of another muscle group.

Determining which components of the triceps surae are responsible for the equinus is best determined by the Silverskiold test, where ankle dorsiflexion range of motion is measured with the knee extended as compared with when the knee is flexed (Fig. 1). Even though this may determine when the gastrocnemius muscle is the primary contracture, this has been challenged by Perry, who felt that the soleus can cause recurrence of the equinus after selective lengthening of the gastrocnemius muscle in the spastic patient. Nevertheless, each of the component muscles of the triceps surae should be analyzed for strength, clonus, rigidity, resistance to rapid passive stretch, and resistance to slow steady pressure with the knee both extended and flexed. In-depth muscle testing includes evaluating the amount of voluntary control, strength,

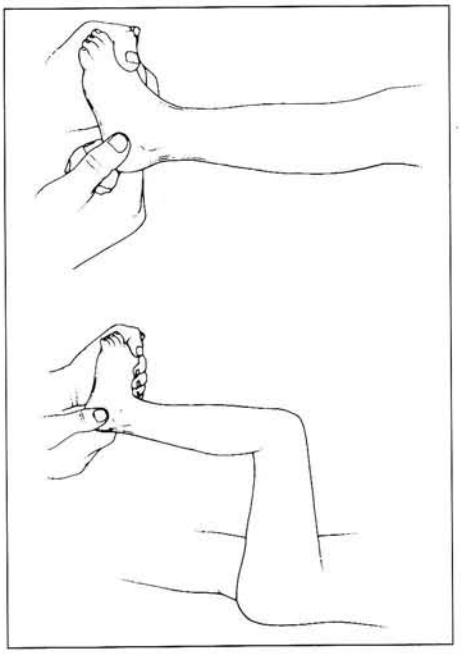

Figure 1. The Silverskiold test to differentiate between gastrocnemius equinus and gastrocnemius-soleus equinus.

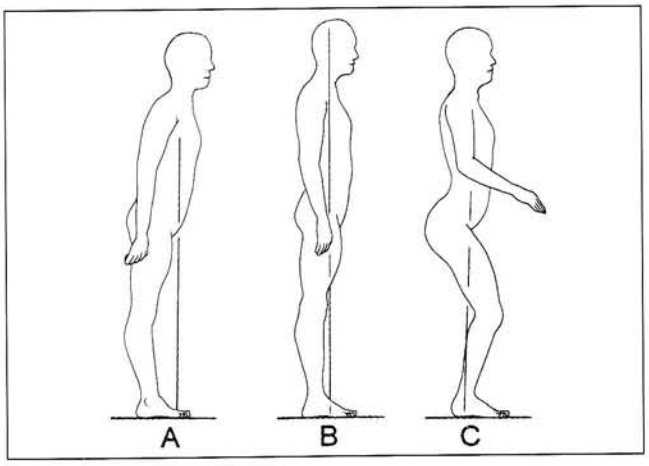

Figure 2. Compensation for equinus in the spastic cerebral palsy patient. A. Fixed ankle equinus compensation. B. Mild equinus compensation. C. Decompensated crouch with lordosis for severe equinus.

spasticity or sensitivity to stretch (including clonus), contracture excursion, synergy, and strength of the antagonist (dorsiflexors).

Observing the spastic cerebral palsy patient in static stance will demonstrate compensation for equinus (Fig. 2). Fixed ankle equinus is compensated for by knee hyperextension and hip flexion. Mild equinus may be compensated for by controlled genu recurvatum. A decompensated crouch posture that makes standing and walking

virtually impossible without assisted devices is the most severe form of compensation, especially when other contractures are present.

Gait analysis begins with an evaluation of shoe wear. Indeed, this will quite often show excessive wear on the toe area of the sole of the foot. In stance phase, the patient should demonstrate a toe-heel pattern due to the equinus deformity (Fig. 3). During swing phase, excessive

