CHAPTER 26

Poliomyelitis

Editor-in-chief's note: *I began work on this chapter with an eye toward revision. I planned to discard all redundant and outdated material and add all that is new. As I read the work, I forgot my mission and reveled in the excellence and precision of the chapter as it stands. Thus, I have chosen to preserve this chapter relatively unaltered, except for occasional additions of newer material. To treat the residua of poliomyelitis, which is still necessary in this world more often than we would like, one must know the fundamentals. Nowhere have I seen a better, more concise summary of those principles than in this chapter. In fact, the sections on tendon transfer, fascia lata contracture, and others are so relevant to the management of other diseases that I would urge the reader to study them regardless of his or her likelihood of encountering poliomyelitis. Thus, this chapter remains as a monument to the genius of* Myke Tachdjian. *My additions to this chapter appear in italics, and the references I have added are compiled in a separate references list.*

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Poliomyelitis is an acute infectious disease caused by a group of neurotrophic viruses that initially invade the gastrointestinal and respiratory tracts and subsequently spread to the central nervous system (CNS) through the hematogenous route. The poliomyelitis virus has a special affinity for the anterior horn cells of the spinal cord and for certain motor nuclei of the brain stem. These cells undergo necrosis, resulting in loss of innervation of the motor units that they supply.

The first description of paralytic poliomyelitis was given by Underwood in 1789.[322]

Infection may be caused by type I, II, or III poliomyelitis virus. There is no cross-immunity between the various types of poliovirus; thus, infection may recur in the same individual.[209,271] Poliovirus is a member of the enterovirus group, which includes coxsackievirus and the ECHO viruses. Paralytic disease that is clinically and pathologically indistinguishable from poliomyelitis can be produced by various other enteroviruses. These viruses may be isolated on tissue culture.

In the past, poliomyelitis was an epidemic disease in the summer and fall months, with sporadic cases occurring throughout winter and spring. The development and widespread use of a prophylactic vaccine greatly reduced the incidence of poliomyelitis; however, sporadic cases still do occur, and the continued rehabilitation of patients who have had the disease is still a concern of the orthopaedic surgeon.[209,269,273,277,302]

In recent years, attention has focused on cases of poliomyelitis caused by oral vaccine, so-called vaccine-associated paralytic poliomyelitis (VAPP). The risk of contracting poliomyelitis from the vaccine remains extremely low, at a rate of one case per 2.5 million doses. Between 1980 and 1989, 80 such cases were reported in the United States. During the same period, there were no cases due to wild virus, and five cases of imported disease were reported. People at risk for contracting vaccine-associated disease were infants receiving their first dose, persons in contact with vaccine recipients who were not vaccinated, and immunologically compromised people. Because of this rare but devastating occurrence, the Centers for Disease Control (CDC) have recommended that children be inoculated first with inactivated vaccine, followed by oral attenuated vaccine administration.[14a] *A laudable effort is being made by Rotary International and the World Health Organization (WHO) to achieve the global eradication of poliomyelitis.*[3a]

Another manifestation of poliomyelitis that has received considerable attention recently is the occurrence of postpolio syndrome. This syndrome is characterized by increasing muscle weakness, fatigue, pain, and loss of function among individuals who were afflicted with poliomyelitis 20 or more years earlier. Perry and associates[13a] *have shown that this occurrence represents a chronic overuse syndrome, with overstressed muscles "wearing out." Another theory of the etiology of this phenomenon is the failure of axonal sprouts that formed during the healing process after poliomyelitis.*[4a,6a] *A discussion of the management of this problem, however, is beyond the scope of a text on pediatric orthopaedics.*

This chapter deals with general principles of management of paralytic deformities of the musculoskeletal system resulting from poliomyelitis. These principles are applicable not only to the treatment of poliomyelitis but also to the

management of similar problems of flaccid paralysis due to other causes. For a detailed account of the disease and its medical aspects of management, the reader is referred to the voluminous literature on the subject.

Pathology

The poliovirus has a definite predilection for the anterior horn cells of the spinal cord and for certain motor nuclei in the brain stem. The lumbar and cervical enlargements of the cord are the most commonly affected. The damaging action on the motor neurons may be *direct,* occurring through the toxic effects of the virus, or *indirect,* occurring through ischemia, edema, and hemorrhage in the neurons' supportive glial tissue.[29]

The motor neurons swell and the Nissl substance in their cytoplasm undergoes chromatolysis. An inflammatory reaction ensues, with infiltration of polymorphonuclear and mononuclear cells into the gray matter, particularly in the perivascular areas. The necrotic bodies are subsequently replaced by scar tissue.

Involvement of the anterior horn cells varies from minimal injury, with temporary inhibition of metabolic activity and rapid recovery, to complete and irrevocable destruction.

Paralysis is of the flaccid type, with the individual motor units following the "all-or-none" law, because the virus affects the anterior horn cells rather than the muscle. The percentage of motor units destroyed varies, and the resultant muscle weakness is proportionate to the number of motor units that are lost. For example, a muscle with "poor" muscle strength will have 20 percent of its motor units functioning, whereas a muscle with "good" motor strength will have 80 percent of its motor units functioning. These remaining functional motor units are called *guiding neuromuscular units* and are of particular importance in retaining the patterns of motion of the individual muscles or muscle groups during the recovery stage. The recovery of muscle power primarily depends on restitution of the anterior horn cells of the spinal cord that have been damaged but not destroyed.

Immediately following onset, it is difficult to make an accurate prognosis as to the rate and extent of spontaneous recovery. It is best to assume that the involved muscles will recover until the subsequent course of the disease demonstrates otherwise. Muscle recovery is most marked in the first 3 to 6 months, with the potential for recovery ceasing at approximately 16 to 18 months after onset.

The two primary factors to consider in the prognosis are the severity of initial paralysis and the diffuseness of its regional distribution. If total paralysis of a muscle persists beyond the second month, severe motor cell destruction is indicated, and the likelihood of any significant return of function is poor. If the initial paralysis is partial, the prognosis is better.

The condition of the neighboring muscles is another consideration. A weakened muscle surrounded by completely paralyzed muscles has less chance of recovery than a muscle of corresponding power that is surrounded by strong muscles. Muscle spasm, contracture of antagonist muscle groups, deformity, and inadequate early treatment are other factors that may interfere with recovery of muscle function.

The course of the disease is subdivided into acute, convalescent, and chronic phases. The *acute phase,* lasting from 5 to 10 days, is the period of acute illness when paralysis may occur. It is further subdivided into the *preparalytic phase* and the *paralytic phase.* The acute phase is ordinarily considered to terminate 48 hours following the return to normal temperature.

The *convalescent phase* encompasses the 16-month period following the acute phase. During this time a varying degree of spontaneous recovery in muscle power takes place. This phase is also further subdivided into the *sensitive phase* (lasting from 2 weeks to several months), characterized by hypersensitivity of muscles, which are tender and "in spasm," and the *insensitive phase,* in which the muscles are no longer sensitive but are still in the period of recovery.

The *chronic* or *residual phase* is the final stage of the disease after the recovery of muscle power has taken place. It encompasses the rest of the patient's life following termination of the convalescent period.[201]

Treatment

The management of poliomyelitis varies with the stage of the disease and the severity and extent of paralysis.[118,120] Treatment in the acute febrile stage is primarily the domain of the pediatrician or internist, with the patients being admitted to infectious disease hospitals or to isolation units of general hospitals. Care of the musculoskeletal system, however, is important from the first day of the disease. It is imperative that the orthopaedic surgeon be consulted to examine a suspected case before a lumbar puncture is performed. The surgeon should be responsible for all orders concerning the management of the musculoskeletal system. The pediatrician is responsible for general care of the patient, especially any problems of respiratory system and bulbar involvement, should they develop. Once the patient has been afebrile for 18 hours (i.e., after termination of the acute stage), he or she should be transferred to the service of the orthopaedic surgeon, who assumes the dominant role. Such delineation and continuity of supervision is mandatory, as it stimulates early attention to deforming tendencies and prevents their development.

ACUTE PHASE

During the initial, febrile phase of the disease, the primary concern of the orthopaedic surgeon is the comfort of the patient and preventing deformity. It is best to place the patient on complete bedrest and restrict physical activities to a minimum. The patient is irritable and apprehensive. It is important to reassure the patient and allay fears.

General medical measures consist of administration of a varied diet with a relatively high fluid intake, attention to urinary retention and bladder paralysis, preventing of constipation and fecal impaction, and providing analgesia for pain. Opiates and other medications that have a depressing action on the CNS should not be given in the presence of impending paralysis of the muscles of respiration.

A detailed determination of the severity and extent of muscle paralysis is not warranted during this febrile period. By gentle handling of the limbs and trunk, however, the

clinician can make an approximate assessment of the degree and distribution of the motor weakness without much distress to the patient. This initial muscle examination has diagnostic and therapeutic implications. It will also provide the necessary information to prevent the development of potential deformities consequent on paralysis.

Ordinarily, paralysis develops 2 or 3 days after the onset of fever and increases in severity for several days. Progressive involvement will cease only after the elevated temperature returns to normal. Characteristically, paralysis in poliomyelitis is asymmetric. In the presence of symmetric paralysis of the limbs and trunk, a paralytic disease other than poliomyelitis should be considered. In a large epidemic, the care of patients will be much simplified if those with paralysis are separated from those without paralysis.

Patients with bulbar and respiratory involvement require specialized intensive care. An early appraisal of the distribution and extent of paralysis will help detect muscle weakness in certain areas, which should alert the clinician to the possible development of such distressing complications. For example, a patient who cannot lift the head because of paralysis of the anterior neck muscles or one who has a nasal intonation to the voice, difficulty swallowing, and weakness of facial muscles should be watched carefully for bulbar involvement. Prompt diagnosis and treatment are essential to keep the patient's airways open, since the condition may be fatal. Aspiration of unswallowed secretions is a definite danger. The foot of the bed is elevated and the patient is placed in a prone or lateral position. Frequent suction or postural drainage is usually required. Occasionally tracheostomy may be necessary.

Another anatomic area that should be observed for muscle weakness is the shoulder girdle. The nerve supply to the deltoid muscle is provided by the fifth cervical root, which is adjacent to the fourth cervical root, innervating the diaphragm. Consequently, progressive paralysis of the deltoid muscle is usually followed by paralysis of the intercostal muscles and the diaphragm. Is the rate of breathing increased? Is the patient using accessory muscles of respiration? Is the patient restless, anxious, and disoriented? These are signs that should alert the physician to the possible need for a mechanical respirator. Paralysis of the diaphragm is easily detected on fluoroscopy. Abdominal muscle weakness is determined by asking the patient to lift the head and shoulder or the lower limbs. Asymmetry of power is indicated by Beevor's sign, which is a shift of the umbilicus toward the stronger muscles.

Patient positioning should provide correct anatomic alignment of the limbs and proper posture of the trunk. The aim is to prevent the development of deformities. The bed should give adequate support and should not sag. A firm foam rubber mattress is preferable. Bedboards should be placed beneath the mattress and should be hinged to permit sitting in the later convalescent period. A padded footboard is used to maintain the ankles and feet in neutral position when the patient is lying supine or prone. Pulling the end of the mattress about 10 cm away from the footboard provides an interspace in which the heels are allowed to fall. Periods in the supine position should be alternated with periods in the prone position, the latter position being important for maintenance of good muscle tone of the gluteus maximus and erector spinae muscles.

When the patient is lying on his or her back, the knees should be held in slight flexion with padded rolls under them and behind the proximal ends of the tibiae in order to prevent genu recurvatum and posterior subluxation of the tibiae. A slightly flexed position of the knees will relax the sensitive hamstrings. Excessive flexion of the knees, however, should be avoided. Sandbags or rolled pads are placed on the lateral sides of the thighs and legs to prevent external rotation deformity of the lower limbs. Intermittent use of rolls between the scapulae will prevent forward hunching of the shoulders.

The limbs should not be maintained in rigidly fixed positions. Several times a day, the joints are carried passively through their range of motion; this will help relieve muscle pain. Overstretching of the muscles, however, should be avoided. The patient should be handled as gently as possible. Passive motion of the joints of a limb is imperative to prevent stiffness and myostatic contractures. At times, when there is severe spasm of the hip flexors, hamstrings, and gastrocnemius, the sensitivity and pain of muscles will be so great that anatomic alignment cannot be assumed without excessive discomfort.

Muscle Spasm. A principal manifestation of poliomyelitis in its early stages, the so-called muscle spasm is characterized by protective contraction of the muscles to prevent a potentially painful movement. Muscle resistance to stretch is more descriptive of this reflex guarding action of the muscles, which resembles the muscle spasm associated with painful phenomena such as hamstring spasm in synovitis of the knee. True spasticity and signs of upper motor neuron involvement are absent. The exact cause of the muscle pain and sensitivity is unknown. Most probably they are due to inflammatory changes in the posterior ganglia and meninges. Other possible causes are lesions in the reticular substance and lesions of the internuncial neurons in which inhibitory fibers to the anterior horn cells are affected.

The degree of muscle pain and sensitivity varies considerably. Some muscle discomfort is usually present in the preparalytic period. Nerve traction tests, such as those of Lasègue and Kernig, increase "muscle spasm" and pain. Spontaneous severe pain is rare but occasionally seen in the adult patient. The important consideration is that the painful strong muscles tend to shorten during the sensitive phase; if they are maintained in their shortened position, myostatic contracture and fixed deformity will develop.

Moist Heat. In the acute and sensitive phase of convalescence, application of moist heat relieves the sensitivity of the muscles and alleviates discomfort. Physiologically, heat increases the local temperature and increases blood flow to the muscle. It has no specific therapeutic effect on the course of the paralysis and the actual recovery of the involved nerve cells. Heat is more beneficial if it is applied intermittently for short periods.

In the acute phase, to minimize handling of the patient, a lay-on wool pack is used. It consists of three layers, one of wool blanket material (wrung out of boiling water by passing it twice through a wringer), and one of waterproof material that, in turn, is covered by an outer layer of wool blanket. The number of these packs and the duration of their use are individualized, depending on the intensity of pain and spasm. In general, two moist heat packs are applied

during a 20-minute period. Continuous and overzealous use of heat should be avoided, as it can be tiring and harmful to the patient. Moist heat is best used prior to physiotherapeutic measures in order to assist in developing greater range of joint motion and to facilitate the performance of active exercises. Warm tub baths are substituted for the lay-on packs within a few days after the patient's temperature has returned to normal and when the patient's general condition permits. The buoyant effect of the water makes it easier for the weakened muscles to move. Active exercises in water should be closely supervised so that the patient does not substitute stronger muscles for the weaker ones. Again, the patient's comfort is the primary consideration. The temperature of the tub baths should be about 100°F, and the total period of immersion in the tub should not exceed 20 minutes. In cases of extensive paralysis, overhead cranes may be used to lower the patient directly into the tub from the stretcher.

CONVALESCENT PHASE

The objectives of treatment during the convalescent stage are (1) the attainment of maximum recovery in individual muscles, (2) the restoration and maintenance of normal range of joint motion, (3) the prevention of deformities and their correction if they occur, and (4) the achievement of as good a physiologic status of the neuromusculoskeletal system as is possible.[120]

In the early part of the convalescent stage, because muscle sensitivity and "spasm" are still present, the use of hot packs is continued for the comfort of the patient. Passive exercises are performed four to six times a day to prevent development of contractural deformity. When there is limitation of joint motion, gentle passive stretching exercises are added to the therapy program. This exercise regimen should not cause the patient discomfort; however, the threshold of pain may be very low in an apprehensive, sensitive person. A firm but sympathetic attitude by the therapist is important, and the patient should be encouraged more each time to gain a greater degree of motion. Tendencies toward deformity should be observed, such as external rotation and abduction of the hips, plantar flexion of the feet, or adduction of the shoulders. Passive stretching exercises should be directed toward preventing and correcting deformity.

Several days after the onset of the convalescent stage, a complete muscle examination should be performed. Ordinarily it is done in stages in order not to fatigue or disturb the patient. This initial motor assessment provides a basis for comparison with subsequent examinations, and it also serves as a guide to the therapy regimen that is to be instituted. The rate and extent of muscle recovery are determined by repeating these muscle tests periodically—monthly during the first 4 months, bimonthly during the following 8 months, and then quarterly during the second year of the disease. The prognostic value of the serial muscle tests is evident: When a muscle exhibits little or no improvement in power during a 3-month period, it is unlikely that it will recover or gain strength of functional significance. In such a case the patient should be fitted with appropriate orthotic support and allowed greater activity. On the other hand, a muscle that shows steady improvement has a good possibility of recovering to a functional level; hence, it is unwise to apply an above-knee orthosis on this patient's weak limb and permit him or her to walk.

In the management of the convalescent stage of poliomyelitis, the following principles of neuromuscular function must be considered.[120]

Patterns of Motor Activity. Limb motions are complex and are not the result of isolated contraction of a single muscle. The functions of many muscles are integrated and coordinated in the execution of a movement and are controlled by the automatic reflexes of the CNS. In dorsiflexion of the ankle, for example, the anterior tibial muscle, toe extensors, and the peroneus tertius are the prime movers that execute the desired movement, whereas the triceps surae and the toe flexors are the antagonist muscles that become relaxed because of the reciprocal innervation of the agonist and antagonist muscles. The synergist and fixation muscles also contract while the prime mover acts.

In the presence of muscle weakness, the tendency is to use a strong group of muscles that can perform the action more easily and readily, thus excluding the weaker muscles from the pattern of motor activity. A muscle that has been temporarily paralyzed will be left out of the pattern of motion permanently if other muscles substitute for its action during the period of its recovery. In the convalescent stage, these muscular substitutions and abnormal patterns of motor activity should be avoided.

Some neuromuscular units often remain intact in the paralyzed muscles; these act as "guiding contractile units," and in the performance of active exercises, these functioning neuromuscular units should be utilized to guide the part in execution of normal motion.

For example, in reeducation of a poor anterior tibial muscle, the ankle joint is first passively dorsiflexed through its full arc of motion, stretching any contracture of the triceps surae muscle that is present. The limb is then placed in a side-lying position to eliminate the force of gravity and the ankle joint is again passively dorsiflexed in some inversion through its full range, with the therapist assisting the patient to localize the action of the anterior tibial muscle and emphasizing that substitution by the toe extensors and peroneus tertius muscle should be avoided.

Next the patient is asked to produce an active, sustained contraction of the anterior tibial throughout its full arc of motion, first with and then without assistance. As the muscle becomes stronger, the limb is placed in supine position to make the muscle work against gravity, and gradually increasing manual resistance is applied. The active exercises are graduated on the basis of performance. Muscles that are overworked will lose strength.

In poliomyelitis, reciprocal innervation between agonist and antagonist muscles is often disturbed, with resultant loss of synergistic muscular action and the normal pattern of motor activity.

Fatigue. A paralyzed muscle is easily fatigued. This is readily shown by its rapid loss of power and its inability to function following several effective contractions. Forcing such a weak muscle beyond its point of maximal action does not increase its strength: on the contrary, it will inhibit the recovery of the paralytic muscle. It is important to observe the level of functional activity of a weak muscle so that it is not forced to exceed its capability.

Contractural Deformity and Progressive Loss of Function. Flaccid paralysis is the chief cause of functional loss. Muscular action is also inhibited by pain, sensitivity, and "spasm." When a muscle is maintained in a shortened position for a prolonged period, it will develop myostatic contracture. Muscle imbalance and increased stress due to abnormal patterns of activity are other factors producing deformity. Growth is an important consideration in the management of poliomyelitis in children. The contour of bony structures is influenced by paralysis and dynamic muscle imbalance. For example, when the triceps surae muscle is weak and the ankle dorsiflexors are of normal motor strength, progressive calcaneus deformity of the hindfoot will result. If the child is permitted to walk without support and protection, the loss of power of the triceps surae muscle will be greater, as it is working against gravity. Figure 26–1 shows the "vicious circle" of factors causing progressive loss of function in poliomyelitis.

In the *asensitive stage,* proper alignment of the limbs and full range of joint motion must be restored and maintained. Passive stretching exercises are performed vigorously. In the presence of muscle imbalance and when there is a tendency to develop contracture, bivalved casts should be used at night to maintain the part in correct position. When a deformity is fixed, wedging casts or traction may be applied.

Active exercises are performed to integrate recovering motor units into the normal pattern of motion; their primary objective is not to produce hypertrophy of muscles that are already functioning normally. Hydrotherapy and active exercises in a pool are utilized for patients with extensive paralysis. Motion of the hips, shoulders, and trunk is greatly facilitated in the pool, as the buoyant effect of the water facilitates the coordinated motion of the parts. Strict supervision by the therapist is mandatory, however, to prevent substitution of strong muscles for those that are weak. Excessive exercises and overwork should be avoided. Patients with extensive paralysis are initially instructed to ambulate in the pool; when there is adequate control of the trunk and lower limbs, this is no longer necessary. Standing balance should be developed first, followed by walking with the help of crutches. The gait pattern should be a reciprocal four-point gait, the amount of body weight borne depending on the degree of paralysis. The physical therapist assists in locomotion so that abnormal mechanisms do not develop. During the convalescent period, use of an orthosis should be kept to a minimum, as it increases the workload on the paralytic levels and tends to produce abnormal gait patterns. In severe paralysis of the lower limbs and trunk, however, locomotion may be impossible without the support of an adequate orthosis. General activities of the patient are gradually increased. During the first few minutes of locomotion the gait may be very effective, but with fatigue it may become very poor. Random, purposeless activity should be discouraged.

CHRONIC PHASE

The purposes of treatment in the residual stage are to enable the patient to attain maximal function and to obtain the greatest amount of productive activity in spite of residual weakness.[118] With continued growth and use of the limb, progressive deformities may develop that will ultimately cause loss of function. Hence, an equally important task during the chronic stage is to prevent deformities and to correct them, should they develop. The residual stage is a dynamic, not a static, period. Much can be done to improve the functional capacity of the patient. Aspects of treatment are discussed under the headings of Physical Therapy, Orthoses and Apparatus, and Surgery.

Physical Therapy. In the residual stage the physical therapy regimen is directed toward (1) preventing or correcting deformity by passive stretching exercises, (2) increasing motor strength of muscles by active or hypertrophy exercises, and (3) achieving maximum functional activity.[194]

ACTIVE HYPERTROPHY EXERCISES. There is little to be gained by exercising zero or trace muscles that remain so after 18 months, and the same is true of muscles that have a good or normal rating. Active hypertrophy exercises are performed primarily for the benefit of marginal muscles, to elevate or maintain their functional level. For example, when the anterior tibial and toe extensor muscles are fair in motor strength and the triceps surae muscles are normal, it is important that active exercises of the ankle dorsiflexors be performed to maintain them at the antigravity functional level. The calf muscles should also be passively stretched to prevent the development of equinus deformity; this is implemented by the use of a night bivalved cast, which holds the foot out of equinus and in neutral position. Progressive resistance exercises utilize activity graded in proportion to the strength of the involved muscles; their use is recommended in the residual stage of poliomyelitis to increase the strength and improve the endurance of such individual muscles or groups of muscles as a fair quadriceps or triceps surae or a fair plus hip abductor muscle to the maximum capacity. Whether progressive resistive exercises are of any permanent value when the motor strength of a muscle is

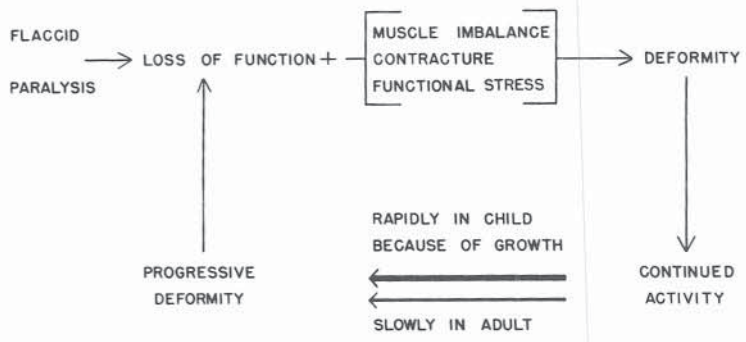


FIGURE 26–1 Principal factors involved in the progressive loss of function in the residual stage of poliomyelitis. (From Green WT, Grice DS: The management of chronic poliomyelitis. Instr Course Lect 1952;9:86.)

less than fair minus is doubtful; a poor quadriceps muscle cannot, through hypertrophy exercises, be improved to fair strength so that it can lift the leg against gravity. Correction of flexion deformity of the knee, however, may provide added strength by eliminating the need for the quadriceps muscle to work against deformity.