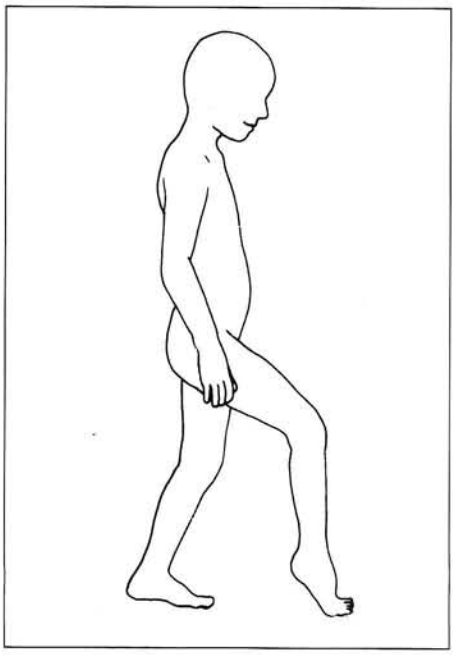

Figure 3. Steppage toe-heel gait in equinus.

knee and hip flexion combines with the equinus to equal an inefficient gait with energy loss due to non-forward motions. The patient will virtually vault over the equinus foot. Often lordosis of the spine is exaggerated during gait.

CONSERVATIVE TREATMENT

Treatment of the spastic patient requires a team approach. Even for spastic equinus, the team should involve the pediatrician, podiatrist, therapist, neurologist, social worker, orthopedist, and the parents.

Exercises are an important part of therapy during the first year, as well as for the next few years. This include passive stretching exercises (Fig. 4), active exercises of the antagonists of spastic muscles, strength enhancement, and the teaching of motor skills. Bracing for equinus includes splinting, the use of a long-leg orthosis at night, a short-leg orthosis for weight bearing, and when indicated, dynamic bracing for ambulation.

For the spastic patient, particularly in dealing with lower extremity deformities, it has been noted that a surgical procedure may be a more conservative approach before exhausting a patient with a series of ineffective non-operative therapies.

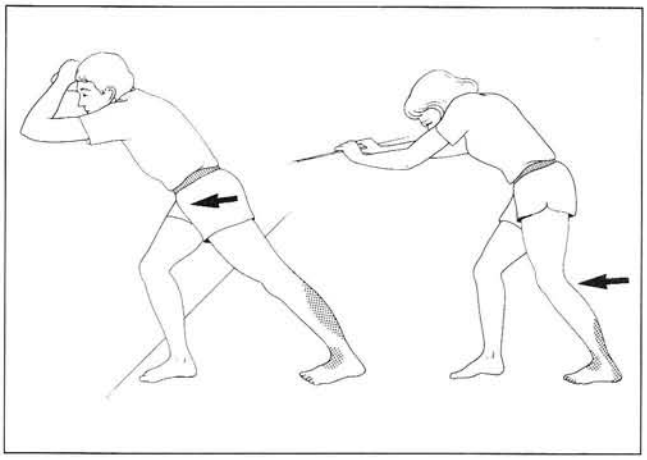

Figure 4. Passive stretching triceps surae components.

Compliance with a non-operative program by the patient and family will give the surgeon some idea as to whether cooperation can be expected for proper postoperative care.

SURGICAL TREATMENT OF SPASTIC EQUINUS

Patient selection and timing are critical to the success of surgery in correcting spastic equinus. With this discussion, we are concentrating on the spastic cerebral palsy patient with a relatively stable deformity. Surgery on an athetoid patient, for example, is highly unpredictable. The spasticity should not be excessive, and the patient's intelligence should be sufficient to allow for proper postoperative management.

Although splinting and stretching are appropriate for the first year of treatment, tendon lengthenings are recommended beyond this age. However, studies have indicated that the later the heel cord is lengthened, the less chance for recurrence.

After four years of age, a combination of surgery plus bracing is indicated for the spastic patient. Ambulatory ability is optimum from eight

to twelve years, so this is the window for optimum surgical intervention of the contracted triceps surae. Such procedures may be augmented by other tendon lengthening and/or tendon transfers.

For bony procedures on patients under ten to twelve years, a calcaneal osteotomy or a subtalar joint arthrodesis is recommended. For patients over ten to twelve years, a triple arthrodesis is more appropriate when indicated. Surgery on teenagers is difficult due to emotional, developmental, and social considerations.

There is an interesting history of surgical procedures developed to correct the various components of spastic equinus. The procedures favored for correction of spastic equinus are similar to those for non-spastic equinus. However, their timing, selection, and combination are very important to insure success and prevent recurrence. The general goal of surgical intervention for spastic equinus is to stabilize the lower extremity, and efficiently use available strength to allow the patient to walk as independently as possible. The idea is to facilitate function, rather than to "stamp out deformity." The specific goal is to allow the foot to be flat on the floor without evoking a stretch reflex.