PASSIVE STRETCHING EXERCISES. Preventing contractual deformity is much simpler than its correction. When a limb is continuously maintained in one position, contracture and fixed deformity will develop as a result of the effects of gravity and dynamic imbalance of muscles. An ankle joint held in plantar flexion because of weak dorsiflexors and strong triceps surae will develop progressive equinus deformity if the ankle is not passively stretched into dorsiflexion every day. Passive stretching exercises should be performed gently, several times a day. In the presence of muscle imbalance, however, they are not adequate to prevent deformity, and other measures should be employed, such as the use of a removable bivalved long-leg night cast, which holds the foot in neutral position, and the wearing of a below-knee dorsiflexion-assist spring orthosis during the day. Later, during the chronic stage, muscle balance may be restored by transfer of muscles.

FUNCTIONAL TRAINING. The purpose of such a therapy program is to enable the patient to overcome the handicaps imposed by the physical disability. The residual deficit in function varies, depending on the extent and severity of paralysis. The needs of a growing child progressively change. In the residual stage the patient is taught how to use all of the available muscles in order to accomplish a task successfully. This is in contrast to the convalescent stage, when the patient is not allowed to substitute stronger muscles for weaker ones. For example, when the anterior tibial is poor in motor strength in the convalescent stage, the child is not permitted to use the toe extensors to dorsiflex the foot when active exercises are performed with the anterior tibial. In the chronic stage, when anterior tibial function is still poor, the child is taught how to dorsiflex the foot by using the toe extensors and peroneal muscles.

At times the activity of stronger muscles is suppressed in order to prevent the development of deformity. For example, an individual with normal sartorius, biceps femoris, and peroneal muscles but poor iliopsoas, medial hamstring, and anterior tibial muscles will walk with marked external rotation deformity of the foot and leg. It is important to supervise his gait, teaching him to suppress the eversion power of the peroneals and the externally rotating power of the biceps femoris and the sartorius to prevent the development of an external rotation deformity of the lower limb.

To teach a child merely to walk with crutches and orthoses is not satisfactory. The child should be instructed in activities of daily living, such as how to get in and out of chairs, open doors, and enter an automobile.

Orthoses and Other Apparatus. Use of an apparatus may be necessary during the asensitive period of the convalescent stage and the residual stage of poliomyelitis. The primary purposes of the orthosis are (1) to support the patient, enabling him to walk and increasing his functional activity, (2) to protect a weak muscle from overstretching, (3) to augment the action of weak muscles or to substitute for those completely lost, (4) to prevent deformity and malposition, and (5) to correct deformity by stretching certain groups of muscles that have been contracted. The support, substitution, and corrective mechanisms may be combined in a single apparatus. In general, dynamic splinting is more desirable than static splinting. For example, when the toe extensor and anterior tibial muscles are paralyzed and the triceps surae muscle is normal, a dorsiflexion-assist spring orthosis (which acts as an active substitute for the weak ankle dorsiflexors) is preferable to a below-knee caliper orthosis with a posterior stop that prevents plantar flexion of the ankle beyond neutral position. In paralysis of the gastrocnemius and soleus muscles, a plantar flexion–assist spring below-knee orthosis with a dorsiflexion stop at neutral position is prescribed (Fig. 26–2). In the presence of a flail ankle and foot, a double-action ankle joint (both plantar flexion–assist and dorsiflexion-assist) is provided, and a varus or valgus T-strap is added to the shoe, as necessary. Also, inside or outside wedges to the shoe are prescribed, depending on the deformity of the foot.

When the muscles controlling the knee are paralyzed, an above-knee orthosis with a drop-lock knee joint is prescribed. This type of orthosis provides knee stability for walking and can be unlocked during sitting. If genu recurvatum results from paralysis of the triceps surae in the presence of some strength of the quadriceps femoris, it can be controlled by an above-knee orthosis with a free knee joint constructed so as to prevent complete extension of the orthosis at the knee. Proper positioning of the thigh and calf bands will also check genu recurvatum. Genu varum or

FIGURE 26–2 Plantar flexion–assist below-knee orthosis with a dorsiflexion stop at neutral position.

knock-knee pads are padded as necessary. When flexion deformity of the knee is present as a result of dynamic imbalance between the hamstrings and quadriceps femoris muscles, a well-padded anterior knee pad is prescribed. An Engen extension knee orthosis is worn at night to correct flexion deformity of the knee.

When the muscles controlling the hip are weak, stability of the hip joint can be provided by an ischial weightbearing thigh socket; crutches are used if necessary. Rotational alignment of the lower limbs is obtained by the addition of rotation straps or twisters. Ordinarily the patient will walk better without a pelvic band and drop-lock hips; however, in a young child with gluteus maximus paralysis, these devices may be used temporarily for balance. Often the spine will also require support. When upright posture is resumed, the abdominal muscles will overstretch, and severe lumbar lordosis and paralytic scoliosis will develop. Any asymmetric involvement of the abdominal and trunk musculature should always be carefully noted. An abdominal corset support with metal stays often serves to control abdominal muscle paralysis. If the trunk extensors are weak, a spinal orthosis with an abdominal corset is provided. If the spine is unstable and collapsing, it may be supported by a molded plastic body jacket constructed from a plaster-of-Paris cast made with the patient standing, with traction provided by a head sling. In paralytic scoliosis, usually a Milwaukee brace is worn, provided that the lower limb paralysis is not very extensive and that wearing such an appliance does not prevent ambulation. In such instances, the Milwaukee brace is used intermittently during periods of recumbency or sitting, or both.

In the upper limb, the paralyzed shoulder muscles, particularly the deltoid, are best protected from the effects of gravity with a sling; this allows functional use of the forearm and hand. During the initial period of 6 to 8 weeks, an abduction shoulder splint may be worn at night and during part of the day to prevent overstretching of the deltoid muscle; this is particularly indicated when there is associated paralytic subluxation or dislocation of the shoulder joint. A cock-up wrist splint is used when the wrist extensors are paralyzed, and an opponens splint is used when there is weakness of the opponens of the thumb. When there is paralysis of the intrinsic muscles of the hand, hyperextension of the metacarpophalangeal joints is prevented by a knuckle-bender dynamic splint.

Certain general principles should be followed in regard to the use of an apparatus in patients with poliomyelitis. Whenever satisfactory recovery of function is expected, an orthosis should be used with caution in the lower limbs, since its use will tend to produce an abnormal gait pattern. Thus, during the early convalescent period, use of an orthosis should be deferred until after maximum recovery of muscle function has taken place. Locomotion without an orthosis but with the support of crutches should be attempted in order to stimulate active muscular function through the exercise of walking. Use of an orthosis should not, however, be postponed if deformities appear likely to develop incident on the stresses of weightbearing. The needs of each patient are different, and the use of a lower limb orthosis depends on the severity of the muscle weakness and the degree of dynamic imbalance of the muscles. If there is extensive paralysis of the lower limbs, use of an orthosis may be the only means of achieving stance and locomotion.

In general, use of an orthosis should be as minimal as the condition permits. For example, when a patient with paralysis of both lower limbs is fitted with two above-knee orthoses, she will also need to use two crutches to walk. If she is to use two crutches, she can do as well with an above-knee orthosis on one leg only, for only minimal motor strength is required of the other leg to walk without an orthosis. During the stance phase on the leg without the orthotic support, the tripod base is completed by the two crutches; the knee is stabilized by being locked in hyperextension. Fair motor strength in the ankle dorsiflexors and hip flexor muscles will allow clearance of the lower limb in the swing phase. To prevent fatigue, however, bilateral above-knee orthoses are used.

It is imperative to explain to the patient the reasons for using an orthosis. She should understand clearly that wearing the orthosis will help her at this stage of the disease, and that, at a later date, it may be discarded following training or reconstructive surgery. For example, the use of a dorsiflexion-assist below-knee orthosis may be unnecessary after a successful anterior transfer of the peroneal tendons, or an opponens splint may be discarded after a satisfactory opponens tendon transfer. In addition, when the child becomes an adult, she may no longer need an above-knee orthosis to prevent genu recurvatum.

The continued use of an orthosis should be reevaluated regularly. Before advising that use of an orthosis be discontinued, the clinician should be quite certain that there is no possibility for the development of progressive deformities and that the level and quality of functional performance will not deteriorate.

Surgery. A multitude of operative procedures can be employed both in the correction of paralytic deformities and in the total physical rehabilitation of the child with poliomyelitis. These procedures may include fasciotomy, capsulotomy, tendon transfers, osteotomy, and arthrodesis. Leg length inequality commonly occurs in poliomyelitis as a result of shortening in the paralyzed leg.

PRINCIPLES OF TENDON TRANSFER. Tendon transfer entails shifting the insertion of a muscle from its normal attachment to another site to replace the active muscular action that was lost by paralysis and to restore dynamic muscle balance. The procedure was originally described by Nicoladoni in 1882. Many surgeons have devised various types of tendon transfers and established their usefulness. Lange, Velpeau, Vulpius, Codivilla, Mayer, Biesalski, Goldthwait, Ober, Steindler, Bunnell, and Green are some who may be mentioned.* The term *tendon transplantation* should not be used interchangeably with the term *tendon transfer,* as the two are not synonymous. Tendon transplantation refers to the excision of a tendon and its use as a free graft. In *muscle transplantation,* both the origin and the insertion of a muscle are detached, and the entire muscle with its intact neurovascular supply is transplanted to a completely new site.

The basic principles of tendon transfers have been outlined by Green[116] and are listed below.

*See references 26, 41, 69, 115, 189, 216–219, 241, 246, 303–311, 328.

1. The muscle to be transferred must have adequate motor strength to carry out the new function. As a rule, the motor rating of the muscle should be good or normal to warrant transfer. The function that the transferred muscle is intended to perform is another consideration. In the lower limb, for example, in the presence of dropfoot, anterior transfer of the peroneus longus is adequate to produce effective ankle dorsiflexion, whereas in calcaneus limp, posterior transfer of the peroneus longus alone to the os calcis is not sufficient to substitute for the gastrocnemius-soleus action, and the additional action of two or three motors such as the flexor digitorum communis and anterior tibial muscles is required. Ordinarily one grade of motor power is lost after a muscle is transferred.

2. The range of motion of muscles on contraction is an important consideration. This range must be similar to that of the muscles for which they are being substituted; also, whenever muscles are transferred in combination, their range of contraction should not differ significantly. The transfer of antagonistic muscles ordinarily is not as effective as the transfer of muscles having similar function or corollary activity. However, with meticulous postoperative care, antagonistic muscles may be transferred effectively with good results. The posterior transfer of the anterior tibial to the os calcis and of the hamstring muscles to the patella are common examples of such antagonistic transfers.

3. In choosing the muscles for transfer, the surgeon must weigh loss of original function that will result from the tendon transfer against the gains to be obtained. For example, in the presence of hip flexor weakness, the hamstring muscles should not be transferred to the patella for quadriceps paralysis, as loss of active knee flexion added to the lack of hip flexion will be a greater disability. Whenever possible, muscle balance must be restored. Ideally, a deforming muscle force must be shifted so as to substitute for an essential weakness. In the foot and ankle, for example, the muscles of inversion and eversion and those of plantar flexion and dorsiflexion should be balanced. A common pitfall is transfer of the peroneus longus muscle posteriorly to the os calcis in the presence of a strong anterior tibial muscle. Normally, the anterior tibial muscle dorsiflexes the first metatarsal and the peroneus longus opposes this action. With posterior transfer of the peroneus longus, the unopposed anterior tibial gradually causes the first metatarsal to ride up, producing a dorsal bunion. Thus the peroneus longus should not be transferred to the os calcis unless the anterior tibial is shifted from its insertion on the first metatarsal to the midline of the foot.

4. The joints on which the transferred muscle is to act should have functional range of motion. All contractual deformity should be corrected by wedging casts or soft tissue release prior to tendon transfer. An anterior transfer for dropfoot, for example, should not be performed in the presence of equinus deformity of the ankle.

5. A smooth gliding channel with adequate space must be provided for excursion of the tendon in its new location. The paratenon and synovial sheath are preserved over the tendon surface during dissection. It is preferable to pass the tendon beneath the deep fascia through tissues that permit free gliding, rather than subcutaneously. A wide portion of the intermuscular septum is excised whenever muscles are passed from one muscle compartment to another. Sufficient space should be provided for the tendon so that adhesions will not form. An Ober tendon passer of appropriate size should be used to redirect the tendon to its new insertion; the tendon passer spreads the tissues and prevents binding.

6. The neurovascular supply of the transferred muscle must not be damaged while transferring the tendon. The surgeon must be careful not to denervate the muscle while freeing it for redirection. When the tendon is pulled up from the distal wound into the proximal incision, traction should not be applied to the origin of the muscle. Stretching of the motor nerve can be prevented by using a double-hand technique: with a moist sponge, the proximal segment of the tendon is held steady, while with another sponge, traction is applied on its distal segment. Acute angulation or torsion of the neurovascular bundle is another cause of injury. Gentle handling is imperative to preserve innervation and function of the transferred muscle.

7. In the rerouting of the tendon a *straight line* of contraction must be provided between the origin of the muscle and its new insertion. Angular courses and passages over pulley systems should be avoided. To allow adequate freeing of the muscle toward its origin, the incision over the belly of the muscle must be long and proximally located.

8. The tendon should be reattached to its new site under sufficient tension so that the transferred muscle will have a maximal range of contraction. The transferred muscle should be tested during the operation to ensure that it will hold the part in optimal position. In the lower limb, where weightbearing forces are involved, the tendon is ordinarily attached to bone, whereas in the upper limb it is sutured to the tendon. An important technical detail is scarification of the distal segment of the tendon that is to be anchored to a bone or tendon; this is achieved by excising the sheath and paratenon and "roughening" the tendon by scraping and crosshatching it with a knife. To diminish any tension on the tendon while it is healing, the position of immobilization in a cast should allow the transferred tendon to be in a relaxed attitude. For example, when the flexor carpi ulnaris is transferred to the extensor carpi radialis longus, the tension on the tendon should be sufficient to hold the wrist in 30 degrees of dorsiflexion. However, when the cast is applied, the wrist is immobilized in the overcorrected position of 45 to 50 degrees of dorsiflexion.[116]

POSTOPERATIVE CARE AND TRAINING. Postoperative care and training are fundamental to achieving a good result. The following principles, given by Green, should be followed meticulously.

First, the age of the patient at the time of tendon transfer is an important preoperative consideration. The child should be old enough, preferably over 4 years of age, to cooperate in the training of the transfer. A delay in tendon transfer in the presence of muscle imbalance will lead to progressive deformity. Usually, conservative measures should be undertaken to control deforming factors, but early surgery may be indicated when a delay of tendon transfer would result in increasing structural deformity. A common example is the rapid development of progressive calcaneus deformity of the foot with paralysis of the gastrocnemius-soleus muscles and strong ankle dorsiflexors. An early posterior transfer will prevent the development of a deformed foot.

Support of the part in overcorrected position should be continued until the muscle has assumed full function and there is no tendency for the deformity to recur. A bivalved cast will hold the transferred tendon in a relaxed position.

It is best to teach the patient preoperatively to localize active contraction in the muscle to be transferred. Active exercises are continued postoperatively as soon as the reaction to surgery and pain has subsided. The surgeon should assist the physical therapist during the initial exercises. When tendon transfer is combined with arthrodesis, muscle reeducation is delayed until adequate bony union has taken place.

The patient is instructed to contract the transferred muscle voluntarily, moving the part through the arc of motion that was the original normal action of the muscle, while the therapist manually guides the part to move in the direction that is intended to be provided by the transfer. For example, when the peroneus longus muscle is transferred anteriorly to the base of the second metatarsal, the active motion called for is eversion in combination with guided dorsiflexion, or, if the anterior tibial muscle has been transferred posteriorly to the os calcis, active inversion is combined with guided plantar flexion of the ankle; in anterior transfer of the hamstrings to the patella for quadriceps femoris paralysis, the patient is placed in a side-lying position and asked to extend the hip actively (using the hamstrings) as the knee is guided into extension. If the flexor carpi ulnaris has been transferred to the extensor carpi radialis longus, the wrist is gently guided into extension as the patient deviates it ulnarward. With one hand the therapist should palpate the belly and tendon of the transferred muscle to ensure its contraction. In the beginning the exercises are performed in the bivalved cast. Motion of the concerned joint is executed slowly, steadily, and smoothly through as full a range as possible. Soon the limb is taken out of the cast and is properly positioned, and measures are taken to prevent stretching of the tendon out of its resting position.

Occasionally the patient is unable to contract the transferred muscle actively and has difficulty in "finding" it. To enable him to use the transfer actively and to assist him in acquiring the feeling desired, the therapist may exert gentle mild tension on the transferred tendon, have the patient shift positions during attempts at active contraction, or advise him in the use of corollary motions. If difficulty in "finding" the transfer persists after 2 weeks, electrical stimulation may be employed to initiate contraction as the patient himself attempts to use the muscle. After a few sessions the patient begins to "feel" the transfer and to contract it voluntarily.

As soon as the patient is able to contract the transferred muscle actively, exercises in the direction of the original action of the muscle are discontinued and only those motions in the new function provided by the transfer are performed.

When the transferred muscle develops poor motor strength—that is, it can carry the part through the full range of motion with gravity eliminated—the physical therapist instructs one of the parents to perform the exercises with the child. The exercise regimen is supervised by the physical therapist and the surgeon, who check it at weekly or biweekly intervals.

Initially the limb should be retained in the bivalved cast for support except during the exercise periods. As soon as the motor strength of the transfer becomes fair, use of a bivalved cast during the day is gradually discontinued. Controlled activities are permitted to develop function. These activities are permitted sooner in the upper than in the lower limb. The age and dependability of the patient are other considerations. Resistive exercises to develop power are begun whenever the transfer has a normal range of action and is fair in strength. It is also important to exercise the antagonistic muscles to prevent disuse atrophy.

The next stage of training is incorporation of the transfer into the new functional pattern. This is particularly important in the lower limb, in which the muscles are concerned with gait. For example, the action of the peroneus transfer may be good, dorsiflexing the ankle through full range and taking moderate resistance, yet during locomotion voluntary control over the transfer may be "lost" and the patient may walk with a dropfoot gait. The transition to walking requires diligent supervision. Of particular importance is the use of crutches, which protect the limb from undue strain and at the same time allow the patient to be taught the use of the transfer and to become accustomed to it. First the patient is asked to take a single step, with the therapist ensuring that the muscle contracts and dorsiflexes the ankle. As soon as the transfer functions throughout all the phases of a single step, the walking periods are gradually increased until the normal gait pattern becomes a conditioned reflex.

The use of orthoses in the postoperative period should be judicious and for specific reasons. Orthotic support protects the part and allows early activity. This is indicated particularly when paralysis is extensive, as in myelomeningocele. In a posterior transfer to the os calcis, for example, a plantar flexion-assist orthosis with a dorsiflexion stop at right angles with crutches may be used to aid developing function in the transfers and prevent stretching. However, standing and walking exercises are also performed without the brace to stimulate function in the transfer.

Prolonged use of a bivalved night cast is very important to prevent development of a contractual deformity that would oppose the action of the transfer—equinus deformity of the ankle, for example, in the instance of anterior transfer for dorsiflexion. From the beginning, daily stretching exercises should be a part of the exercise regimen. Stretching and night support are continued over a long period of time, until the muscle has developed full strength and there is balanced function between the agonistic and antagonistic muscles with no tendency for recurrence of the original deformity. In fact, stretching and active exercises should be a simple rule of daily living.

Arthrodesis to provide stability and correct osseous deformity may be indicated, particularly in the foot. However, if dynamic balance is established prior to development of structural deformity, arthrodesis may be unnecessary. When it is necessary to combine arthrodesis with tendon transfer, muscle reeducation must be delayed until adequate bony union has taken place.

The Hip

SOFT TISSUE CONTRACTURE

The common deformity of the hip secondary to soft tissue contracture is one of flexion, abduction, and external rota-

tion. Several factors must be considered in its pathogenesis. During the acute and convalescent stages of poliomyelitis, the patient lies supine in the so-called frog-leg attitude with the hips flexed, abducted, and externally rotated, the knees flexed, and the feet in equinovarus posture. This position is assumed because of spasm of the hamstrings, hip flexors, tensor fasciae latae, and hip abductor muscles and because of the force of gravity acting on the flail lower limbs. Maintenance of the lower limbs in malposture results in permanent shortening of the soft tissues. Contracture of the intermuscular septa and enveloping fasciae occurs first. This fact can be easily observed at surgery. Upon sectioning of the contracted fasciae that cover normal muscle fibers and retraction of the cut edges of the fascia 2 to 3 cm, the underlying muscle tissue will be found to be in relaxed condition when it is elevated with tissue forceps. Partially paralyzed muscle becomes shortened because of contracture of the involved fibrosed muscle fibers scattered throughout the normal muscle tissue. Adaptive shortening of normal muscle occurs later. Structural bony deformity develops with growth in the presence of soft tissue contracture and dynamic muscle imbalance.

The iliotibial band (or tract) is the thickened lateral portion of the fascia lata located along the entire lateral aspect of the thigh and extending from the greater trochanteric region to below the knee. Superiorly, the iliotibial band is attached to the iliac crest by three prongs: a middle one through the aponeurosis over the gluteus medius, an anterior one through the tensor fasciae latae, and a posterior one through the gluteus maximus (Fig. 26–3).

Throughout its extent on the lateral aspect of the thigh, the iliotibial tract is continuous on its deep surface with the lateral intermuscular septum, through which it is firmly attached to the linea aspera on the posterior aspect of the femur. At the knee joint level, fascial expansions from the anterior border of the iliotibial tract join expansions that emanate from the quadriceps muscle to form the lateral patellar retinaculum. The lower end of the iliotibial band is attached to the lateral condyle of the tibia and the head of the fibula. Proximally the iliotibial band is located in a plane that is anterior and lateral to the axis of the hip joint, whereas distally, in a normal limb, the iliotibial tract inserts on the tibia in front of the axis of the knee joint. Irwin states, however, that the lower part of the iliotibial tract lies in a plane posterior and lateral to the axis of the knee joint.[170]

Contracture of the iliotibial band may contribute directly or indirectly to the development of the following deformities.[99,103,170,174,346]

The Lower Limb

FLEXION, ABDUCTION, AND EXTERNAL ROTATION CONTRACTURE OF THE HIP. The shortened iliotibial band, which is in a plane anterior and lateral to the hip joint, will draw the femur into flexion and abduction at the hip, with the pelvis as the fixed point. External rotation deformity is due to maintenance of the malposture of the frog-leg position. The related muscles—the tensor fasciae latae, reflected head of the rectus femoris, sartorius, and external rotators of the hip—undergo myostatic contracture if the fascial contracture is not corrected. The fixed soft tissue contracture will cause anteversion of the proximal femur.

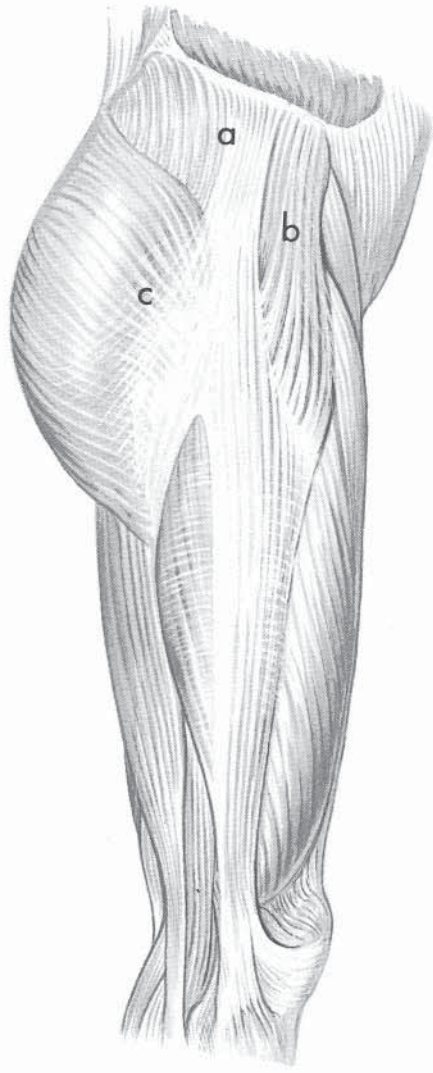


FIGURE 26–3 The three-pronged attachment of the upper part of the iliotibial band to the iliac crest. There is a middle prong (*a*) through the aponeurosis over the gluteus medius, an anterior one (*b*) through the tensor fasciae latae, and a posterior one (*c*) through the gluteus maximus. Proximally, the location of the iliotibial tract is anterior and lateral to the axis of the hip, whereas inferiorly, in the normal knee, it inserts on the tibia well in front of the axis of the knee joint.

FLEXION AND VALGUS DEFORMITY OF THE KNEE AND EXTERNAL TORSION OF THE TIBIA. The iliotibial band crosses lateral to the axis of the knee. When it is contracted a force is exerted on the lateral aspect of the joint and the tibia is gradually abducted on the femur. Its deforming action resembles that of a taut string on the concavity of an archer's bow. Irwin proposed that flexion deformity of the knee developed as a result of the location of the band in a plane posterior to the axis of motion of the knee joint.[170] However, subsequent studies have not supported this observation. The short head of the biceps takes its origin in part from the intermuscular septum, which in turn is attached to the iliotibial band. Flexion deformity of the knee will develop as a result of spasm and subsequent myostatic contracture of the short head of the biceps. Prolonged maintenance of the knee in flexion will cause contracture of the patellar retinacula and soft tissues behind the knee.

EXTERNAL TORSION OF THE TIBIA AND SUBLUXATION OF THE KNEE JOINT. The pull of the laterally located iliotibial band and the short head of the biceps femoris gradually rotates the tibia and fibula externally on the femur. When the contracture is not controlled, the deforming forces will cause posterolateral subluxation of the knee with displacement of the fibular head into the popliteal space.

POSITIONAL PES VARUS. Positional pes varus results from an ill-fitted orthosis that fails to compensate for the external tibial torsion. The axes of the knee and ankle joints do not occupy the same horizontal plane in external torsion of the tibia. When an above-knee orthosis manufactured with these joints in the same horizontal plane is fitted to a limb with external tibial torsion, the appliance will force the foot into varus position so that the ankle is in line with the knee joint. Initially the varus deformity is a purely functional one (the foot will assume normal alignment when the lateral upright of the orthosis is allowed to rotate externally on the thigh); it will later become fixed, owing to permanent shortening of the soft tissues and adaptive osseous changes in the tarsal bones.

The Pelvis and Trunk

PELVIC DEFORMITY, LUMBAR SCOLIOSIS, AND SUBLUXATION OF THE CONTRALATERAL HIP. In abduction deformity of the hip due to iliotibial band contracture, the pelvis is level with or at a right angle to the vertical axis of the trunk as long as the affected hip is maintained in abduction; however, when it is brought parallel to the vertical axis of the body in the weightbearing position, the pelvis is forced to assume an oblique position. This pelvic obliquity is due to contracture below the iliac crest. A lumbosacral scoliosis, convex to the low side of the pelvis, simultaneously develops. The contralateral hip will subluxate.

EXAGGERATED LUMBAR LORDOSIS. Exaggerated lumbar lordosis is produced when there is bilateral flexion contracture of the hips. It is a compensatory response to the increased pelvic inclination when the trunk assumes an upright position.

PELVIC OBLIQUITY. *Fixed pelvic obliquity is a common deformity following poliomyelitis and may be caused by suprapelvic, intrapelvic, or infrapelvic abnormalities. In an extensive study conducted in Korean patients, pelvic obliquity was classified into two major types and four subtypes relative to the resultant scoliosis.*[8a] *In major type I, the pelvis is lower on the short-leg side and the authors recommended ipsilateral abductor fasciotomy and, at times, contralateral lumbodorsal fasciotomy to correct the deformity. In type II deformities, the pelvis is high on the short-limb side owing to adduction contracture of the ipsilateral hip, abduction contracture of the contralateral hip, or ipsilateral lumbodorsal fascial contracture.*

Treatment. Static malpostural deformities of the lower limbs in the acute and subacute stages of poliomyelitis can be prevented by the use of bivalved casts, which maintain the joints in neutral position. A horizontal bar in the posterior half of the cast or a rotational strap will control malrotation at the hips. The knees should be in slight flexion to prevent genu recurvatum. Passive exercises are performed to maintain full range of joint motion.

Minimal contracture of the iliotibial band can be corrected by passive stretching exercises, which follow the same steps as in the Ober test. They can also be performed with the patient supine and the hip that is to be stretched hanging over the edge of the bed. In the older patient the iliotibial band can be stretched by the following exercise: The patient should stand sideways about 2 feet away from the wall with the hip that is to be stretched placed facing it. With the feet on the ground and the legs together, the hip is brought toward the wall to the count of 10 and is then returned to the original position. This exercise should be performed for 20 repetitions, three times a day.

When the iliotibial band is contracted to such a degree that fixed deformity at the hip and knee with tilting of the pelvis has resulted, correction cannot be obtained by manipulative stretching or application of a series of plaster casts. The pelvis cannot be locked securely enough to permit stretching forces to be exerted on the shortened iliotibial band; instead, the pelvis will be tilted into an oblique and hyperextended position, stretching the lateral and anterior abdominal muscles on the side of the contracture.

Surgical intervention is the only way to correct the deformity. The shortened soft tissues must be sectioned proximally as well as distally by combining Ober's fasciotomy with Yount's procedure.[243,346] As stated previously, the primary cause of the deformities is contracture of the intermuscular septa, the enveloping fascia, and the fibrosed muscular tissue in the partially involved muscles. Normal muscle tissue should not be divided.

Ober's and Yount's fasciotomies are performed as follows. Both lower limbs and hips are prepared and draped sterilely. *Ober's fasciotomy* is performed through an incision that starts at the junction of the posterior and middle thirds of the iliac crest and then extends distally to the anterior superior iliac spine, where it swings posterolaterally for a distance of 10 cm. The wound flaps are mobilized to expose the sartorius, rectus femoris, tensor fasciae femoris, and gluteus medius and minimus muscles. The enveloping fascia of these muscles, the intermuscular septa, the intervening fibrosed muscular tissue, and the iliotibial band are sectioned as far back as the greater trochanter. The Ober and Thomas tests are performed to determine by palpation the presence of any tight bands, which, if present, are divided. Normal muscle tissue and the anterior capsule of the hip joint should not be divided. The contracted fibers of Bigelow's ligament can be released without entering the hip joint.

Yount's procedure consists of excision of a segment of the iliotibial band and of the lateral intermuscular septum in the distal thigh. A midlateral longitudinal incision is made beginning immediately above the knee joint line and extending cephalad for a distance of 10 cm. The subcutaneous tissue is divided and the wound flaps are mobilized by blunt dissection to expose the anterolateral aspect of the thigh in its distal one-fourth. Next, a 7-cm block of the iliotibial band, the fascia lata covering the vastus lateralis muscle, and the lateral intermuscular septum are excised. It is important to divide the lateral intermuscular septum down to the femur. If it is contracted and contributes to flexion deformity of the knee, the lateral patellar retinaculum is also divided.

In severe cases with lateral rotatory subluxation of the knee, the biceps femoris muscle is lengthened by the fractional method, extreme care being taken not to injure the common peroneal nerve (Plate 26–1). This can be per-

Text continued on page 1336

Fractional Lengthening of Hamstrings

OPERATIVE TECHNIQUE

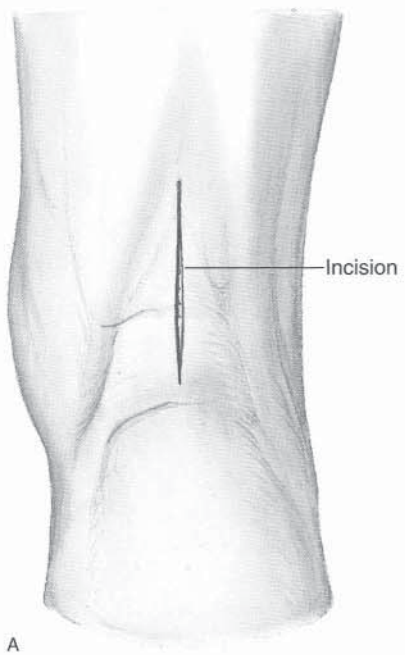
A, The patient is positioned prone with a pneumatic tourniquet placed high on the proximal thigh. A 3- to 4-inch-long midline incision is made, starting just proximal to the popliteal crease. The subcutaneous tissue is divided and the incision carried to the deep fascia. The posterior femoral cutaneous nerve will be in the proximal aspect of the wound and should not be damaged.

B, The deep fascia is incised and the hamstring tendons are identified by blunt dissection. It is imperative to divide the tendon sheath of each hamstring tendon separately and mark it with 000 silk sutures for meticulous closure later.

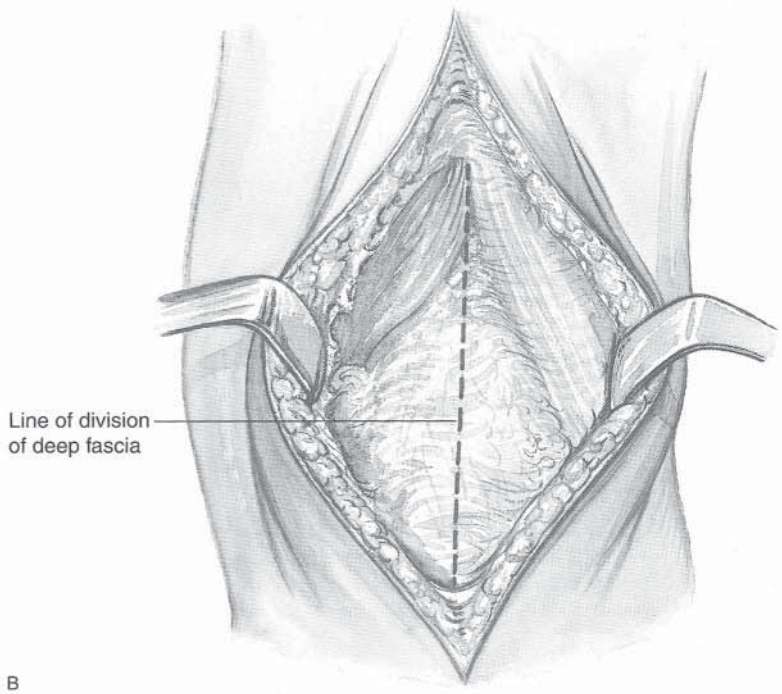
C, In the lateral compartment of the wound, the biceps femoris tendon is exposed. It should be gently dissected away from the common peroneal nerve, which lies on its posteromedial surface. A blunt instrument, such as a staphylorrhaphy probe or a joker, is passed deep to the biceps tendon.

D, With a sharp knife, the tendinous portion of the biceps femoris is incised transversely at two levels 3 cm apart, leaving the muscle fibers intact. The tendon is lengthened in continuity by straight-leg raising with the knee in extension.

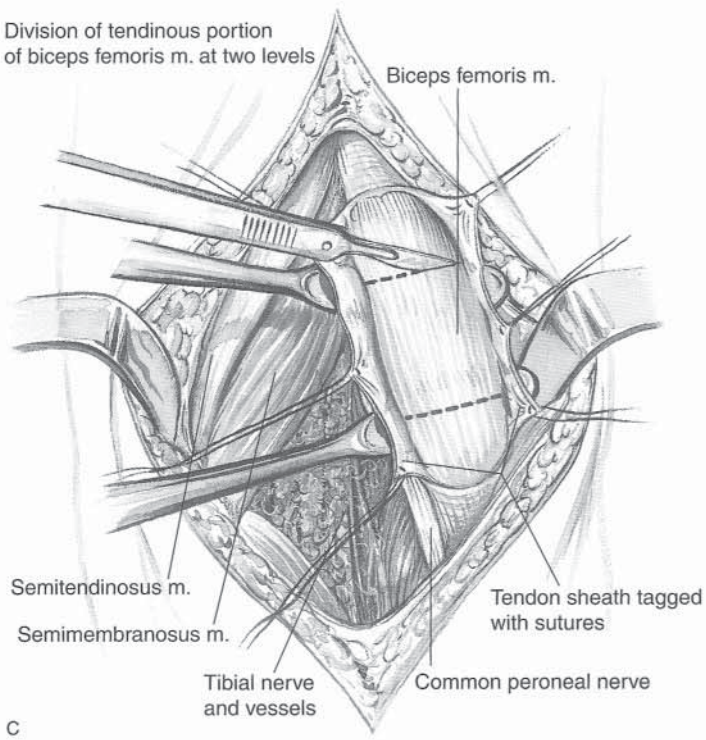
PLATE 26–1. Fractional Lengthening of Hamstrings



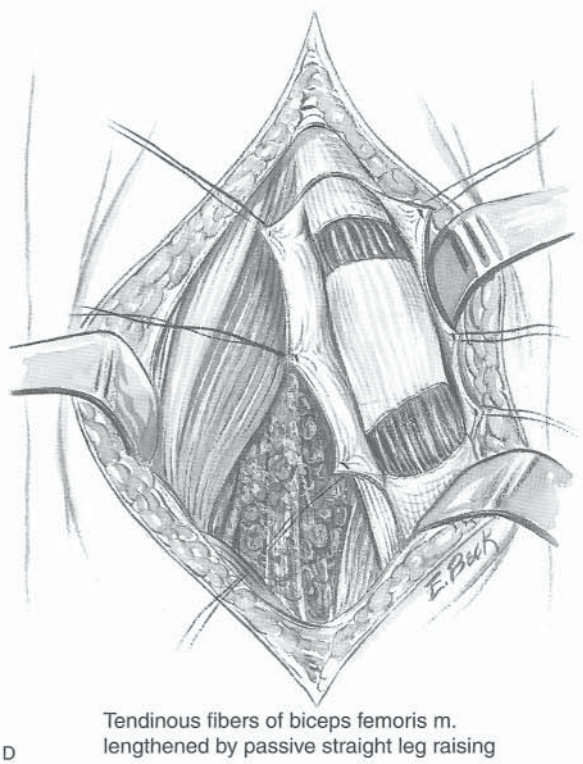
A



B



C



D

Fractional Lengthening of Hamstrings *Continued*

E, The semimembranosus tendon is then isolated in the medial compartment of the wound. The tendinous portion lies on its deep surface; to expose it the muscle is everted. The tendinous fibers are divided at two levels (similar to the biceps tendon), leaving the muscle fibers in continuity. Again, by extending the knee and flexing the hip, a sliding lengthening of the semimembranosus is performed.

F, Next, the semitendinosus is exposed. The tendinous portion is divided proximal to the musculotendinous junction.

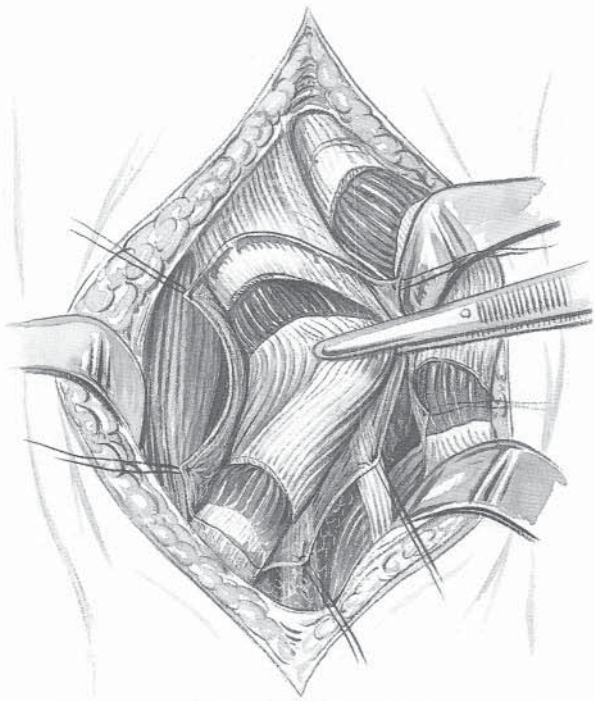
G, If inadvertently the semitendinosus tendon ruptures, a Z-plasty is performed.

H, The tendon sheath of each tendon is meticulously closed. The deep fascia is not sutured. The subcutaneous tissue and skin are closed in routine manner and bilateral long-leg casts are applied with the knees in full extension.

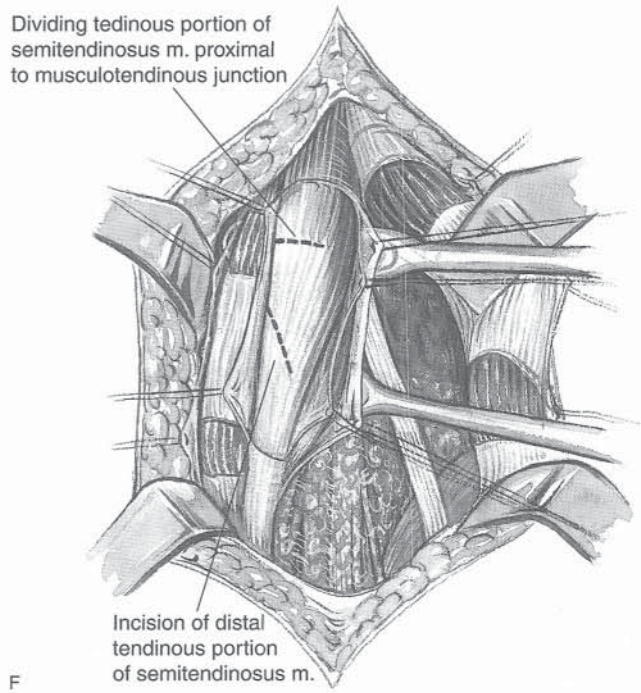
POSTOPERATIVE CARE

While the patient is in the solid cast, straight-leg-raising exercises are performed 15 times, once a day, for further stretching of the hamstrings. At the end of 3 to 4 weeks the casts are removed and new above-knee bivalved casts are made. Active and passive exercises are performed to develop knee flexion, first with the patient side-lying, with gravity eliminated, and then against gravity. The motor strength of the quadriceps is developed. Whenever functional range of motion of the knees is present, the patient is allowed to be ambulatory with appropriate support.

PLATE 26–1. Fractional Lengthening of Hamstrings



E Forceps everting semimembranosus m. to expose tendinous portion. Division at two levels



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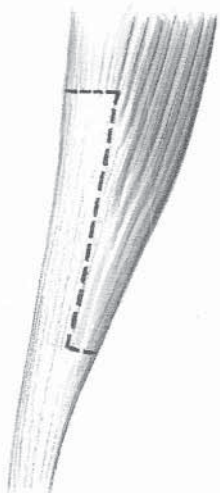
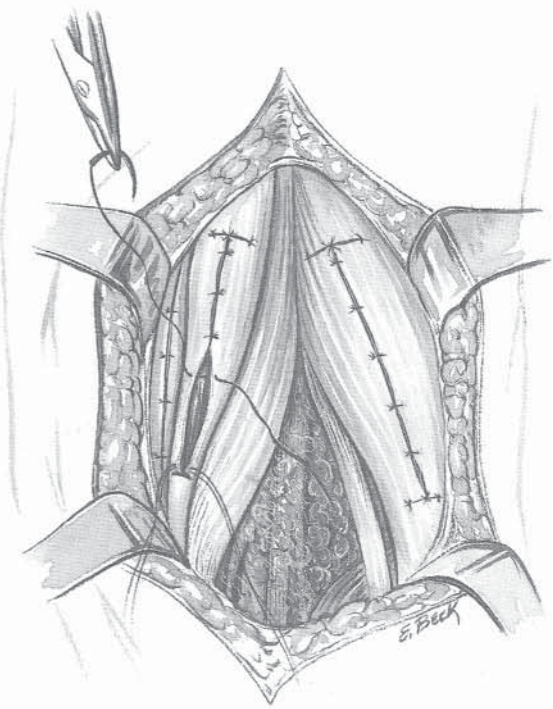


Alternate Z-plasty

G



H Separate meticulous closure of each tendon sheath
Deep fascia is not sutured

formed through the same incision. Then an attempt at reduction is made by forcibly extending and internally rotating the knee. Often a Z-lengthening of the fibular collateral ligament will be necessary to achieve reduction.

Both the hip and the thigh wounds are closed routinely. Bilateral long-leg casts are applied, holding the knees in full extension. Metal rings are anchored on the cast on both its anterior and its posterior aspects so that the patient can be placed in suspension traction. One set of rings is placed in the distal one-fourth of the leg and another set of rings in the proximal one-fourth of the leg. Rotational straps can be added to the plaster cast if necessary. The patient is placed on two or three half-mattresses so that the lower limbs can hang free at the edge of the mattress and the hips can be hyperextended or flexed by suspension (Fig. 26–4). An infant or small child can be placed on a bent, hyperextended Bradford frame to achieve the same result. The opposite lower limb is flexed at the hip to obliterate the lumbar lordosis. The affected limb is gradually hyperextended, adducted, and internally rotated at the hip, stretching out all remaining contractual deformity. The same position of the hips can be achieved with the patient prone or supine. In bilateral cases the hips are alternated several times a day. Manipulative stretching exercises are performed three times a day. Meticulous observation of circulation and sensation in the toes is imperative, especially if excessive shortening of neurovascular structures was observed at operation.

In myelomeningocele patients with impaired sensation, stretching by the method described may cause pressure sores. Atrophied bones of these children may also be fractured easily by vigorous manipulations or stretching procedures.

Passive stretching by the suspension-traction method is continued for a period of 3 weeks. As the child grows, with progressive longitudinal growth of the femur, contracture of the iliotibial band will recur unless passive stretching exercises and proper positioning of the joints in bivalved casts are continued during periods of growth.

GLUTEUS MEDIUS PARALYSIS

When the hip abductor muscles are paralyzed, the trunk will sway toward the affected side and the contralateral side of the pelvis will drop during the weightbearing phase of gait. Lateral stability of the hip joint is best achieved by transferring the *iliopsoas muscle* from the lesser trochanter to the greater trochanter (Plate 26–2). The author commonly performs the Sharrard modification of the Mustard iliopsoas transfer, making the hole in the ilium as far posteriorly as the nerve supply to the iliacus will allow.[235,236] The importance of using a nerve stimulator while transferring the iliopsoas muscle cannot be overemphasized. The hip should be protected with crutches until the transferred iliopsoas is fair plus or good in motor strength and the Trendelenburg test is negative. During this period the patient should sleep in a bivalved hip spica cast, which maintains the hip in 40 to 60 degrees of abduction. Active hip abduction exercises should be performed diligently, with the child graduating from the supine position to side-lying against gravity, and then to a standing Trendelenburg position.