Attention must be paid to associated deformities, such as hip and knee contractures, which need to be corrected prior to or simultaneously with equinus repair especially to prevent over stretching of a lengthened gastrocnemius in gait postoperatively. Hip contracture must be released prior to equinus correction, and knee contracture can be treated at the same time or later. If the patient has a short limb, it is important not to fully correct the equinus so as to allow compensatory weight bearing.

Surgical correction is primarily directed to either lengthening of the Achilles tendon or the gastrocnemius aponeurosis. Tendoachillis lengthening (TAL) can be accomplished via a Z-slide of the tendon in the sagittal plane, cutting three fourths of the tendon on opposite sides at different levels. If a strict Z-slide is to be done based on the orientation of the Achilles fibers, then attention must be paid to the torsion in the tendon itself.

White observed the torsion of the Achilles tendon. Looking posteriorly, the posterior fibers rotate towards the lateral malleolus. Thus, the left achilles tendon rotates clockwise and the right achilles tendon rotates counter-clockwise. Cummins et al. recommended specific cuts by refining White's technique. They suggested cutting the posterior two thirds of the Achilles tendon proximally, and the medial two thirds distally, to effect a Z-slide. With this orientation in mind, the cuts can be made either percutaneously, or through an open incision for the more severe deformity. A frontal plane Z-lengthening is also an excellent approach to severe deformities, by allowing controlled reapproximation of the tendon with the foot in its corrected position.

In spastic equinus surgery, one of the difficulties is in determining the amount of lengthening necessary for adequate correction. Over-lengthening can result in a functionally disastrous calcaneus deformity which is virtually impossible to correct. Garbarino and Clancy devised a "geometric method" of calculating tendoachillis lengthening (Figs. 5A, B). They determined that the increased length of the achilles tendon necessary to bring the foot into its corrected position is approximately one-half of the distance that the forefoot can travel. This can be calculated by measuring the distance between the heel pad and the ball of the foot in its equinus position, and dividing it in half. This will be the approximate measurement for lengthening the tendon. By this method, they experienced 0% recurrence in 100 patients.

Gastrocnemius recession is a commonly used method to lengthen the isolated gastrocnemius muscle when it is the primary source of the myostatic or spastic contracture. However, a low recession can involve the aponeuroses to include both the soleus and the gastrocnemius muscles. In this case, healing and rehabilitation is much faster than for a tendoachillis lengthening. The reverse Baker technique, described by McGlamry and Fulp, for tongue-in-groove recession is the recommended procedure.

Craig and VanVuren were so concerned about recurrence that they recommended a combination tendoachillis lengthening and gastrocnemius recession. This was treated postoperatively in a full-length plaster cast, with the knee fully extended and the foot in about 40 degrees of dorsiflexion, for three weeks. The patient was then switched into a weight-bearing cast for an additional three weeks, followed by a retention brace until active dorsiflexion was of adequate strength. Of 276 cases, they recorded an overall recurrence rate of 9%.

Neurectomies of the gastrocnemius muscle

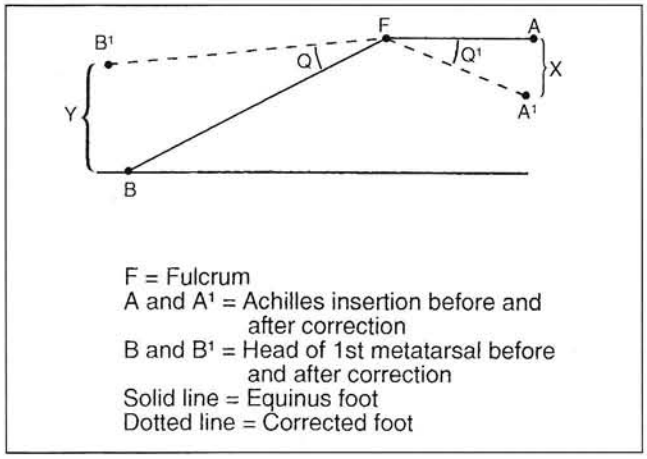

Figure 5A. Garbarino and Clancy's "Geometric method" of calculating the correct amount of tendoachillis lengthening.