The *external oblique abdominal muscle* can be used to restore hip abduction power. Lowman used part of the external oblique muscle and attached it to the greater trochanter with a strip of fascia lata.[202–204] Thomas, Thompson, and Straub transferred the entire muscle belly of the external oblique.[315] The remaining abdominal muscles (rectus abdominis, internal oblique, and transverse muscles) will maintain integrity of the abdominal wall. The author has had no personal experience with external oblique muscle transfer for paralysis of the hip abductors. Physiologically, the procedure is sound; for details of operative technique, the reader is referred to the original article.[315] Also, the tensor fasciae latae muscle may be transferred posteriorly on the iliac crest to increase hip abduction strength.[193]

GLUTEUS MAXIMUS PARALYSIS

Instability of the hip and exaggerated lumbar lordosis result from paralysis of the gluteus maximus muscle. In gait, the trunk lurches backward when the body weight is borne on the affected side. When the hip flexor muscles are of normal strength, increasing flexion deformity of the hip will develop.

For motor evaluation of the gluteus maximus muscle, the patient is placed prone, with the lower limbs hanging off the examining table. The knee is in flexion to eliminate action of the hamstrings. The patient is asked to extend the hip against gravity and manual resistance. The position also allows the examiner to evaluate the degree of flexion deformity of the hip when it is extended passively. If the patient is unable to lift the thigh against gravity, he or she is placed in a side-lying position to eliminate the force of gravity. Any abduction contracture is best determined by the Ober test, as the degree of hip abduction noted on maximal extension of the hip in prone position is not as accurate.

In gluteus maximus paralysis, stability of the pelvis may be achieved by adding posterior gluteal crisscross straps between the pelvic band and the thigh band of the above-knee orthosis. An alternative method is to discard the pelvic band and fit an ischial weightbearing quadrilateral socket to the upper thigh segment of the orthosis. Often, however, the additional support of one or two crutches is required.

Muscle transfers to restore gluteus maximus function are not always successful and should be undertaken only after considerable deliberation. Lange transferred the erector spinae muscle to the greater trochanter, using silk sutures to obtain length.[183,189] Ober and Hey Groves used a strip of fascia lata to attach the erector spinae muscle to the greater trochanter.[127,240]

The technique of Ober was further improved by Barr, who used a wide strip of fascia lata, including the iliotibial tract and tensor fasciae latae muscle (Fig. 26–5).[11] Contractures about the hip, such as fascia and tight intermuscular septa, are released, particularly those that are anterior and lateral to the hip joint. Complete mobilization of the iliotibial tract and shift of its pull laterally to the greater trochanter removes a major deforming force. Release of contracted investing fascia about the shortened erector spinae muscle permits rotation of the pelvis to a nearly normal position and diminishes the severity of fixed lumbar lordosis.

Malrotation of the limb is prevented and corrected by transfer of the insertion of the tensor fasciae latae into the greater trochanter. Stability of hip is provided if there is power in the erector spinae and tensor fasciae latae muscles, which act in conjunction as a digastric muscle transfer. The

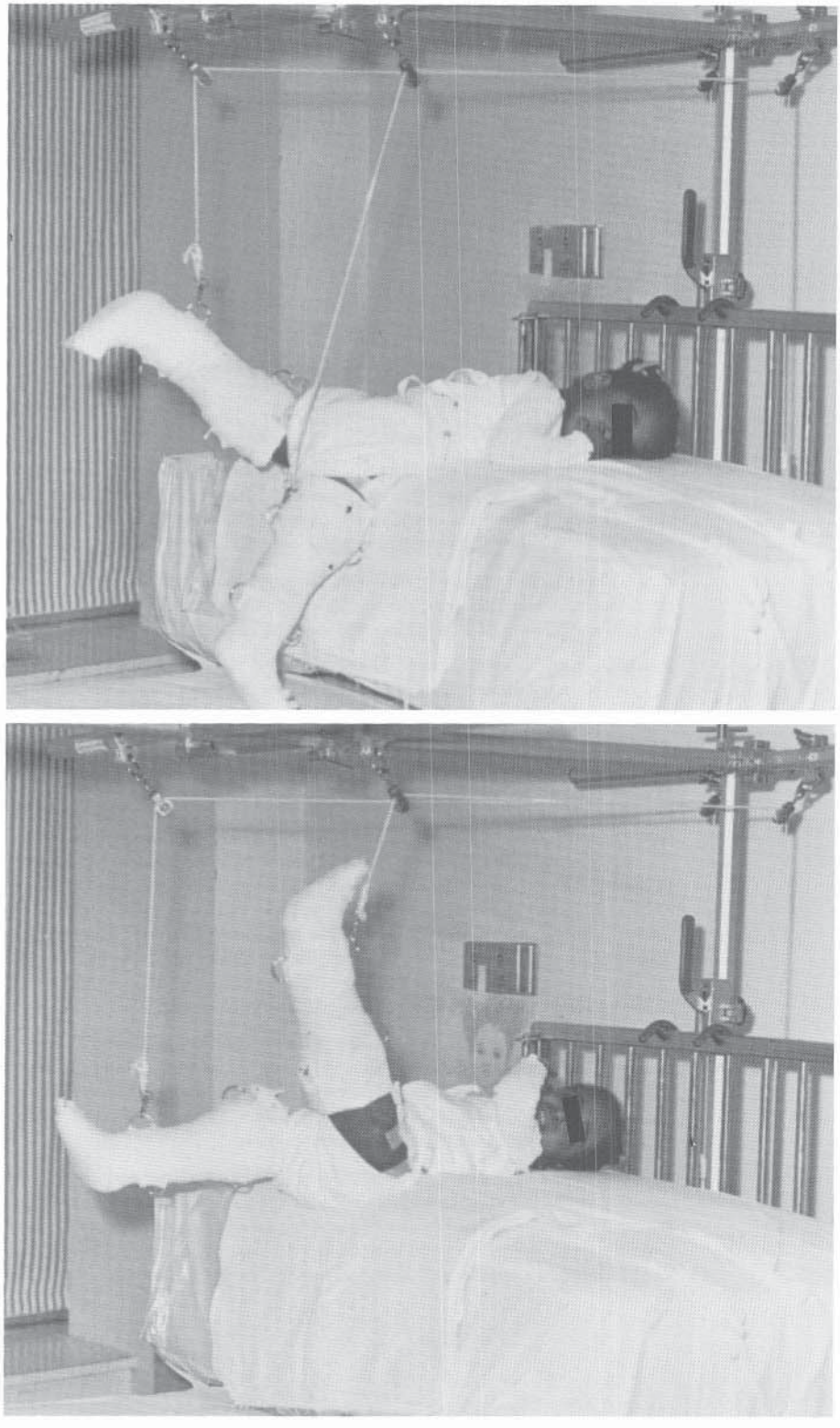


FIGURE 26–4 Method of suspension traction following an Ober-Yount release of the iliotibial band.

operation does not significantly improve the extensor or abductor power of the hip but appears to produce a more dynamic fasciodesis. Stance and gait are improved by relief of hip flexion contracture, stabilization of the hip, and relief of lumbar lordosis.[293]

The operative technique, as advocated by Barr in 1964, is as follows.

Operative Technique [Barr]. The patient, under general anesthesia, is placed in the lateral position with both limbs flexed 90 degrees at the hip and knee; the affected limb is uppermost, abducted, and resting on pillows. The skin of the lumbar region, buttock, and limb is prepared from the ribs to the midcalf. The operative field is draped so that the limb can be moved freely. The incision in the thigh begins

Text continued on page 1348

Iliopsoas Muscle Transfer for Paralysis of the Hip Abductors

OPERATIVE TECHNIQUE

The patient is positioned supine with a small sandbag under the sacrum and a larger sandbag under the ipsilateral scapula. The entire involved lower limb, the hip, the lower abdomen and chest, and the iliac and sacral regions are prepared sterilely and draped so that the limb that is to be operated on can be freely manipulated and the incision extended to the posterior third of the iliac crest without contamination.

A, The skin incision extends forward from the junction of the posterior and middle thirds of the iliac crest to the anterior superior iliac spine; it is then carried distally into the thigh along the medial border of the sartorius muscle for a distance of 10 to 12 cm, ending 2 cm distal to the lesser trochanter.

B, The deep fascia is incised over the iliac crest and the fascia lata is opened in line with the skin incision.

The lateral femoral cutaneous nerve is identified; it usually crosses the sartorius muscle 2.5 cm distal to the anterior superior iliac spine and lies in close proximity to the lateral border of the sartorius. The nerve is mobilized by sharp dissection and protected by retracting it medially with a moist hernia tape. The wound flaps are undermined and retracted. The anterior medial margin of the tensor fasciae latae muscle is identified and, by blunt dissection, the groove between the sartorius and rectus femoris muscles medially and the tensor fasciae latae muscle laterally is opened. The dissection is carried deep through the loose areolar tissue that separates these structures, and the adipose tissue that covers the front of the capsule of the hip joint is exposed. The ascending branch of the lateral femoral circumflex aertery and the accompanying vein cross the midportion of the wound; they are isolated, clamped, cut, and ligated.

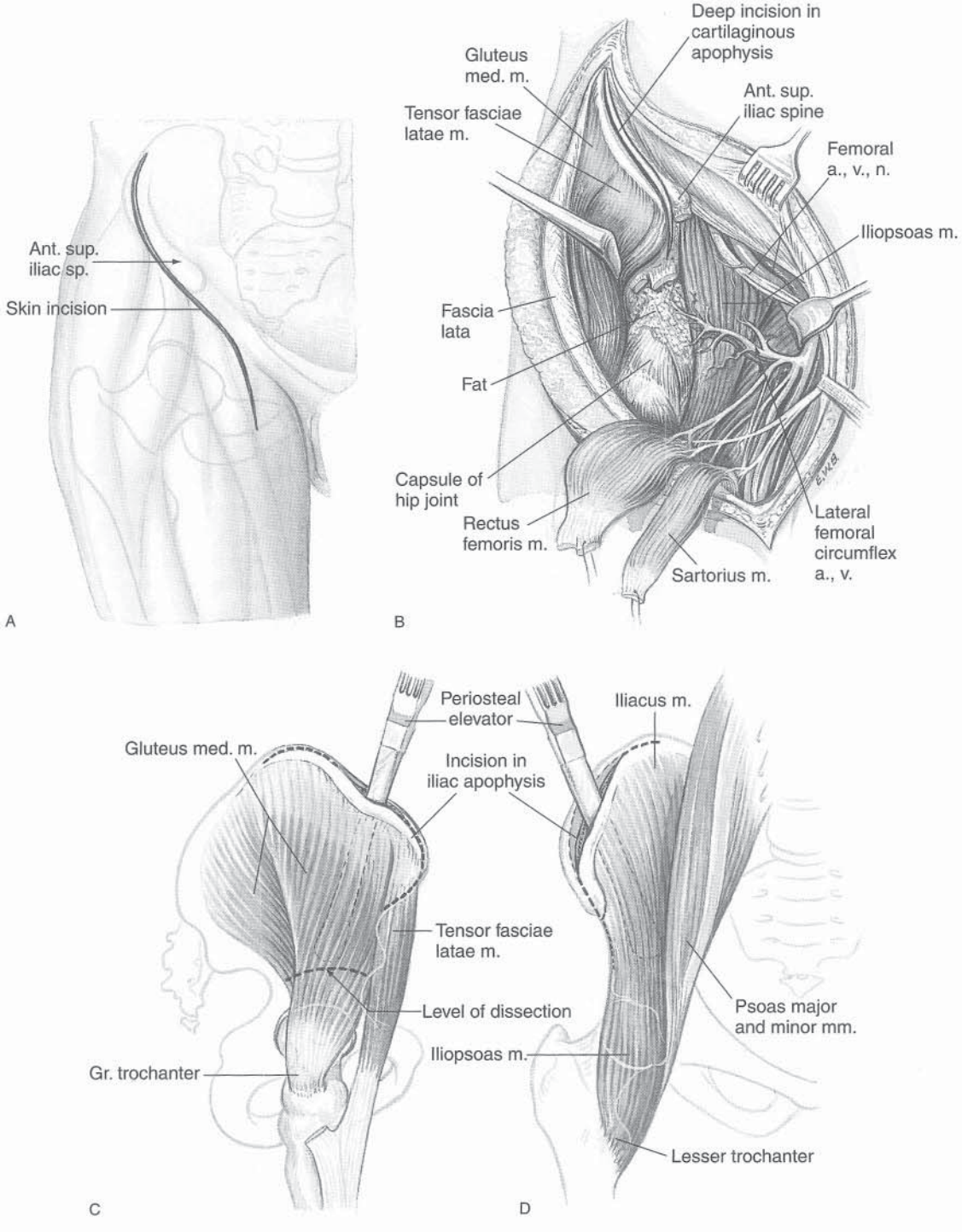
The origin of the sartorius muscle from the anterior superior iliac spine is detached and the muscle is reflected distally and medially. The free end is marked with a silk whip suture for later reattachment. The origins of the two heads of the rectus femoris are divided and reflected distally. The femoral nerve and its branches to the sartorius and rectus femoris are identified. A moist hernia tape is passed around the femoral nerve for gentle handling. The femoral vessels and nerve are retracted medially.

C, The cartilaginous apophysis of the ilium is split and the dissection is deepened along the iliac crest down to bone. With a broad periosteal elevator the tensor fasciae latae and the gluteus medius and minimus muscles are stripped subperiosteally from the lateral surface of the ilium and reflected in one continuous mass laterally and distally to the superior margin of the acetabulum. Bleeding is controlled by packing the interval between the reflected muscles and ilium with laparotomy pads.

D, Then, with a large periosteal elevator, the iliacus muscle is subperiosteally elevated and reflected medially, exposing the inner wall of the wing of the ilium from the greater sciatic notch to the anterior superior iliac spine.

By careful blunt dissection with a periosteal elevator, the iliacus muscle is freed, elevated, and mobilized from the inner wall of the ilium and the anterior capsule of the hip joint. It is important to stay lateral and deep to the iliacus muscle and work in a proximal to distal direction.

PLATE 26–2. Iliopsoas Muscle Transfer for Paralysis of the Hip Abductors



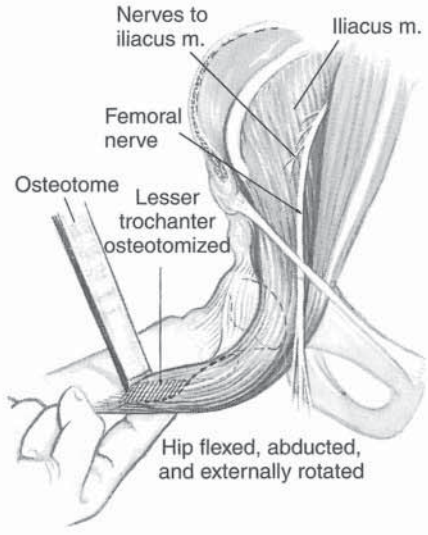
Iliopsoas Muscle Transfer for Paralysis of the Hip Abductors
Continued

E to **G,** Next, the hip is flexed, abducted, and laterally rotated, and with the index finger the lesser trochanter is cleared of soft tissues proximally, posteriorly, and distally. The index finger is then placed on the posteromedial aspect of the lesser trochanter and is used to direct a curved osteotome to the superior and deep aspect of the base of the lesser trochanter.

The lesser trochanter is osteotomized and the distal insertion of the iliacus muscle on the linea aspera of the femur is freed with a periosteal elevator.

H, The iliacus and psoas muscles are reflected proximally by sharp and dull dissection. It is very essential not to injure the nerve to the iliacus, which at times enters the muscle belly quite distally; also, the femoral nerve should not be damaged. The author finds the use of a nerve stimulator of great help. Circumflex vessels are clamped, cut, and ligated as necessary.

PLATE 26–2. Iliopsoas Muscle Transfer for Paralysis of the Hip Abductors



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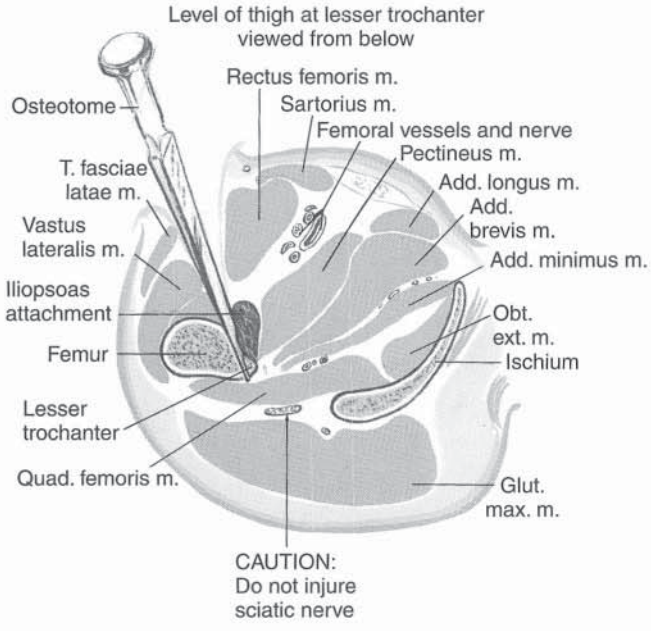

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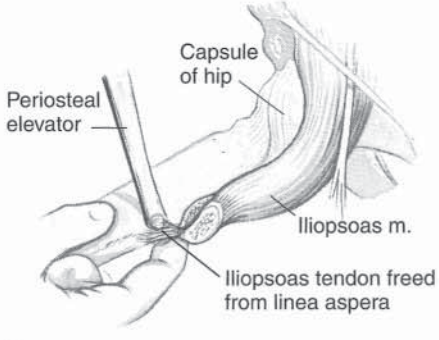

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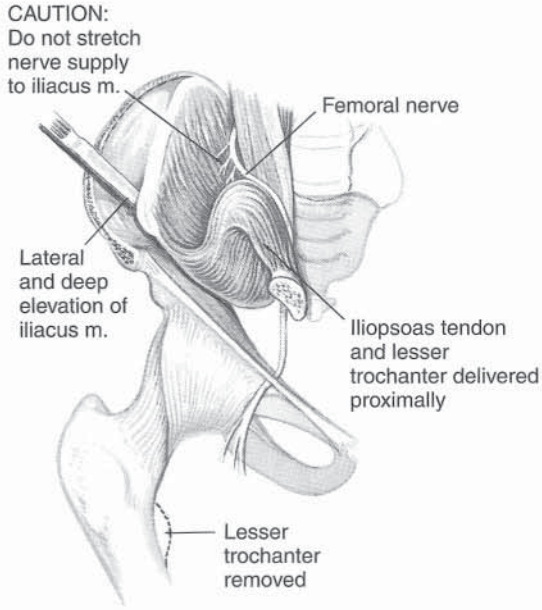


H

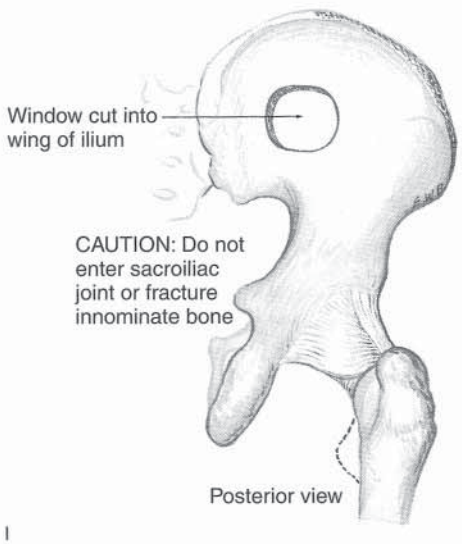
Iliopsoas Muscle Transfer for Paralysis of the Hip Abductors
Continued

I, In the middle third of the wing of the ilium a rectangular hole, usually 1½ to 2 inches, is cut with drill holes and osteotomes. The hole should be large enough to accommodate the transferred muscle. It should be located as far posteriorly as possible to allow a more direct line of muscle action. The limiting factor is the nerve supply to the iliacus, which should not be stretched.

J, With the hip in extension and medial rotation, the greater trochanter is exposed by a longitudinal lateral incision. The vastus lateralis muscle is split and the lateral surface of the proximal 4 to 5 cm of femoral shaft is subperiosteally exposed.

K, It is important not to damage the greater trochanteric apophyseal growth plate.

PLATE 26–2. Iliopsoas Muscle Transfer for Paralysis of the Hip Abductors



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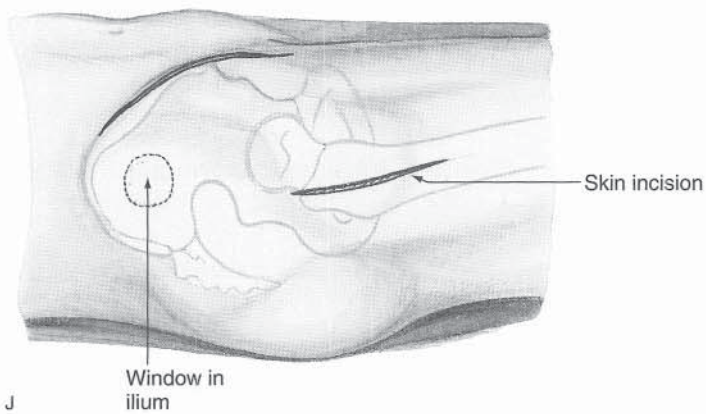

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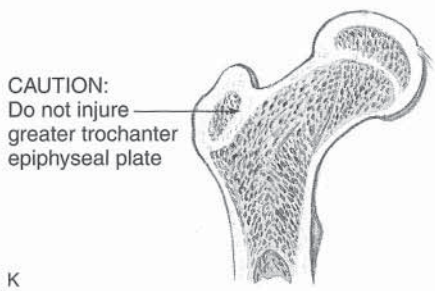


K

Iliopsoas Muscle Transfer for Paralysis of the Hip Abductors
Continued

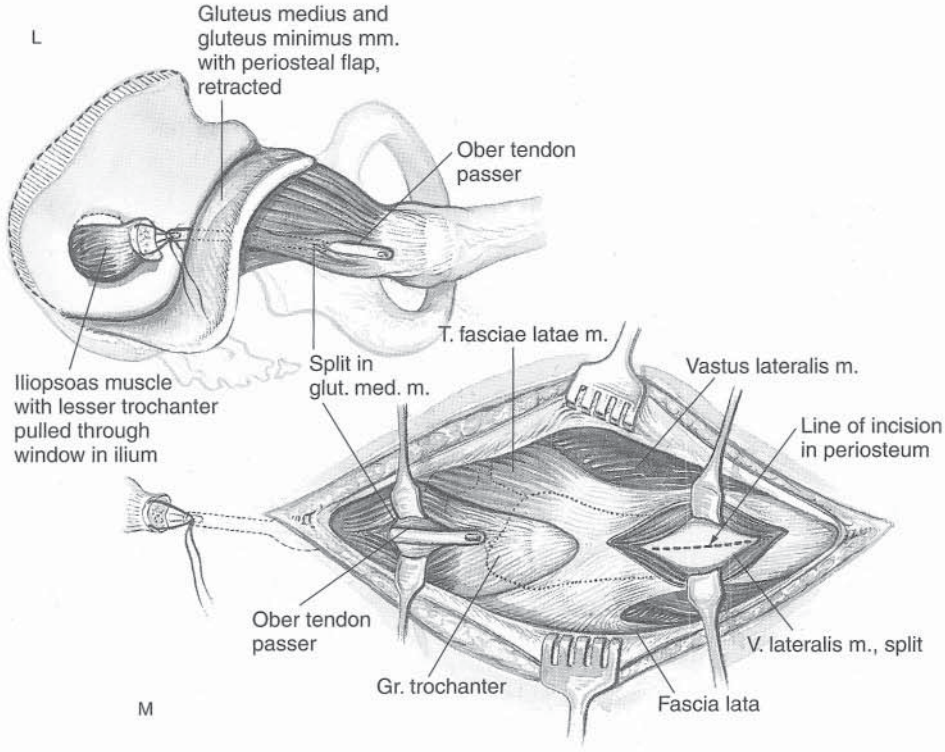
L, Next, a large Ober tendon passer is inserted through the hole in the wing of the ilium, directed deep to the glutei, and brought out in the greater trochanteric region by splitting the insertion of the fibers of the gluteus medius muscle.

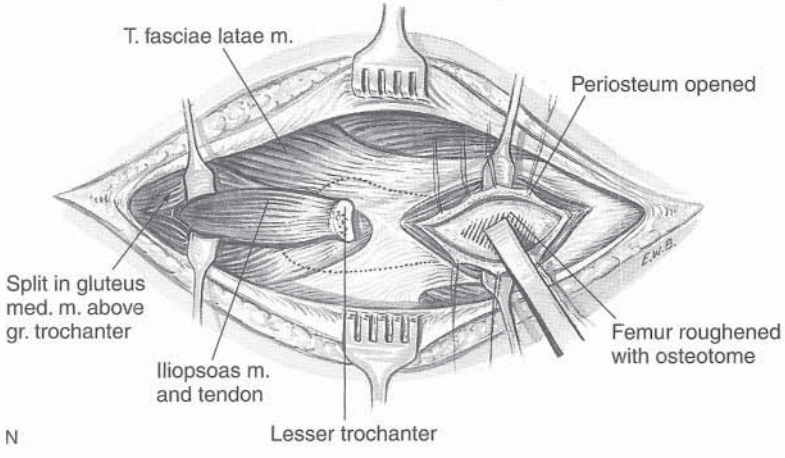
M and **N,** The iliopsoas muscle is then transferred laterally by this route with the Ober tendon passer. The nerve supply to the iliacus is again checked to be sure it is not under great tension. Next, the hip is abducted at least 45 to 60 degrees and internally rotated 10 to 15 degrees. The site of insertion of the iliopsoas tendon on the femoral shaft is determined and is roughened with curved osteotomes. The muscle should be under proper tension.

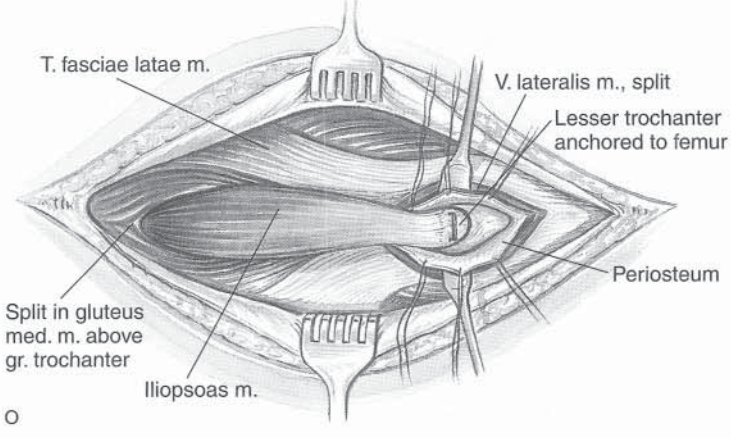
O, The lesser trochanter is anchored to the proximal femur by one or two transversely placed small staples. Mustard recommends making a trap door in the femur into which the lesser trochanter is drawn and anchored by heavy wire sutures.

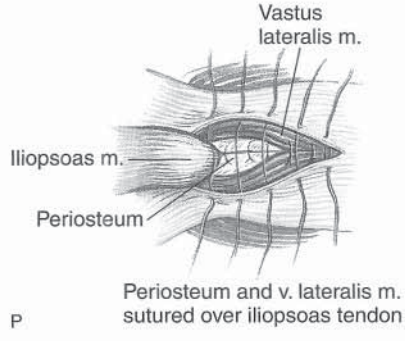
P, The periosteum and vastus lateralis muscle are sutured to the edges and over the iliopsoas tendon.

PLATE 26–2. Iliopsoas Muscle Transfer for Paralysis of the Hip Abductors









Iliopsoas Muscle Transfer for Paralysis of the Hip Abductors
Continued

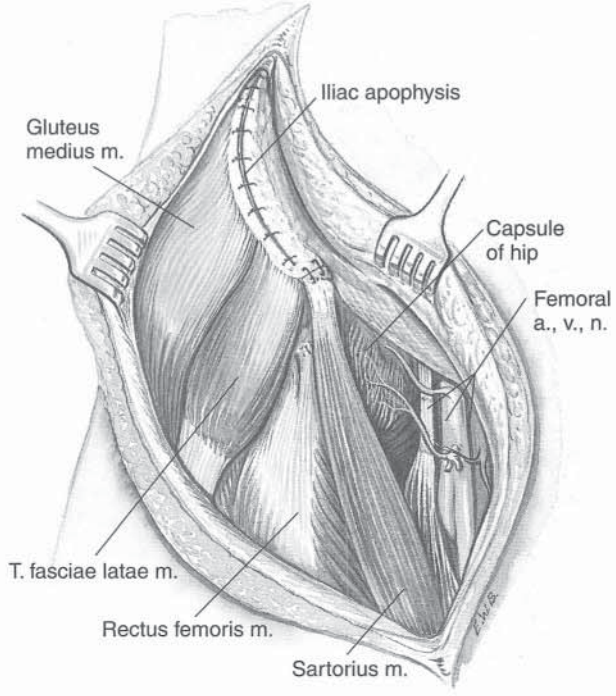
Q and **R,** The rectus femoris and sartorius muscles are sutured to the inferior and superior iliac spines, respectively. The tensor fasciae latae, the gluteus medius and minimus, and the abdominal muscles are sutured to the iliac crest. The wound is closed in layers in routine manner. A one-and-one-half-hip spica cast is applied, with the hip in 60 degrees of abduction, 10 to 15 degrees of medial rotation, and slight flexion.

POSTOPERATIVE CARE

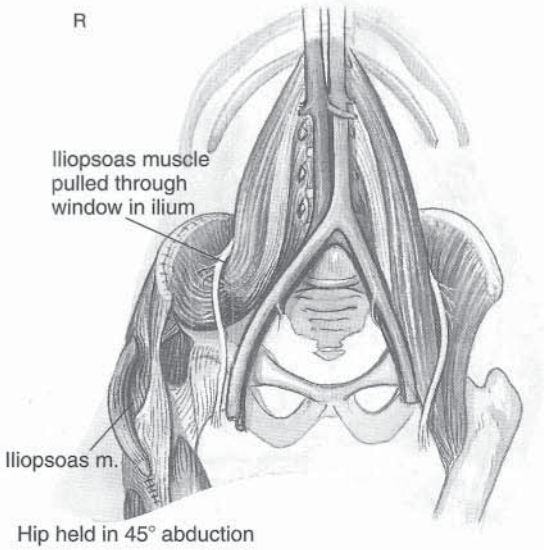
Four to 6 weeks after surgery, the patient is readmitted to the hospital and the cast is removed and a new bivalved hip spica cast made. This should be cut low on the lateral side so that hip abduction exercises can be performed in the posterior half of the cast. Radiographs of the hips are obtained to determine the stability of the hip joint. Great care should be exercised so that a pathologic fracture of the femur is not caused when the child is lifted out of the cast.

Training of the iliopsoas transfer follows the same general principles as training of tendon transfers in poliomyelitis. In myelomeningocele, however, there is extensive paralysis of the lower limb, necessitating orthotic support, and the patient is much younger. Thus, as soon as the transferred iliopsoas has fair motor strength and the lower limbs can be adducted to neutral position, weightbearing is permitted in bilateral above-knee orthoses. The butterfly pelvic band will keep the hips in 5 to 10 degrees of abduction during locomotion. At night, the hips and the transfer are protected in the bivalved hip spica cast or in a plastic hip-knee-ankle-foot orthosis (HKAFO).

PLATE 26–2. Iliopsoas Muscle Transfer for Paralysis of the Hip Abductors



Q Reattachment of muscles



Hip held in 45° abduction

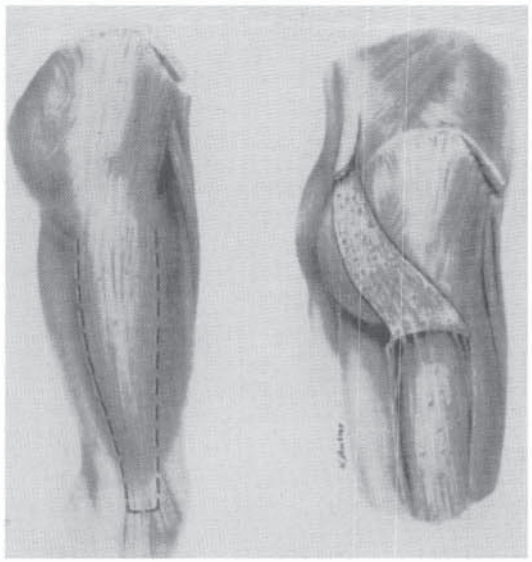


FIGURE 26–5 Erector spinae transfer or fascia lata transfer to the greater trochanter. (From Hogshead HP, Ponseti IV: Fascia lata transfer to the erector spinae. J Bone Joint Surg 1964;46-A:1390.)

just anterior to the head of the fibula and ends proximally just distal to the anterior superior iliac spine passing over the greater trochanter. The iliotibial band is exposed through its full length and breadth and is divided transversely at the level of the distal pole of the patella. A stout silk suture is passed through its free end and as wide a strip of fascia as it is possible to obtain is dissected upward and preserved as the tendon of insertion of the tensor fasciae latae muscle. Beginning at the trochanteric level, the dissection is carried toward the anterior iliac spine, mobilizing the distal half of the tensor fasciae latae muscle and preserving its neurovascular bundle. The intermuscular septa and other contracted fascial structures at the knee and anterior to the hip are divided as necessary while an assistant holds the hip and knee in as much extension as possible. The sartorius and rectus muscles are tenotomized if they are contracted and totally paralyzed. The iliopsoas tendon, if need be, may be divided at its insertion but should be transposed to a more proximal and anterior position in the intertrochanteric region. The anterior capsule of the hip may also be divided through the same incision if it prevents extension of the hip. The neurovascular bundle is preserved.

Subperiosteal anchorage of the fascial strip to the femur is accomplished by making two parallel longitudinal incisions, usually 5 to 6 cm long, through the origin of the vastus lateralis and the periosteum, one on the anterolateral, the other on the posterolateral aspect of the femur just below the greater trochanter and tunneling beneath the periosteum to join the two incisions. The strip of fascia is then passed through the tunnel and secured to the periosteum by silk sutures. This must be done with the hip held in as much extension as possible, without putting undue force on the tissues, and maintaining slight abduction and neutral position as regards rotation.

The lumbar incision is about 15 cm long. It is made parallel to and 5 to 8 cm lateral to the line of the spinous processes of the fourth and fifth lumbar and first sacral vertebrae. The inferior end of the incision is located medial to and about 5 cm distal to the posterior superior iliac spine. The incision is deepened through the lumbodorsal fascia, which is reflected to expose the underlying erector spinae muscle. By blunt dissection along a vertical line, the lateral two-thirds of this muscle mass is mobilized and freed from the medial one-third, which is left attached to the adjacent spinous processes and laminae. The mobilized muscle is freed by sharp dissection from its origin to the ilium and sacrum. Since the nerve and blood supply to this muscle is segmental and enters from its ventral surface, it may be necessary to sacrifice one or two of the most distal neurovascular bundles in order to mobilize a 10-cm length of muscle mass.

By means of a long tendon carrier, the free end of the fascia lata is passed within the gluteal muscle compartment entering the lumbar incision just medial to the posterior superior iliac spine. The tunnel at its point of emergence is carefully dilated by the surgeon's finger so that the fascia can glide freely. The gliding deep surface of the fascia should be placed as it lies ventrally. With the hip held in extension, the fascia is attached, under moderate tension, to the free end of the mobilized erector spinae muscle. This is best done by laying the ventral surface of the muscle on the subcutaneous surface of the fascial strip, passing the suture in the end of the fascia through the muscle, as far proximally as possible, and then fixing the edges of the fascia to the edges of the muscle flap by a series of interrupted sutures. The distal end of the muscle is thus covered on its deep surface by the fascia lata. The lumbar incision is closed in layers; it is usually possible to close the lumbodorsal fascia over the transplant partially. The thigh incision is closed in a routine manner. No attempt should be made to close the defect in the fascia of the thigh. After application of sterile dressings, the extremity is immobilized by elastic bandages and long plaster splints which extend from the ribs to the toes. The hip is immobilized in as much extension as can be obtained comfortably. No attempt is made to correct the hip-flexion contracture completely at this time.

Technique for Correction of Remaining Contractures in Poliomyelitic Deformities. After 10 days to 2 weeks, when the incisions have healed, the remaining contractures are gradually stretched out. The lumbar spine and the opposite lower extremity are immobilized in a spica with that hip in sufficient flexion to obliterate the lumbar lordosis. A separate toe-to-groin cast is applied to the affected limb with the knee preferably in almost complete extension. With the patient supine the affected limb in its plaster cast is suspended from an overhead frame. The contracture can then be stretched gradually and completely by lowering the limb in day-to-day increments until the hip comes into hyperextension. During this procedure, the circulation and sensation in the toes should be watched carefully, especially if excessive shortening of the femoral vessels and nerves was observed at operation.

If a knee-flexion deformity is present it may be corrected simultaneously by wedging the cast.

As a rule the deformity is satisfactorily corrected in two to three weeks. The apparatus is then removed and assistive muscle reeducation exercises are begun with the patient in

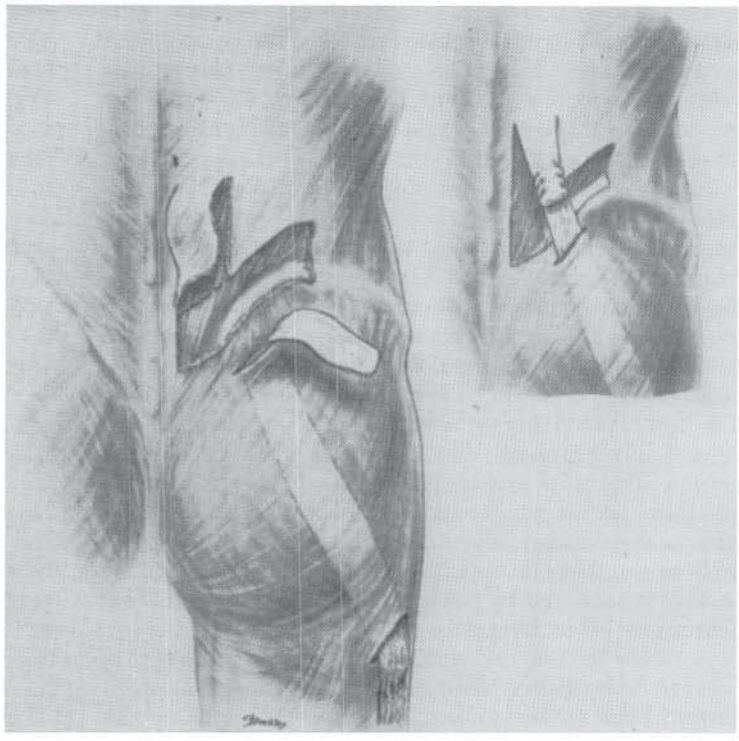


FIGURE 26–6 Fascia lata transfer to lumbodorsal fascia to provide posterior stability to the hip joint. (From Hogshead HP, Ponseti IV: Fascia lata transfer to the erector spinae. J Bone Joint Surg 1964;46-A:1404.)

recumbency. Underwater exercises are of value. A bivalved long spica to hold the hip in the corrected position should be worn at night for several months. Walking with crutches is permitted as soon as the transplant functions satisfactorily, usually about 6 weeks postoperatively. Many patients require bilateral transplants and should undergo operation in two stages, 4 to 6 weeks apart. Careful gait training is essential if the best results are to be obtained.*

Hogshead and Ponseti found the formation of an erector spinae flap in myelomeningocele to be difficult. The procedure was bloody and the ramifications of the meningocele sac were inadvertently entered, resulting in troublesome drainage of cerebrospinal fluid through the wound. Since, in their experience, erector spinae transfer did not provide active power of hip extension or abduction, they recommended attachment of the distal end of the fascia lata band to the freed lumbodorsal fascia at the level of the third or fourth lumbar vertebra (Fig. 26–6). They termed the operative procedure fascia lata transfer to the erector spinae.[157] The route of the transfer should be subfascial, and its direction from the greater trochanter to the region of the posterior superior iliac spine should be as far posterior as possible.

Caution should be exercised in the anterior release of soft tissue contracture of the hip. Every effort should be made to preserve viable muscles and their nerve and blood supply. The anterior capsule of the hip should not be sectioned, in order to prevent anterior dislocation of the femoral head. When contracture of the anterior capsule is fixed and it limits extension of the hip, it is *lengthened.*

In the Sharrard modification of the Mustard operation a hole is made in the posterior part of the ilium and the iliacus muscle is sutured to the lateral surface of the ilium (see Plate 26–2). The operation was designed to provide power of hip extension as well as hip abduction. Unfortunately, the motor nerve supply of the iliacus muscle is frequently distal in its location, limiting the degree of posterior positioning of the iliac hole. In the author's experience, Sharrard iliopsoas transfer has not been successful in providing active power of hip extension against gravity in the presence of complete paralysis of the gluteus maximus muscle. When the hamstring muscles are normal in motor strength and the gluteus maximus is only partially paralyzed, it will restore functional strength of hip extension and give substantial improvement in gait.

PARALYTIC DISLOCATION OF THE HIP

Hip dislocation in poliomyelitis is an acquired deformity caused by flaccid paralysis and the resulting muscular imbalance that develops. When, in a young child, the gluteus maximus and medius muscles are paralyzed and the hip flexors and adductors are of normal strength, eventual luxation of the hip is almost inevitable. Loss of hip abductor power causes retardation of growth from the greater trochanteric apophysis. Disparity of relative growth from the capital femoral epiphysis and the greater trochanteric apophysis causes increasing valgus deformity of the femoral neck. In severe cases the angle between the neck and shaft of the femur increases to 180 degrees. Excessive anteversion of the femoral neck may also develop. When the angle between the femoral neck and the horizontal plane of the pelvis approaches 90 degrees, the hip joint becomes mechanically unstable. Gradually, under the forces of body weight, the capsule becomes lax and the femoral head rides out of the acetabulum. The empty acetabulum retains an adequate depth for several years following paralytic dislocation. With lack of concentric pressure of the femoral head in the acetab-

*From Barr JS: Discussion. J Bone Joint Surg 1964;46-A;1402.

ulum, however, progressive shallowness and obliquity of the acetabular roof develop. Thus, factors in the pathogenesis of true paralytic dislocation are muscle imbalance, coxa valga, and laxity of the capsule. In treatment, it is important to remember that coxa valga precedes subluxation and shallowness of the acetabulum.[176,177,295]

Acquired hip dislocation does not usually occur in a totally flail lower limb, particularly if the patient has been walking with the support of an orthosis. If inadequately treated, however, the flail hip may develop abduction-flexion-external rotation contracture as a result of shortening of the iliotibial band. When the lower limbs are aligned parallel to the vertical axis of the body in the weightbearing position, the pelvis will be forced into an oblique position. The contralateral hip—the one on the high side of the pelvis—is in a markedly functional valgus position and will become dislocated eventually. Pelvic obliquity may result also from the foregoing factors; another cause may be severe structural scoliosis in the suprapelvic region. This type of scoliosis should be distinguished from the positional scoliosis that is produced by pelvic obliquity due to contractual deformities below the pelvis.

A review by Lau and associates of surgical treatment of paralytic dislocations of the hip in poliomyelitis patients demonstrated that the keys to successful reductions are restoration of muscle balance, correction of the femoral neck-shaft angle, correction of anteversion, and restoration of acetabular coverage.[7a] *The authors also emphasized the importance of posterior acetabular coverage.*

Treatment. Dislocation of the hip in poliomyelitis may be prevented by restoring dynamic balance about the hip; this is achieved by appropriate muscle transfers. If the age at onset of paralysis and muscle imbalance is less than 2 years, iliopsoas transfer to restore power of hip abduction is performed when the child is 4 or 5 years old. If the coxa valga deformity is less than 150 degrees, a preliminary varization osteotomy is unnecessary; the valgus deformity will correct itself with growth once hip abductor power is restored. If the coxa valga deformity is greater than 150 degrees, it is best to correct the deformity and obtain a femoral neck-shaft angle of 110 degrees prior to iliopsoas transfer.

If at the time of paralysis the patient is more than 2 years old, iliopsoas transfer may be postponed and the stability of the hip followed periodically with radiographs. When the coxa valga exceeds 160 degrees and the femoral head starts to subluxate laterally, varization osteotomy is performed. In patients less than 6 years old, the femoral neck-shaft angle is reduced to 105 degrees; in older patients the angle is corrected to 125 degrees. Often, if dynamic muscle imbalance persists, valgus deformity will recur with growth. The procedure should be followed in 6 months to a year with an iliopsoas transfer.

The operative technique of varization osteotomy follows the same principles as those of valgus osteotomy. First, if there is any adduction contracture of the hip, it should be passively stretched and corrected by split Russell traction, gradually bringing the hips into wide abduction. Adductor myotomy of the hip should be avoided whenever possible. The anterolateral surface of the subtrochanteric region of the femur is subperiosteally exposed, as described in Plate 26–3. The line of osteotomy is shaped like a modified dome with a lateral buttress of cortical bone in the proximal segment to lock the upper end of the distal segment while the femoral shaft is adducted. This procedure is the reverse of valgus osteotomy. Rotational malalignment can be corrected at the same time. The author uses Crow pins or threaded Steinmann pins and Roger Anderson apparatus to fix the fragments together. Others may use a bone plate with four screws, a blade plate, or two staples. It is a matter of personal preference and depends on past experience. Blundell Jones exposes the trochanteric region of the proximal femur posterolaterally with the patient in prone position and corrects the valgus deformity by excising a wedge of bone with its base medially.[176,177]

When the hip is completely dislocated, the hip joint capsule is stretched out and lax. Paralytic hip dislocation is very easily reduced. In the beginning the femoral head can be relocated into the acetabulum by simple abduction of the hip. Later on, however, soft tissue contracture may develop, and an initial period of skin or skeletal traction will then be indicated. Prolonged immobilization of the hip following reduction in a spica cast is not recommended. Once the cast is removed, the dislocation will recur. The use of a solid hip spica cast does not correct the etiologic factors, and it has the additional disadvantage of causing disuse atrophy of muscles and bone. To stimulate normal growth of the proximal femur, weightbearing should be restored as soon as possible.

Reefing and repair of the capsule is essential. It is described and illustrated in Plate 15–3. An iliopsoas transfer is performed at the same time to restore power of hip abduction and muscle balance about the hip. If the acetabulum is shallow and maldirected, the procedure may be combined with a Salter innominate osteotomy.

Arthrodesis of the Hip. Fusion of the hip in poliomyelitis may increase the ability to walk and eliminate the need for orthotic support. The procedure does have serious disadvantages, however, which should be carefully considered. Sharp and colleagues reported a series of 16 hip fusions performed in children for paralysis caused by poliomyelitis.[289] There was a high percentage of fractures (eight of the femur and one of the tibia). In addition, there were three cases of pseudarthrosis and one of slipped capital femoral epiphysis. In three patients the hip was fused and subsequently required correction by femoral osteotomy. One patient had marked limitation of knee motion following prolonged immobilization in the cast; in another, amputation was indicated because of excessive shortening of the limb.

A stiff hip burdens the spine and knee with abnormal stress and strain. Thus, ligamentous instability of the knee, progressive lumbosacral scoliosis, and trunk instability due to extensive paralysis of abdominal muscles are absolute contraindications to hip fusion in poliomyelitis. A functional quadriceps femoris is desirable but not absolutely necessary, provided there is no flexion deformity of the knee, and stability of the foot and ankle is provided by a strong triceps surae muscle or by pantalar arthrodesis in a 15-degree equinus position. Stability of the flail knee is achieved as the body weight falls on the ball of the foot, forcing the heel onto the ground and driving the knee into hyperextension (Fig. 26–7).