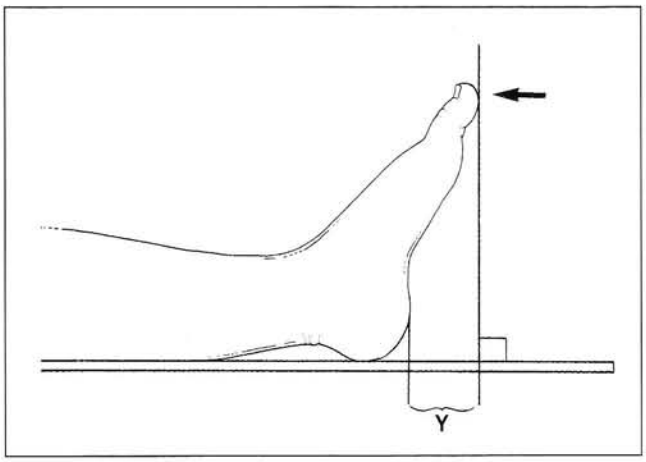

Figure 5B. Garbarino and Clancy's "Geometric method" of calculating the correct amount of tendoachillis lengthening. 1/2 "Y" is the appropriate amount of lengthening required.

heads should be avoided to prevent the development of myofibrosis. Attention should be paid to the plantaris tendon, which must be released in all cases of equinus correction. The posterior ankle and subtalar joint capsules rarely have to be released. Younger patients spontaneously rupture during forced dorsiflexion at the time of the lengthening procedures. Other posterior group flexors must be evaluated and lengthened as necessary.

Postoperative care is essential to obtaining a good result. For the spastic patient, a straight leg, knee extended cast, is recommended for the first two to four weeks, followed by a below-knee cast, as long as the quadriceps are not in severe spasm. The position of the foot should be at least ten degrees dorsiflexed on the leg. For a tendo achillis lengthening, total cast time is six weeks non-weight bearing. For gastrocnemius recession, four weeks of non-weight bearing is followed by two weeks in a weight-bearing cast.

Once the cast is removed, gait training exercises are instituted with crutch assistance. Strengthening and stretching exercises are taught to the patient, including active dorsiflexion and plantarflexion exercises, especially to augment the dorsiflexion antagonist muscles. A night splint is essential throughout the growth period, or recurrence can be expected. Bracing is usually not necessary.

The success of the surgery is measured by the rate of recurrence. The rate of recurrences for tendoachillis lengthening has ranged up to 27% (Table 1). It seems that the younger the patient at the time of the lengthening, the greater the chance of recurrence. For gastrocnemius recession, the rate of recurrence was generally slightly less, but in one study was as high as 29% (Table 2). Even when the gastrocnemius recession was combined with a TAL, the recurrence rate was 9%.

Table 1

RECURRENCE - TAL

YEAR	AUTHOR(S)	NUMBER	RECURRENCE RATE
1958	Banks & Green	164	5.5%
1959	Silver & Simon	?	4.5%
1959	Graham & Fixsen	76	17%
1969	Conrad & Frost	98	23%
1969	Lemperg	48	12.5%
1971	Frost	210	27%
1972	Sherrard & Bernstein	77	27%
1975*	Sgarlato	158	4%
1976#	Craig & vanVuren	100	9%
1977	Banks	200	7%
1980	Lee & Bleck	71	9%
1985	Garbarino & Clancy	26	0%

* Nonspastic equinus
# Combined TAL and gastroc recession

Table 2

RECURRENCE - GASTROCNEMIUS RECESSION

YEAR	AUTHOR(S)	NUMBER	RECURRENCE RATE
1959	Calandriello	?	8%
1966	Bassett and Baker	447	4%
1970	Baker, Basset & Dyas	768	5.6%
1972	Sharrard & Bernstein	15	15%
1976*	Craig and vanVuren	100	9%
1980	Lee and Bleck	51	29%
1989#	Downey and Banks	36	0%

* Nonspastic equinus
\# Combined TAL and gastroc recession

SUMMARY

Although the technical procedures for lengthening of the achilles tendon in spastic equinus are well known and similar to those for non-spastic equinus, the importance of timing, patient selection, and postoperative management cannot be overstated. This is a very special patient who requires a team approach for correction of spastic equinus, which is the most common deformity seen in the cerebral palsy patient.

REFERENCES

Baker LD: Triceps surae syndrome in cerebral palsy.*Med* 36:88, 1955.

Banks H, Green W: The correction of equinus deformity in cerebral palsy. *J Bone Joint Surg*, 40A: 1359-1379, 1958.