Hallock in 1942, 1950, and 1958 reported an enlarging

series of hip fusions performed in patients with flail lower limbs resulting from poliomyelitis.[135–137] At first the procedure was employed only in those instances in which there was painful arthritic subluxation or dislocation of the hip, or when previous reconstructive operations such as open reduction, shelf stabilization, or muscle transfers failed. Later, Hallock extended his indications to include several individuals with severe hip lurch from extensive hip muscle paralysis without dislocation. He reported gratifying results: The arthrodesis relieved pain, achieved stability, and decreased the limp. Hallock recommended that the optimum position of fusion be 35 degrees of flexion, neutral rotation, and neutral abduction-adduction position, except in females or when considerable shortening is present, in which cases 10 or 15 degrees of abduction is advised for biologic reasons and to compensate in some measure for the inequality of leg length.[135]

When there is marked shortening of the flail limb, making equalization impractical, hip fusion should not be performed. The age of the patient is another consideration; it is imperative that the patient be mature enough to understand the disadvantages of a stiff hip. Hip fusion in a paralytic flail lower limb is controversial and should be considered only after thorough and meticulous assessment of the patient.

The Knee

QUADRICEPS FEMORIS PARALYSIS

The quadriceps is commonly affected by poliomyelitis. When there is slight genu recurvatum with adequate strength of the triceps surae and hamstring muscles, the knee is stabilized by locking it in hyperextension (Fig. 26–8). Patients so treated are able to walk quite satisfactorily. During the stance phase of gait, quadriceps weakness is compensated for by tilting the trunk and center of gravity of the body forward. The only functional disabilities are difficulty in climbing steps and running. In the presence of knee flexion deformity, however, the knee joint becomes unstable because it cannot be locked in hyperextension.

When the hamstring muscles are normal, they can be transferred anteriorly to the patella and the ligamentum patellae to provide extension and stability of the knee. This procedure is advised when instability of the knee interferes with ordinary walking or when with such a transfer the patient will be able to dispense with an orthosis. Each case, however, must be considered individually. When the hip flexors are less than fair in motor strength, anterior transfer of hamstrings is absolutely contraindicated. After surgery the patient will be unable to clear the limb from the floor, and consequently the disability will be greater. The triceps surae muscle must be at least fair in strength; if not, with loss of all dynamic posterior knee support, marked genu recurvatum will develop. It is preferable to have adequate strength of the gluteus maximus and hip abductor muscles. Prior to tendon transfer, any flexion contracture of the knee and equinus deformity of the ankle should be fully corrected by wedging casts. The mechanics of patellofemoral articulation should be normal. Any significant malalignment of the lower limb, such as marked genu valgum, should also be corrected preoperatively.

A number of muscles have been transferred to restore knee extension power, namely, the biceps femoris, semitendinosus, sartorius, tensor fasciae latae, and adductor longus.*

Transfer of both the biceps femoris and the semitendinosus muscle is the procedure of choice. The strength of the tensor fasciae latae and sartorius muscles is not sufficient to substitute for the quadriceps. In an electromyographic study of 21 patients with paralysis of the lower limb due to poliomyelitis, in whom 39 muscle transfers for quadriceps paralysis were performed, Sutherland and associates reported the following results: 10 to 14 hamstring transfers achieved conversion from swing phase to stance phase activity (roughly comparable to that of the normal quadriceps femoris); 2 of 11 sartorius transfers and 4 of 12 tensor fasciae latae transfers achieved stance phase activity.[314]

The operative technique of transfer of the biceps femoris and semitendinosus muscles, as described by Crego and Fischer[73] and Schwartzmann and Crego,[285] is as follows (Fig. 26–9). The patient is placed supine with a large sandbag under the ipsilateral hip so that the patient is tilted 45 degrees to the opposite side and the knee to be operated on is in semiflexion. A longitudinal incision is made over the posterolateral aspect of the thigh, starting immediately above the head of the fibula and extending proximally to terminate at the junction of the proximal and middle thirds of the thigh. The subcutaneous tissue and deep fascia are incised in line with the skin incision. The common peroneal nerve, located posteromedial to the biceps tendon, is identified and gently retracted posteriorly with a moist umbilical tape. The biceps femoris tendon is dissected free of its surrounding soft tissues and is retracted anterolaterally. At its point of attachment the lateral collateral ligament to the fibular head is quite adherent to the biceps tendon; great caution must be exercised to protect it and not divide it. Next, the biceps tendon is detached from its insertion to the head of the fibula. Using sharp and blunt dissection, the surgeon frees the muscle bodies of the short and long heads of the biceps muscle proximally as high as possible, taking care to preserve their nerve and blood supply. The new direction of the line of pull of the transfer must be as nearly vertical as possible; if they run horizontally, the muscles will pull the patella in a posterior direction.

Next, a transverse incision is made over the anterior aspect of the knee, centered over the distal third of the patella. The subcutaneous tissue and deep fascia are divided. The wound flaps are undermined to expose the patella and patellar tendon. With a large Ober tendon transfer, a wide subcutaneous tunnel is made, extending from the patella incision to the one on the lateral thigh. A 10- to 15-cm-long segment of the intermuscular septum and the iliotibial band is excised to allow free gliding of the transferred muscle belly.

Next, the sandbag is removed and placed under the opposite hip so that the patient is positioned semilaterally, being turned to the ipsilateral side. A longitudinal incision is made over the posteromedial aspect of the thigh, beginning 3 cm proximal to the popliteal crease and extending to the junction of the middle and proximal thirds of the thigh. The

Text continued on page 1356

*See references 36, 44, 73, 148, 182, 184, 252, 264, 285, 320.

Lloyd Roberts Technique of Intertrochanteric Oblique Osteotomy of Proximal Femur and Internal Fixation with Coventry Apparatus (Lag Screw and Plate)

The patient is placed supine on a radiolucent operating table. The operation is performed under image intensification radiographic control. The iliac region, hip, and entire lower limb are prepared sterilely and draped so that the limb can be manipulated freely.

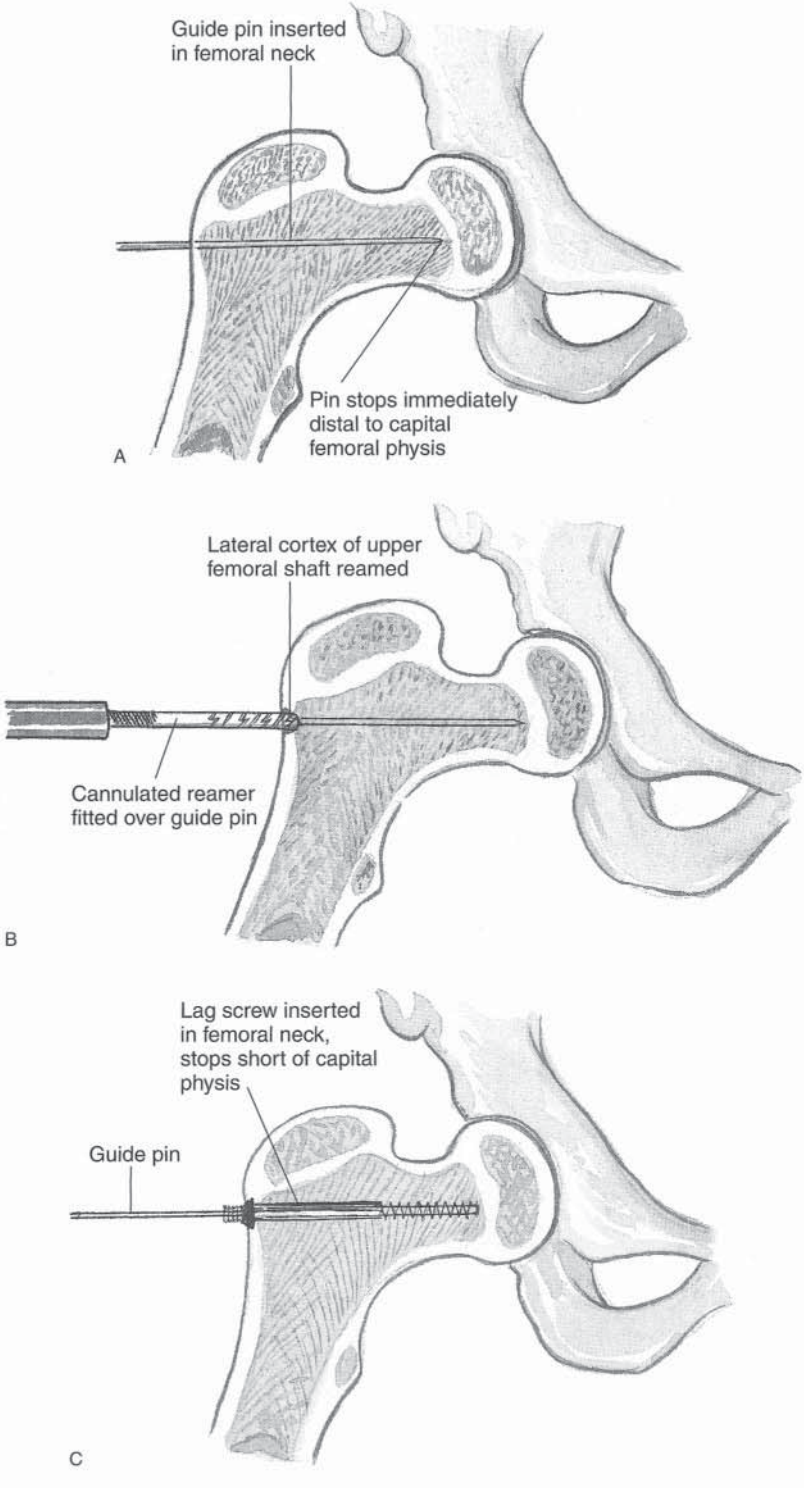
OPERATIVE TECHNIQUE

A, The incision begins 1 cm posterior and inferior to the anterior superior iliac spine, curves across to the top of the greater trochanter, and continues distally along the femoral shaft for a distance of 6 to 8 cm. The subcutaneous tissue is divided in line with the skin incision. The deep fascia is incised and the interval between the tensor fasciae latae anteriorly and the gluteus medius posteriorly is developed by blunt dissection. The vastus lateralis is divided longitudinally by an L-shaped or U-shaped incision, and the part of it that originates from the anterior aspect of the intertrochanteric area is detached. With a periosteal elevator, the intertrochanteric region and the upper femoral shaft are exposed. At this time the calcare femorale is visualized, and the femoral head can be palpated within the capsule. A sturdy stainless steel pin of appropriate diameter, usually 0.062 inches, is chosen; the operator must be sure its diameter fits the hole in the lag screw. With the hip in full medial rotation, a 3-mm hole is drilled through the center of the lateral cortex of the upper femoral shaft, 0.75 to 1.0 cm below the growth plate of the greater trochanter. To avoid injury to the growth plate of the apophysis, the surgeon should verify its site with image intensification radiography. Next, the guide pin is inserted into the femoral neck parallel to the floor in a proximally inclined oblique plane parallel to the long axis of the femoral neck. The tip of the pin should stop immediately distal to the capital femoral physis. Proper placement of the guide pin is crucial and is confirmed with AP and lateral image-intensified radiographs.

B, A cannulated reamer (with a stop to prevent more than $\frac{1}{2}$-inch penetration) is fitted over the guide pin. The lateral cortex of the upper femoral shaft is reamed to permit firm fixation of the lag screw in the cancellous bone.

C, Next, with the special lag screw inserter, a lag screw of appropriate length is inserted into the femoral neck. It should stop short of the capital physis. To avoid injury to the growth place, the surgeon should confirm the position of the screw with AP and lateral radiographs.

PLATE 26–3. Lloyd Roberts Technique of Intertrochanteric Oblique Osteotomy of Proximal Femur and Internal Fixation with Coventry Apparatus (Lag Screw and Plate)



Lloyd Roberts Technique of Intertrochanteric Oblique Osteotomy of Proximal Femur and Internal Fixation with Coventry Apparatus (Lag Screw and Plate) *Continued*

D, With an oscillating saw, the femoral osteotomy is performed at the intertrochanteric level parallel to the calcare; use the guide pin, which protrudes from the lag screw, to guide the direction of osteotomy and verify it with image intensification radiography. (Drill holes may be used to mark the line of osteotomy.) Once the osteotomy is completed, gently strip the adjacent periosteum to mobilize the bone fragments and permit free rotation of the femoral shaft.

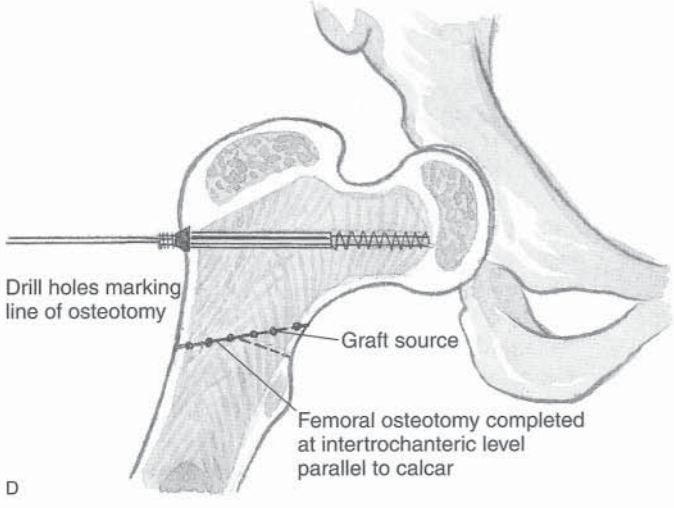
E, The side plate is bent to the appropriate angle. The guide pin is removed and the top hole of the side plate is engaged to the protruding end of the lag screw. A cannulated level with a handle is attached to the lag screw for firm control of the upper fragment. The distal fragment is adducted and rotated laterally to the desired degree. The oblique line of the osteotomy will often make a triangle of bone at the upper end of the femoral shaft that will protrude anteriorly; this is excised and used as a local bone graft. The osteotomized fragments are apposed and secured by attaching the side plate to the femoral shaft with screws and a nut at the top of the lag screw and the proximal fragment. Final radiographs are made to double check security of the fixation device. A one-and-one-half-hip spica plaster of Paris cast is applied.

POSTOPERATIVE CARE

The child is usually sent home 3 to 4 days postoperatively and readmitted to the hospital 6 weeks later. The plaster cast is removed, and the hip and knee are mobilized. When able to ambulate with crutches (three-point partial weightbearing on the affected limb), the patient is discharged, usually within 2 to 4 days.

The plate and screws are removed 6 months postoperatively.

PLATE 26–3. Lloyd Roberts Technique of Intertrochanteric Oblique Osteotomy of Proximal Femur and Internal Fixation with Coventry Apparatus (Lag Screw and Plate)



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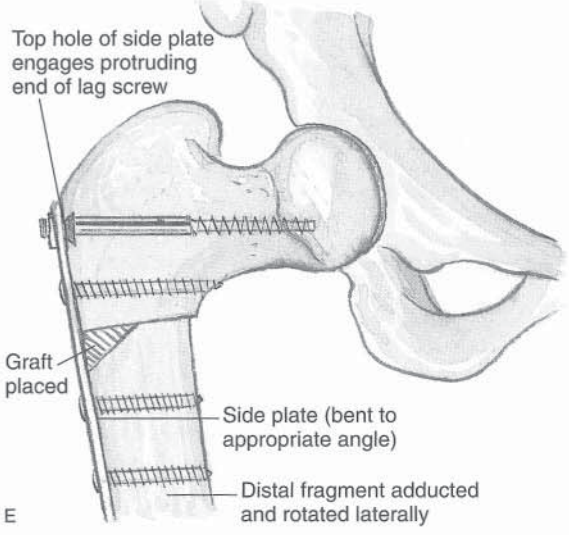

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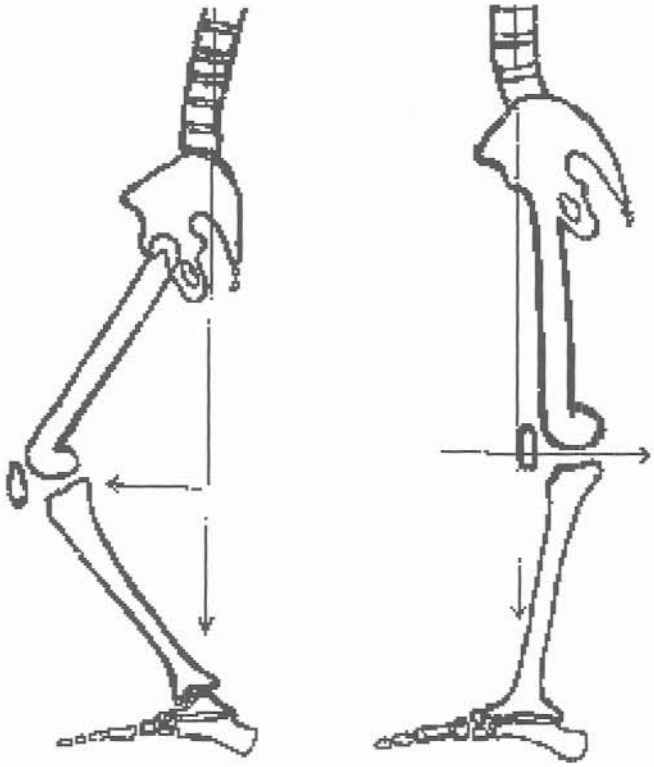


FIGURE 26–7 The principle of dynamic knee stabilization when the hip is fused and the ankle is fixed in slightly equinus position. On the left is shown the collapsible knee, when the hip and ankle are flail; on the right, stability of the knee is achieved when the body weight falls on the ball of the foot, forces the heel to the ground, and locks the knee, driving it into hyperextension. (From Sharp N, Guhl JF, Sorenson RI, et al: Hip fusion in poliomyelitis children. J Bone Joint Surg 1964;46-A:122.)

subcutaneous tissue and deep fascia are divided. The semitendinosus tendon is isolated and, through a separate small incision over the anteromedial aspect of the proximal leg, it is detached from its insertion on the tibia. It is easy to identify the semitendinosus tendon in the distal leg wound by pulling on it in the proximal thigh wound; anatomically, at its insertion, the semitendinosus tendon is located posterior to the sartorius tendon and inferior to the tendon of the gracilis. Next, the semitendinosus tendon is delivered into the proximal wound and dissected free to the middle third of the thighs. Through a wide subcutaneous tunnel from the anterior transverse knee incision to the posteromedial thigh incision, the semitendinosus tendon is rerouted and delivered into the prepatellar area. Again, the deep fascia is widely incised to avoid angulation and to permit free gliding of the semitendinosus tendon.

Next, the prepatellar bursa is reflected and retracted to one side and an I-shaped incision is made through the quadriceps tendon and periosteum over the anterior surface of the patella. These tissues are stripped and reflected medially and laterally. With a $\frac{9}{64}$-inch drill two oblique longitudinal tunnels are made through the patella, starting at the superolateral and superomedial poles of the patella and emerging on each side of the patellar tendon. The tunnels are enlarged with progressively increasing sizes of hand drills and curets. The operator must be careful not to damage the articular surface of the patella.

With braided silk whip sutures on their ends, the biceps femoris tendon and the semitendinosus tendon are each pulled through their respective tunnels in the patella and sutured to the patellar tendon under tension. Additional interrupted sutures are placed proximally and distally, fixing the biceps and semitendinosus tendons to the rectus femoris and patellar tendons. The soft tissues are sutured over the anterior aspect of the patella and the wounds are closed. A long-leg cast is applied that holds the knee in neutral position but not in hyperextension.

Meticulous postoperative care is very important to obtain a satisfactory result. Tension on the transferred hamstring is prevented by avoiding flexion of the hip. The patient is kept supine in bed for 3 weeks, and it should be strongly emphasized to personnel that the patient is not to sit.

Functional training of the transfer is begun 3 to 5 days after surgery, or as soon as the patient is comfortable. The patient is placed on the side to eliminate the force of gravity. The knee and hip are slightly flexed, and the patient is asked to extend the hip and knee. Active contraction of hamstrings as knee extensors is initiated by having them execute their

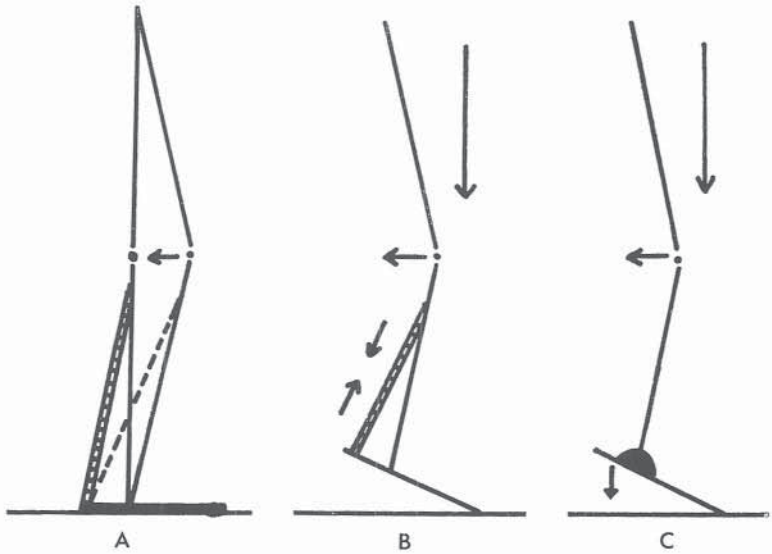


FIGURE 26–8 The effect of muscle-controlled or fixed talipes equinus on extension of the knee. **A,** Normal action of the soleus as an extensor of the knee with the foot on the ground. **B,** The soleus as a fixator of the foot in equinus position. **C,** A rigid equinus foot, showing the effect of body weight in extending the knee. *Vertical arrows* represent body weight; *horizontal arrows* indicate direction of movement on the knee joint. (From Robins RHC: The ankle joint in relation to arthrodesis of the foot in poliomyelitis. J Bone Joint Surg 1959;41-B:340; modified from Steindler, after von Baeyer.)

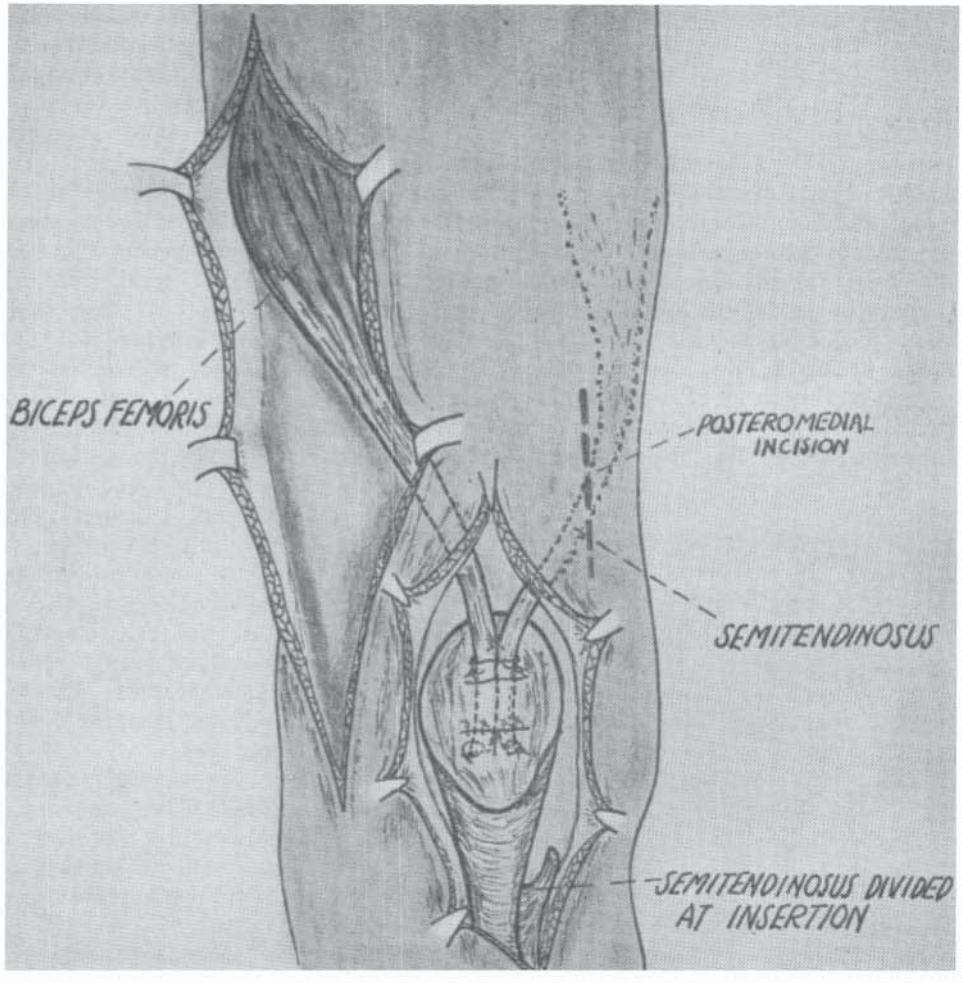


FIGURE 26–9 Transfer of semitendinosus and biceps femoris tendons to patella to restore knee extension. (From Schwartzmann JR, Crego CH: Hamstring tendon transplantation for the relief of quadriceps femoris paralysis in residual poliomyelitis. J Bone Joint Surg 1948;30-A:545.)

former action of hip extension. Then active guided knee extension exercises are performed from starting positions of greater knee flexion and decreasing hip flexion; the patient should soon be encouraged to divorce the two movements of knee and hip extension. The active exercise of knee extension is performed with the hip in the partially flexed position of the normal pattern of locomotion, without, however, extending the hip.

The function of the antagonistic muscles should not be ignored. Active knee flexion exercises are carried out (through a limited range initially), making sure that the transferred muscle is not used in both extensor and flexor functions.

As soon as the transferred muscle is fair minus in motor strength, the patient, while still supine in bed, is asked to go slowly through the motions of walking: namely, ankle-foot dorsiflexion and hip flexion, followed by knee extension (using the transfer), hip extension, and ankle plantar flexion. The same exercises are performed standing, first in parallel bars, and then in crutches. During the stance phase, hyperextension of the knee should be avoided. A bivalved cast is worn at night for 8 to 12 months to prevent stretching of the transferred muscles. Orthotic devices to support the knee usually are not necessary, unless their use is indicated for control of the foot and ankle.

Genu recurvatum is a not infrequent complication; it occurs in 10 to 20 percent of reported cases, being a natural consequence of an operation in which the hamstring muscles that normally provide dynamic support to the knee posteriorly are removed and transferred anteriorly. Other contributory factors in its pathogenesis are (1) pes equinus, (2) selection of patients with inadequate (less than fair) strength in the triceps surae muscle, (3) immobilization of the knee in hyperextension in the postoperative period, (4) lack of an adequate and diligent postoperative exercise regimen, with resultant failure to develop active knee flexion against gravity, and (5) improper use of orthotic support following surgery. The development of genu recurvatum can be minimized if the preceding factors are circumvented.

Lateral instability of the knee often results from inadvertent operative division of the tibial or fibular collateral ligaments while detaching the semitendinosus and biceps tendons from their insertion.

Lateral dislocation of the patella commonly occurs when the biceps femoris alone is transferred. This complication can be prevented by transfer of both the biceps and the semitendinosus muscles.

Failure of transfer may be due to denervation of the muscles during proximal dissection, to inadequate postoper-

ative training, or to binding down of the transfer by adhesions in sharp angular pathways to the patella.

FLEXION DEFORMITY OF THE KNEE

Contracture of the iliotibial band due to static forces of malposture of a flail lower limb will cause flexion contracture of the knee, along with flexion-abduction-external rotation deformity of the hip, genu valgum, and external tibial torsion. This deformity is preventable and, if minimal, can be corrected by passive exercises and wedging casts.[72,143] When it is marked, Ober-Yount open surgical release of the contracted iliotibial band will be required.

Flexion contracture of the knee may also result from a dynamic imbalance between the quadriceps femoris and hamstring muscles (Fig. 26–10). As stated previously, when there is flexion deformity of the knee, paralysis of the quadriceps muscle cannot be compensated for by locking the knee in hyperextension, and the knee will then be unstable. Thus, it is imperative that knee flexion deformity be fully corrected.

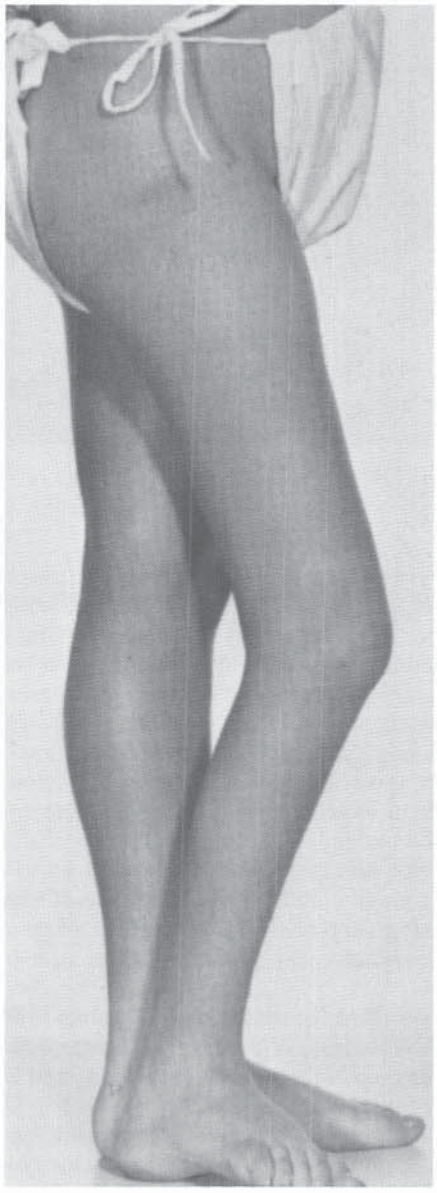


FIGURE 26–10 Flexion deformity of the right knee in poliomyelitis. A dynamic imbalance between the quadriceps femoris and hamstring muscles caused the deformity. Note also the calcaneovalgus deformity of the right foot.

It is important to understand the pathomechanics of a knee that has become fixed in flexion. In the normal knee, the last 5 degrees of extension is accompanied by medial rotation of the femur on the tibia—a movement that tightens the collateral ligaments and oblique posterior ligament, thus locking the knee in extension. Because the axis of knee motion passes not through the joint line but through the upper attachments of the collateral ligaments, the tibial plateau has to glide forward on the femoral condyles. In fixed flexion deformity of the knee this normal gliding movement does not take place; instead, a simple rocking motion occurs. When the knee is forced into extension, the tibia subluxates posteriorly, and the knee joint becomes incongruous and painful. In correcting fixed flexion deformity of the knee, it is important to preserve joint congruity by pulling the tibial plateau forward on the femoral condyles. This is accomplished by applying skeletal traction through a pin in the proximal tibia, following section of the contracted iliotibial band and patellar retinacular expansions that are usually adherent to the joint capsule and that obliterate the lateral recesses. Posterior capsulotomy of the knee is usually not required.

Supracondylar osteotomy may be indicated in cases in which fixed flexion deformity is very marked and there are structural bony changes in the femoral condyles. Osteotomy is also indicated to align the lower limb when significant genu valgum persists following correction of soft tissue contracture.[131]

Asirvatham and associates have warned against the use of a proximal tibial extension and medial rotation osteotomy to simultaneously correct knee flexion contracture and tibial lateral rotation deformity.[1a] *Recurrence of contracture, genu recurvatum, and peroneal palsy complicated the outcome.*

GENU RECURVATUM

Hyperextension of the knee in poliomyelitis may develop as a result of stretching of the soft tissues in the back of the knee, or it may be due to structural bone changes, with depression and downward sloping of the anterior portion of the tibial plateau.

The first type occurs when there is extensive paralysis of the lower limb with marked weakness of the hamstrings, triceps surae, and quadriceps femoris muscles (Fig. 26–11). There is often calcaneus deformity of the foot. With continued weightbearing, the hamstring and triceps surae muscles and the capsule and ligaments in the posterior aspect of the knee will stretch and elongate. The degree of genu recurvatum rapidly increases with loss of the support normally provided by the muscles and ligaments. Functional disability is usually great; an above-knee orthosis with a posterior knee strap is frequently required to support the knee. Heyman recommends the use of peroneal tendons to construct posterior check ligaments to prevent hyperextension of the knee.[152–154] When there is associated excessive lateral instability of the knee, the collateral ligaments are also reinforced. The tendons are passed through drill holes that are placed superior to the epiphyseal plate at the lower end of the

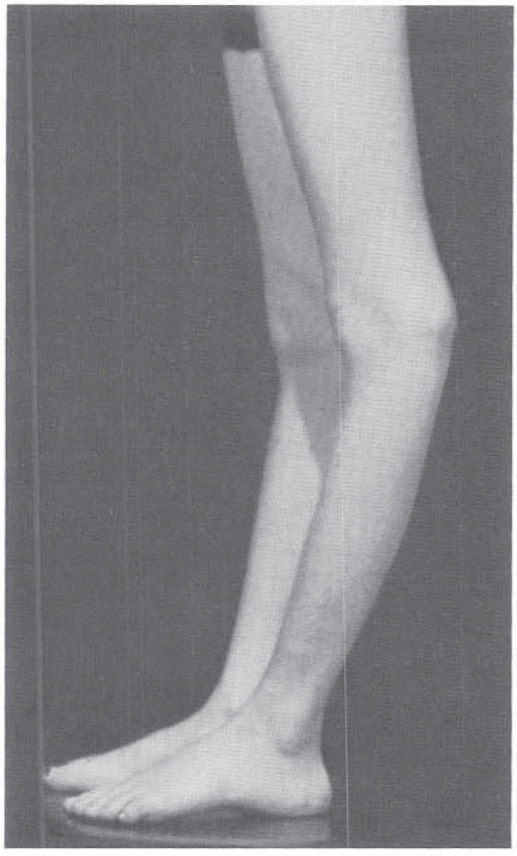


FIGURE 26–11 Genu recurvatum in a patient with chronic poliomyelitis.

femur and inferior to the epiphyseal plate of the upper end of the tibia, thus avoiding any injury to the epiphyseal plate. The tendons are firmly anchored with the knee in 30 degrees of flexion. An above-knee cast is worn for 6 weeks. The knee is then further protected for 3 months in an above-knee orthosis that limits extension of the knee at 5 degrees less than neutral. In a long-term follow-up note, Heyman reported complete and lasting correction in five cases, with extension of the knee limited to a point just short of neutral. In the experience of the author, however, under the forces of body weight, the tendons and shortened soft tissues eventually become stretched and the deformity recurs. The author recommends the Heyman tenodesis operation for genu recurvatum in a patient less than 10 years old, in whom osseous structural changes in the tibial plateau have not yet taken place. To prevent deformity from recurring until skeletal growth has been completed, the patient sleeps in a night bivalved cast, which holds the knee in 40 degrees of flexion. For walking, the knee is held in 5 to 10 degrees of flexion in an above-knee orthosis.

The second type of genu recurvatum develops when there is equinus deformity of the ankle with normal triceps surae and hamstring muscles but a weak quadriceps femoris muscle. The paralyzed quadriceps muscle is unable to lock the knee in neutral extension, and on heel strike the proximal end of the tibia is forced into hyperextension with limited dorsiflexion of the ankle. With continued walking and stresses of weightbearing, the anterior portion of the tibial plateau becomes depressed and is tilted inferiorly. The bony deformity is corrected either by open-up wedge osteotomy or by close-up wedge osteotomy of the proximal tibia.[169,312] It is usually performed at the subcondylar level distal to the proximal tibial tubercle. It is best to delay surgery until skeletal growth is completed. The technique described by Irwin is simple and very satisfactory (Figs. 26–12A and B).[169] A modified dome-shaped osteotomy will achieve the same result (Figs. 26–12C and D).

The author, however, prefers an open-up wedge osteotomy (Figs. 26–12E and F). The operative technique is as follows. A curved transverse incision is made across the anterior aspect of the leg, centered 1.5 cm distal to the proximal tibial tubercle. The lateral limb of the incision is continued proximally to terminate immediately superior and posterior to the upper end of the fibula. Subcutaneous tissue and fasciae are divided in line with the skin incision, and the wound flaps are mobilized and retracted. First, the neck and 2 cm of the proximal shaft of the fibula are extraperiosteally exposed. Meticulous attention must be paid to avoiding damage to the common peroneal nerve and proximal fibular epiphyseal plate (if open). With drill holes and a sharp thin osteotome, a simple short oblique osteotomy of the proximal shaft of the fibula is performed. Often it is desirable to excise a wedge of bone from the proximal fibula with its base posteriorly.

Next, a T-shaped incision is made in the periosteum over the anteromedial surface of the proximal tibia. The growing apophysis of the proximal tibial tubercle and the upper epiphyseal plate of the tibia should not be disturbed by stripping the periosteum. The level of osteotomy is immediately distal to the proximal tibial tubercle; its line is marked with a starter, and then drill holes are made through the anteromedial and lateral cortices, leaving the posterior cortex of the tibia intact.

Next, three large threaded Steinmann pins are chosen and their fit in the Roger Anderson apparatus is double-checked. Starting from the medial side, the first threaded Steinmann pin is placed transversely through the distal portion of the proximal fragment. The pin should just engage in the lateral cortex of the tibia (avoiding injury to the common peroneal nerve), and it should be more posterior in position, away from the proximal tibial tubercle. The second and third Steinmann pins are placed transversely through the distal fragment of the tibia 5 and 10 cm, respectively, distal to the osteotomy site. Then the tibia is divided with an osteotome, leaving the posterior cortex intact. If the proximal tibial fragment is kept in maximal hyperextension by a forward pull on the first Steinmann pin and manual pressure on the anterior surface of the knee and distal thigh, the leg and the distal segment of the tibia are forced posteriorly, creating a wedge-shaped defect at the osteotomy site with its base anteriorly. A lamina spreader may be used effectively to open up the wedge.

Osteotomes of different widths are placed into the osteotomy site to determine the size of the iliac bone graft wedges, which are taken in routine manner with both cortices intact. It is best to obtain radiographs with the proper osteotome placed at the osteotomy site to double-check the correction. The degree of angulation at the osteotomy site should be approximately 10 degrees greater than that of the genu recurvatum, and the longitudinal axis of the distal fragment of the tibia should be parallel with that of the femur. The proximal tibial fragment should be in hyperextension.

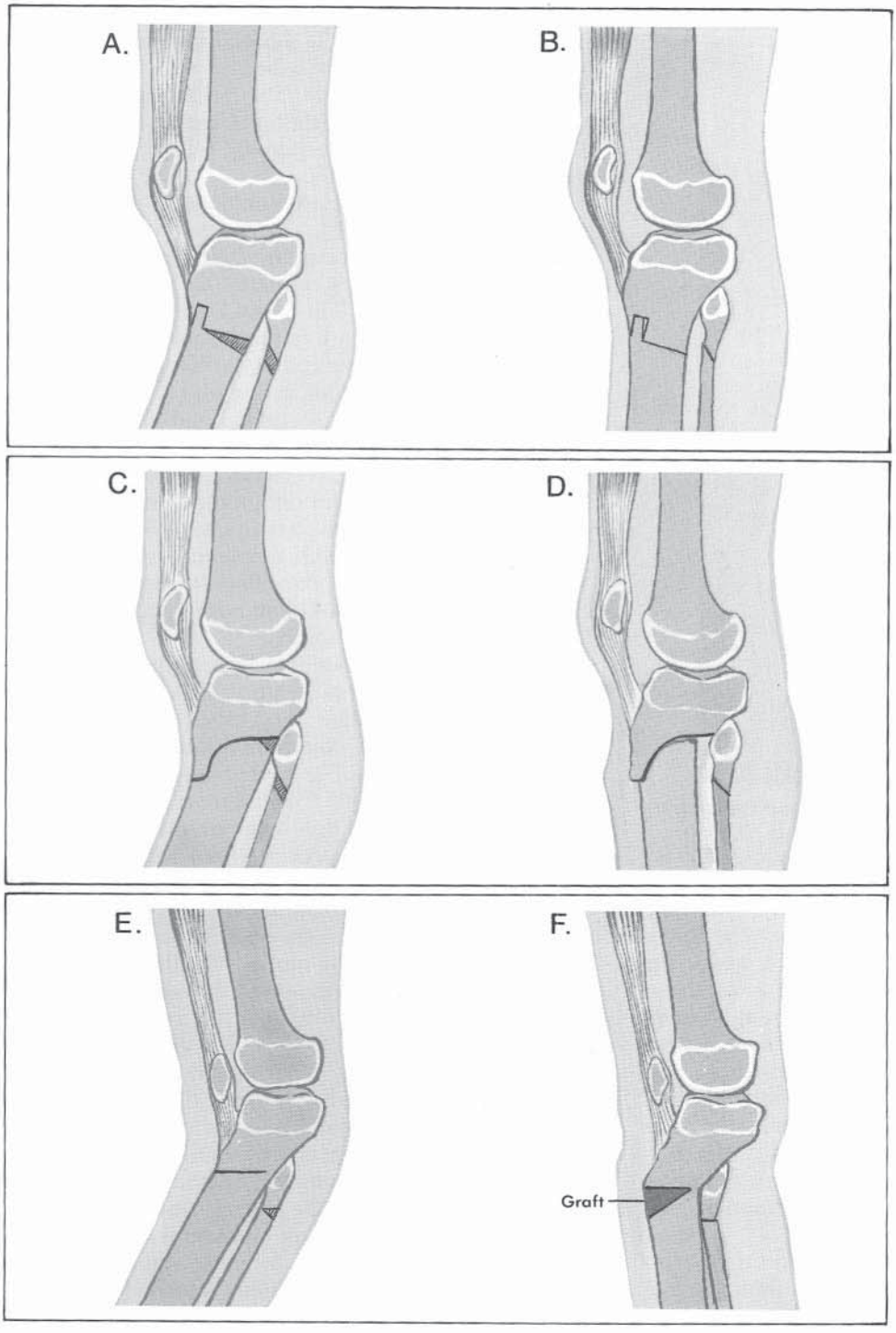


FIGURE 26–12 Surgical methods of correcting genu recurvatum. **A** and **B,** Irwin technique. **C** and **D,** Modified dome osteotomy. **E** and **F,** Open-up wedge osteotomy.

Next, two iliac bone graft wedges are placed at the osteotomy site (one is medial, the other lateral to the tibial crest); these are locked in place with an impactor. The surrounding spaces are firmly packed with bone graft chips. The lateral bars of the Roger Anderson apparatus are tightened to provide additional stability to the osteotomy site. The correction obtained is then rechecked with radiographs. The wound is closed in the usual manner. The Roger Anderson apparatus is padded with petrolatum gauze to prevent its incorporation into the cast. An above-knee cast is applied with the knee in extension.

Mehta and Mukherjee reported successful use of a femoral osteotomy to correct genu recurvatum, with flattening of the femoral condyles.[11a] *Deformity recurred in only one case.*

FLAIL KNEE

A flail knee is unstable (Fig. 26–13). For weightbearing it requires the support of an above-knee orthosis with a drop-lock knee. With such an orthosis, the patient is able to flex the knee while sitting. Arthrodesis of the knee should not be performed in children; it is best postponed until adult

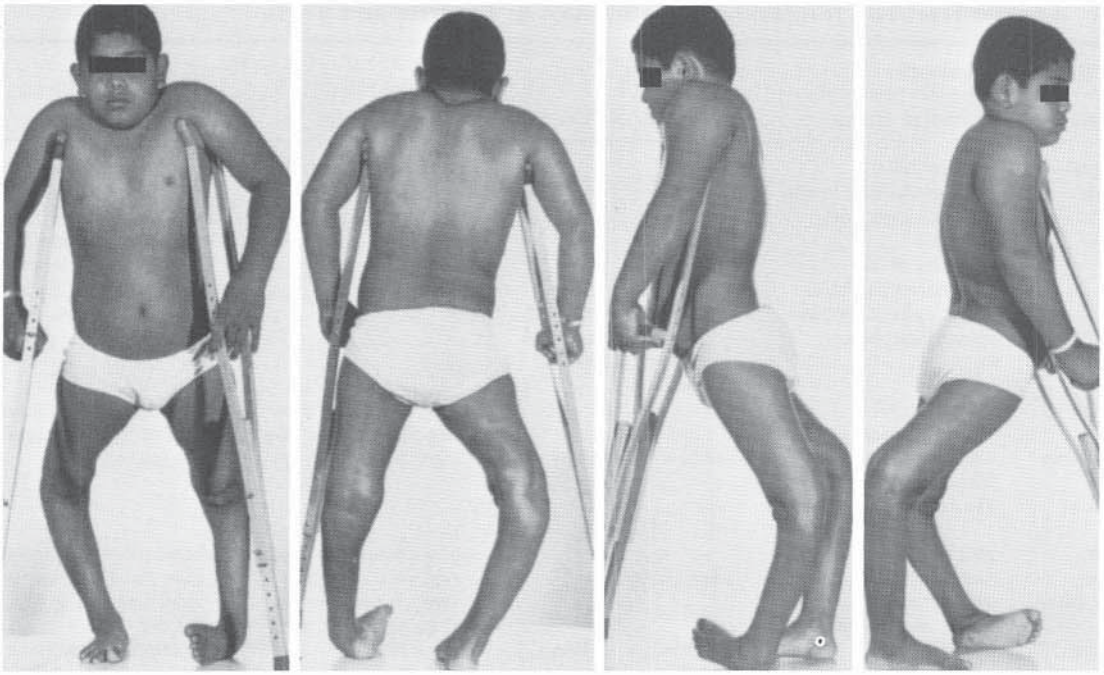


FIGURE 26–13 Bilateral unstable flail knees in a patient with chronic poliomyelitis.

life, when the patient is mature enough to understand and assess the advantages and disadvantages of a fused stiff knee. In unilateral involvement the author does not recommend arthrodesis of the knee, especially if there is associated muscle weakness of the hip and foot. When both lower limbs are paralyzed, however, one limb can be supported in an above-knee orthosis and the other knee stabilized by fusion, provided the hip has normal musculature and the foot is fixed in slightly equinus posture. The technical details of arthrodesis of the knee have been described in the literature.[66,228]

Men and associates reported good results in a large series of patients using soft tissue releases, extension osteotomies of the femur, and a patellar bone block for genu recurvatum.[12a]

Specific Deformities of the Foot and Ankle

Paralysis of the muscles acting on the foot may result in various deformities and functional disability of the foot, depending on the particular muscle or muscles involved and the strength of the remaining musculature.

Stability of the foot depends on several factors: the contour of the bones and the articular surfaces, the integrity of the ligamentous and capsular support, and the motor strength of the muscles. The combined mobility of the foot and ankle is equal to that of a universal joint. Motions of the ankle, subtalar, and midtarsal joints are related to each other. In inversion of the hindfoot, for example, the os calcis is displaced forward, producing adduction and inversion of the forefoot; when the hindfoot is everted, the os calcis moves backward and the forefoot is abducted and everted. When the ankle joint is plantar flexed, the hindfoot inverts, whereas in dorsiflexion of the ankle, the hindfoot everts. The foot is most stable in eversion and dorsiflexion and least stable in equinus position and inversion.

The muscles that produce *plantar flexion* are the gastrocnemius-soleus, flexor hallucis longus, flexor digitorum longus, peroneus longus, peroneus brevis, and posterior tibial. *Dorsiflexor muscles* are the anterior tibial, extensor hallucis longus, extensor digitorum communis, and peroneus tertius. The muscles that produce *inversion* are the posterior tibial, flexor hallucis longus, and anterior tibial; the *evertors* of the foot are the peroneus brevis, peroneus tertius, extensor digitorum communis, and extensor hallucis longus. The muscles that plantar flex the ankle and foot provide the force for forward propulsion of the body during locomotion. The dorsiflexor muscle group clears the foot during the swing phase of gait.