Banks HH, Panagakos P: Orthopaedic Evaluation of the Lower Extremity in Cerebral Palsy. *Clin Orthop Rel Res* 47:117, 1966.

Bassett FH, Baker LD: Equinus Deformity in Cerebral Palsy. In Adams JP (ed). *Current Practice in Orthopaedic Surgery* Vol 3. St. Louis, CV Mosby, 1966, p. 59.

Conrad JA, Frost HM: Evaluation of subcutaneous heel-cord lengthening. *Clin Orthop Rel Res* 64:121-127, 1967.

Craig JJ, VanVuren J: The importance of gastrocnemius recession in the correction of equinus deformity in cerebral palsy. *J Bone Joint Surg* 58B:84, 1976.

Cummins EJ, Ansun BJ, Carr BW: The structure of the calcaneal tendon in relation to orthopedic surgery with additional observation on the plantaris muscle. *Surg Gynecol Obstetr* 83:107, 1946.

Downey MS, Banks AS: Gastrocnemius recession in the treatment of non-spastic ankle equinus: A retrospective study. *J Am Podiatr Med Assoc* 79:159-174, 1989.

Frost HM: Subcutaneous tendo Achilles lengthening. *Am J Orthop* 5:256-257, 1963.

Fulp MJ, McGlamry ED: Gastrocnemius tendon recession: tongue in groove procedure to lengthen gastrocnemius tendon. *J Am Podiatry Assoc* 64:163-171, 1974.

Garbarino JL, Clancy M: A geometric method of calculating tendo achilles lengthening. *J Pediatr Orthop* 5:573-576, 1985.

Green WT, McDermott LJ: Operative Treatment of Cerebral Palsy of Spastic Type. *J Am Med Assoc* 118:434, 1942.

Lee CL, Bleck EE: Surgical correction of equinus deformity in cerebral palsy. *Der Med Child Neurol* 22:287, 1980.

Martz CD: Talipes equinus correction in cerebral palsy. *J Bone Joint Surg* 62A: 679, 1960.

Perry J, Hoffer MM, Grovan P, et al.: Gait analysis of the triceps surae in cerebral palsy: A preoperative and postoperative clinical and electromyographic study. *J Bone Joint Surg* 56A:511-520. 1974.

Phelps WM: Long-term Results of Orthopaedic Surgery in Cerebral Palsy. *J Bone Joint Surg* 39A:53, 1957.

Pollock GA: Lengthening of the Gastrocnemius Tendon in Cases of Spastic Equinus Deformity. *J Bone Joint Surg* 35B:148, 1953.

Pollock GA: Surgical Treatment of Cerebral Palsy. *J Bone Joint Surg* 44B, 68, 1962.

Schwartz JR, Cau W, Basset FH, Coonrad RW: Lessons learned in the treatment of equinus deformity in ambulatory spastic cerebral palsy. *Orthop Trans* 1:84, 1977.

Sharrard WJ, Bernstein S: Equinus deformity in cerebral palsy. *J Bone Joint Surg* [Br], 54B:272-276, 1972.

Silver CM, Simon DD: Gastrocnemius muscle recession (Silverskiold operation) for spastic equinus deformity in cerebral palsy. *J Bone Joint Surg* 41A: 1021, 1959.

Silverskiold N: Reduction of the uncrossed two-joint muscles of the leg to one-joint muscles in spastic conditions. *Acta Chir Scandivav* 56:315, 1923-1924.

Stoffel A: The Treatment of Spastic Contractures. *Am J Orthop Surg* 10, 611, 1913.

Strayer LM Jr: Recession of the gastrocnemius and operation to relieve spastic contracture of the calf muscles. *J Bone Joint Surg* 32A:671-676, 1950.

Strayer LM Jr: Gastrocnemius recession. Five year report of cases. *J Bone Joint Surg* 40A: 1019, 1958.

White JW: Torsion of the Achilles tendon: Its surgical significance. *Arch Surg* 46:784, 1943.

Tardieu C, Lespargot A, Tabary C, et al: Toe walking in children with cerebral palsy: contributions of contracture and excessive contraction of triceps surae muscle. *Phys Ther* 69:8, 1989.

Vulpius O, Stoffel A: *Orthopadische Operationshlehre.* Second edition. Stuttgart: Ferdinand Enke