About two-thirds of the total musculature of the leg is constituted by the triceps surae, one of the strongest muscles in the body. It acts on the foot as a first-class lever with the ankle joint as a fulcrum. The working capacity of the triceps surae is 6.5 kg/m, whereas that of the dorsiflexors of the ankle joint is only 1.4 kg/m, or a relative ratio of 4 : 1. This gross discrepancy of muscle mass between the plantar flexors and dorsiflexors of the ankle is the result of developmental and mechanical factors. The strength of the calf muscles is a necessary antigravitational force against the elevated center of gravity of the body in the upright posture. Also, since the center of gravity of the human body falls anterior to the ankle joint, there is a strong rotatory component in ankle dorsiflexion that the triceps surae must counteract. The muscles that provide lateral stability to the foot in plantar flexion are the posterior tibial and peroneals, whereas in dorsiflexion it is provided by the action of the anterior tibial and extensor digitorum communis.

Muscle imbalance will produce progressive deformity. This deformity is flexible in the beginning, but with skeletal

TABLE 26–1 **Tendon Transfers for Paralytic Deformities of the Foot and Ankle**

Dynamic Imbalance				
Paralyzed or Weak	*Normal or Strong*	**Deformity of Foot**	**Tendon Transfer**	**Remarks**
Peroneus longus Peroneus brevis	*Anterior tibial* Extensor hallucis longus Extensor digit. communis Posterior tibial Gastrocnemius-soleus Flexor hallucis longus Flexor digit. longus	Varus Dorsal bunion (first metatarsal dorsiflexed because of unopposed action or anterior tibial)	Lateral transfer of anterior tibial to base of second metatarsal	Perform transfer before fixed deformity develops. Lateral stability will be retained. Do not transfer more lateral than second metatarsal in presence of strong extensor digit. communis (will cause pes valgus).
Peroneus longus Peroneus brevis Extensor digit. communis Extensor hallucis longus	*Anterior tibial* Posterior tibial Gastrocnemius-soleus Flexor hallucis longus Flexor digit. longus	Varus, some equinus	Lateral transfer of anterior tibial to base of third metatarsal	Do not transfer further lateral than base of third metatarsal (will cause pes valgus).
Peroneus longus Peroneus brevis Extensor digit. communis Extensor hallucis longus Anterior tibial	*Posterior tibial* Gastrocnemius-soleus Flexor hallucis longus Flexor digit. longus	Equinovarus	Anterior transfer of posterior tibial tendon through interosseous space to base of third metatarsal	Postoperatively, equinovarus deformity should be fully corrected by stretching cast or soft tissue surgery. May consider reinforcing posterior tibial transfer by adding flexor hallucis longus or flexor digit. longus to anterior transfer through interosseous space; anterior tenodesis to prevent dropping down of foot is another choice. Postoperatively, support transfer by dorsiflexion-assist below-knee orthosis.
Anterior tibial	*Peroneus longus* Peroneus brevis Extensor hallucis longus Extensor digit. communis Gastrocnemius-soleus Posterior tibial Flexor hallucis longus Flexor digit. longus	Equinovalgus Cock-up deformity of toes (overactivity of toe extensors displaces proximal phalanges of toes into hyperextension and depresses metatarsal heads) Occasionally cavovarus deformity of foot results (unopposed peroneus longus acts as depressor of first metatarsal)	Anterior transfer of peroneus longus to base of second metatarsal (suture peroneus brevis to distal stump of peroneus longus)	Do not attach peroneus longus to first metatarsal (will displace it upward and cause dorsal bunion). Transfer long toe extensors to heads of metatarsals if cock-up deformity of toes is present. If both peroneals are transferred, lateral instability of foot will develop, necessitating stabilization by subtalar extra-articular or triple arthrodesis.
Gastrocnemius-soleus (motor strength zero or trace)	*Peroneus longus* *Peroneus brevis* *Flexor hallucis longus* *Posterior tibial* Flexor digit. longus *Anterior tibial* Extensor hallucis longus Extensor digit. communis	Calcaneus or calcaneocavus	Posterior transfer (to os calcis) of both peroneals, posterior tibial, and flexor hallucis longus	*Caution*—Prevent development of dorsal bunion by lateral transfer of anterior tibial to base of second metatarsal within a year. In adolescent patient with fixed calcaneus deformity, before tendon transfers, perform triple arthrodesis with posterior shift of os calcis to correct bony deformity. In young child, calcaneus deformity will correct with subsequent growth; however, subtalar extra-articular arthrodesis may be required for lateral stability.

growth, fixed soft tissue and structural osseous deformity will develop. The deformities of the foot and loss of function produced by muscle imbalance are predictable. The *dynamic imbalance* from paralysis of the major muscle groups, the resultant deformity, and its treatment are presented in Table 26–1.

PARALYSIS OF PERONEAL MUSCLES

When the peroneus longus and brevis muscles are paralyzed, the os calcis is pulled into inversion by the strong posterior tibial muscle. The forefoot adducts following inversion of the hindfoot and also because of the unopposed action

TABLE 26–1 **Tendon Transfers for Paralytic Deformities of the Foot and Ankle** *Continued*

Dynamic Imbalance				
Paralyzed or Weak	*Normal or Strong*	**Deformity of Foot**	**Tendon Transfer**	**Remarks**
Gastrocnemius-soleus (motor strength poor)	As above	Calcaneus or calcaneocavus	Posterior transfer (to os calcis) of posterior tibial and peroneus longus	Suture distal stump of peroneus longus to peroneus brevis. Watch closely for possible development of dorsal bunion; lateral transfer of anterior tibial to base of second metatarsal may be indicated.
Gastrocnemius-soleus Posterior tibial Peroneus longus Peroneus brevis	*Anterior tibial* *Flexor hallucis longus* Extensor hallucis longus Extensor digit. communis Flexor digit. longus	Calcaneovarus	Posterior transfer (to os calcis) of anterior tibial and flexor hallucis longus	Suture distal stump of flexor hallucis longus to flexor hallucis brevis. Interphalangeal joint fusion of great toe may be necessary.
Anterior tibial Gastrocnemius-soleus	*Peroneus longus* *Peroneus brevis* *Posterior tibial* Flexor hallucis longus Flexor digit. longus Extensor hallucis longus Extensor digit. longus	Calcaneovarus	Posterior transfer (to os calcis) of both peroneals and posterior tibial	Perform triple arthrodesis in adolescence to provide lateral stability to hindfoot.
Gastrocnemius-soleus Posterior tibial Peroneus longus Peroneus brevis Flexor hallucis longus Flexor digit. longus	*Arterior tibial* Extensor hallucis longus Extensor digit. communis	Calcaneovarus	Posterior transfer (to os calcis) of anterior tibial	Protect transfer with plantar flexion–assist orthosis until skeletal maturity. Consider tendo Achillis tenodesis. In adolescence, if adequate function exists in transferred anterior tibial, foot is stabilized by triple arthrodesis. If anterior tibial function is inadequate, Chuinard-type ankle fusion is performed (will provide stability and gait will improve considerably).
Gastrocnemius-soleus Posterior tibial Peroneus longus Peroneus brevis Flexor hallucis longus Flexor digit. longus Anterior tibial	Extensor hallucis longus Extensor digit. communis	Calcaneovalgus (minimal)	Ankle fusion (Chuinard type)	Stability and muscle control of knee should be adequate. Full knee extension and functioning hamstrings are prerequisite.
Flail ankle and foot (all muscles paralyzed)	None except short toe flexors and intrinsic muscles of foot	Flexion of toes and metatarsus varus Hindfoot neutral or valgus (may be in inversion due to contracture of plantar fascia)	Pantalar arthrodesis Resect motor branches of plantar nerves	As above
Anterior tibial Extensor hallucis longus Extensor digit. communis Peroneus longus Peroneus brevis Posterior tibial	Gastrocnemius-soleus Flexor digit. longus Flexor hallucis longus	Equinis	Anterior transfer of flexor digit. longus and flexor hallucis through interosseous space Anterior tenodesis	Do not lengthen tendo Achillis (will produce calcaneus deformity). Disability is little (patient must lift leg to clear toes). Stretch triceps surae, use night support to prevent fixed equinus deformity.

of the anterior tibial muscle. Gradually, a varus deformity of the foot is produced (Fig. 26–14). Normally the peroneus longus depresses the first metatarsal and the anterior tibial raises it. When the peroneus longus muscle is paralyzed, the first metatarsal becomes dorsiflexed by the unopposed action of the anterior tibial, and a dorsal bunion will result. The opposing actions of the peroneus longus and anterior tibial muscles on the first metatarsal should always be considered whenever there is a dynamic imbalance between the two.

Treatment consists of lateral transfer of the anterior tibial to the base of the second metatarsal bone. Lateral stability of the foot will then be adequate, and arthrodesis is not required.

PARALYSIS OF PERONEALS, EXTENSOR DIGITORUM LONGUS, AND EXTENSOR HALLUCIS LONGUS

The deformity resulting from paralysis of these muscles will be moderately varus and somewhat equinus. Dynamic bal-

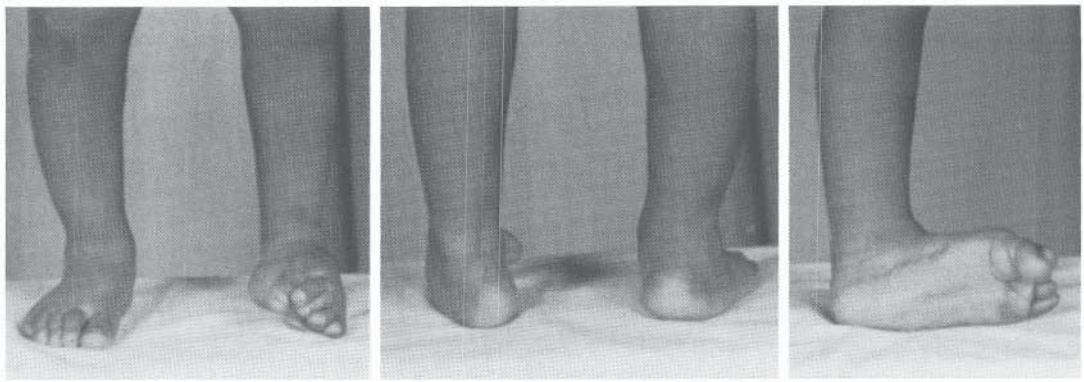


FIGURE 26–14 Paralytic pes varus. The deformity is the result of paralysis of the peroneus longus and brevis muscles. The hindfoot is inverted by the pull of the strong posterior tibial muscle, and the forefoot is adducted and inverted by the unopposed action of the anterior tibial muscle. Note that the first metatarsal is dorsiflexed, producing a dorsal bunion.

ance of the foot is restored by lateral transfer of the anterior tibial muscle to the base of the third metatarsal bone. Pes valgus may result if the anterior tibial is transferred to the fourth or fifth metatarsal bone. The operative technique of lateral transfer of the anterior tibial tendon is as follows.

A longitudinal incision is made over the medial aspect of the foot, beginning at the base of the first metatarsal bone and extending proximally parallel to the course of the anterior tibial tendon for a distance of 3 cm. The anterior tibial tendon is detached from its insertion into the base of the first metatarsal bone and the medial surface and undersurface of the first cuneiform bone. A Mersilene or Dacron whip suture is inserted into the distal end of the tendon. By sharp dissection, the tendon is mobilized over the dorsum of the foot. The dorsalis pedis artery, lying between the tendon of the extensor hallucis longus and the first tendon of the extensor digitorum longus, should not be divided.

Then a second 8- to 10-cm longitudinal incision is made over the anterior tibial compartment in the distal third of the leg, beginning at the upper border of the transverse crural ligament. The subcutaneous tissue and deep fascia are divided. The anterior tibial tendon is located immediately on the tibia. The anterior tibial vessels lie between the anterior tibial and extensor hallucis longus muscles in the middle third of the leg. At the ankle, the extensor hallucis longus tendon crosses the anterior tibial vessels from the lateral to the medial side. The deep peroneal nerve is located on the lateral side of the anterior tibial vessels in the upper third of the leg, in front of the artery in the middle third, and then again lateral in the distal third. Care should be taken not to injure the deep peroneal nerve and the anterior tibial vessels. The anterior tibial sheath is divided and by gentle traction, using the two-hand technique, the tendon is delivered into the proximal wound. Transfer of the anterior tibial tendon on the dorsum of the foot distal to the transverse crural ligament from the medial to the lateral side will not correct the varus action of the muscle.

Next, an incision 3 cm long is made over the dorsum of the foot with its center over the base of the third metatarsal bone. With an Ober tendon passer, the anterior tibial tendon is delivered into the dorsum of the foot, passing deep to the transverse crural ligament to produce straight dorsiflexion. It is securely fixed to the base of the third metatarsal bone with the ankle joint in neutral position or dorsiflexed 5 degrees. The muscle should be under physiologic tension. The wounds are closed in routine fashion and a long-leg cast is applied, with the ankle in 5 degrees of dorsiflexion and the knee in 45 degrees of flexion.

PARALYSIS OF ANTERIOR TIBIAL MUSCLE

Dorsiflexor and inversion power of the foot is lost when the anterior tibial muscle is paralyzed, and equinovalgus deformity of the foot will develop (Fig. 26–15). The toe extensors are overactive in an attempt to substitute for the action of the anterior tibial muscle in dorsiflexion of the ankle. The proximal phalanges of the toes become hyperextended and depress the metatarsal heads, causing cock-up deformity of the toes. Equinus deformity of the ankle gradually results from contracture of the triceps surae. Occasionally cavovarus deformity of the foot may result because of the action of the peroneus longus muscle, which acts as a depressor of the first metatarsal. On active dorsiflexion of the ankle, the forefoot is everted, but on weightbearing it goes into inversion to permit horizontal contact of all metatarsal heads with the ground. The heel will invert following the forefoot inversion.

During the convalescent phase of poliomyelitis, aggressive measures should be taken to retain passive range of dorsiflexion of the ankle joint. Passive heel cord stretching exercises are performed every day. At night, a bivalved cast or a plastic splint is used to hold the ankle in neutral position, and during the day an ankle-foot dorsiflexion-assist orthosis supports the ankle and foot.

If proper treatment is neglected, fixed equinus deformity may develop. In that event, the heel cord should not be lengthened, and every effort should be made to retain full function of the triceps surae muscle. Range of dorsiflexion of the ankle may be obtained with a wedging cast or a below-knee walking cast with an anterior heel. In severe fixed equinus deformity, posterior capsulotomy of the ankle and subtalar joints is performed and the heel cord is stretched with skeletal traction through a threaded Steinmann pin in the os calcis. Functional disability is great following loss of plantar flexion power.

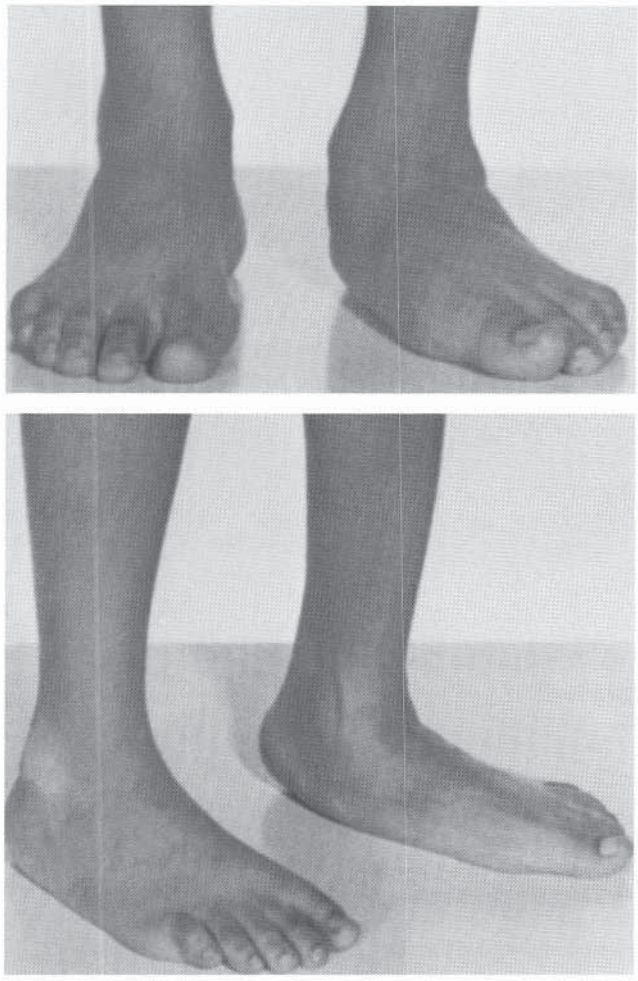


FIGURE 26–15 Equinovalgus deformity of the foot as a result of paralysis of the anterior tibial muscle.

Dorsiflexion power of the ankle is restored by anterior transfer of the peroneus longus tendon to the base of the second metatarsal bone. The peroneus brevis is sutured to the distal stump of the peroneus longus. The operative technique and postoperative care for anterior transfer of the peroneus longus are described and illustrated in Plate 26–4. The peroneus longus tendon should not be attached to the base of the first metatarsal, since it will displace the bone upward and cause a dorsal bunion. If there is a cock-up deformity of the toes, the long toe extensors are transferred to the heads of the metatarsals. If both peroneals are transferred, lateral instability of the foot develops, requiring stabilization of the hindfoot by extra-articular subtalar arthrodesis or triple arthrodesis.

PARALYSIS OF ANTERIOR TIBIAL MUSCLE, TOE EXTENSORS, AND PERONEALS

Equinovarus deformity of the foot will develop from the unopposed action of the posterior tibial and triceps surae muscles (Fig. 26–16). Treatment consists of anterior transfer of the posterior tibial tendon through the interosseous space to the base of the third metatarsal or second cuneiform (Plate 26–5). Preoperatively, equinovarus deformity should be fully corrected by a stretching cast. Soft tissue release may be indicated for correction of the fixed pes varus.

The flexor digitorum longus or flexor hallucis longus may be transferred anteriorly through the interosseous route to reinforce the strength of dorsiflexion power of the posterior tibial transfer. Anterior tenodesis is another method of preventing the foot from dropping down in plantar flexion. In the postoperative period, the anterior transfer and tenodesis should be supported in an ankle-foot dynamic dorsiflexion-assist orthosis during the day and a bivalved cast or plastic splint at night.

PARALYSIS OF TRICEPS SURAE MUSCLE

When the gastrocnemius and soleus muscles are weak or paralyzed, the patient walks with a calcaneus limp; that is, there is weakness or lack of push-off. The tibia is displaced posteriorly on the talus by the forward thrust of the trunk, and the foot is forced into excessive dorsiflexion at the ankle joint.

The tendo Achillis inserts into the posterior aspect of the apophysis of the os calcis. Normally the force exerted by the triceps surae muscle elevates the heel, depresses the anterior end of the os calcis, and pushes the body forward. The longitudinal arch is flattened as the head of the talus plantar flexes with the anterior end of the os calcis. In paralysis of the gastrocnemius and soleus muscles, the head of the talus and the anterior end of the os calcis are displaced upward to a more vertical position. This results in disappearance of the normal prominence of the heel and an increase in the range of dorsiflexion of the ankle (Fig. 26–17). When the accessory plantar flexor muscles (i.e., the posterior tibial, flexor hallucis longus, flexor digitorum longus, and peroneals) are strong, the forefoot is forced into equinus position, producing a calcaneocavus deformity. The foot is shortened by plantar flexion of the metatarsals and by rotation of the os calcis into a vertical position. Soon the plantar fascia and short flexors of the toes will contract and act as a bowstring, pulling together the metatarsal heads and the os calcis and increasing the cavus deformity (Fig. 26–18). The calcaneocavus deformity progressively increases with every step. With paralysis of the triceps surae, growth of the apophysis of the os calcis is retarded. This is particularly important in a young child, in whom, following an early and successful posterior tendon transfer to the os calcis, the calcaneus deformity of the heel may be restored to normal.

The triceps surae muscle is the strongest muscle of the foot. Therefore, it is desirable to transfer three or four muscles posteriorly to the os calcis, depending on their availability and the degree of weakness of the triceps surae. Plantar flexion at the ankle is more important functionally than dorsiflexion.

When the motor strength of the triceps surae muscle is zero, both peroneus longus and brevis, the posterior tibial, and the flexor hallucis longus are transferred to the os calcis. The anterior tibial is transferred laterally to the base of the second metatarsal within a year to prevent formation of a dorsal bunion. In adolescents with fixed calcaneus deformity, the hindfoot is stabilized by triple arthrodesis, the bony deformity is corrected, and the os calcis is shifted posteriorly. In a young child, calcaneus deformity will be

Text continued on page 1372

Anterior Transfer of Peroneus Longus Tendon to Base of Second Metatarsal

OPERATIVE TECHNIQUE

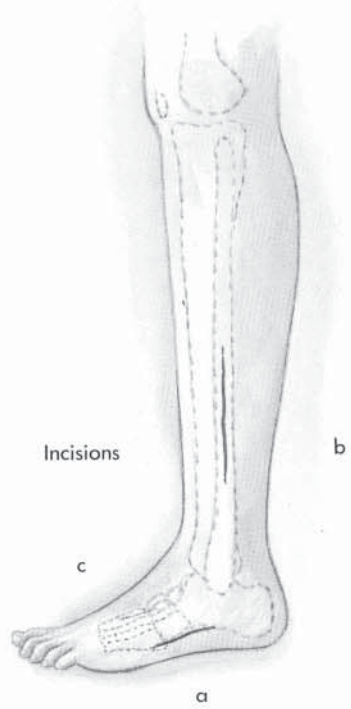
The patient is placed in semilateral position with a sandbag under the hip on the affected side.

A, A 3- to 4-cm-long incision is made over the lateral aspect of the foot, extending from the base of the fifth metatarsal to a point 1 cm distal to the tip of the lateral malleolus. Subcutaneous tissue is divided, and the tendons of the peroneus longus and brevis are exposed. Then a second incision is made over the fibular aspect of the leg; it begins 3 cm above the lateral malleolus and extends proximally for a distance of 7 cm. Subcutaneous tissue and deep fascia are incised, and the peroneal tendons are exposed by dividing their sheath. The peroneus longus tendon lies superficial to that of the peroneus brevis. The muscle is inspected to ensure that it is of normal gross appearance.

B, Next, the peroneus brevis muscle is detached from the base of the fifth metatarsal and a whip suture is inserted into its distal end.

C and **D,** The peroneus longus tendon is divided as far distally as possible. The peroneus brevis is sutured to the distal stump of the peroneus longus to preserve the longitudinal arch and depression of the first metatarsal.

PLATE 26–4. Anterior Transfer of Peroneus Longus Tendon to Base of Second Metatarsal



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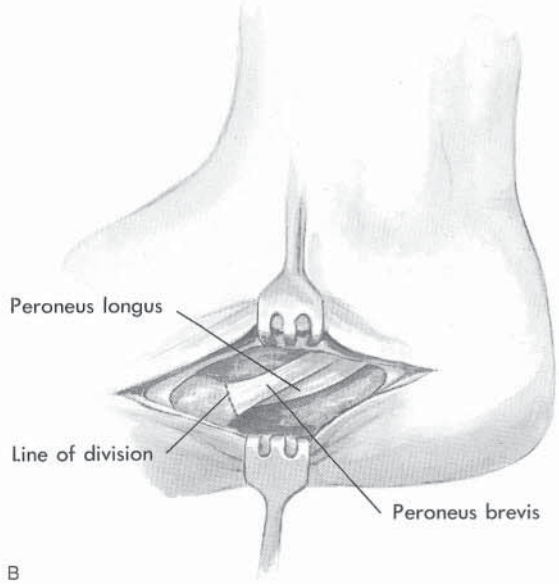

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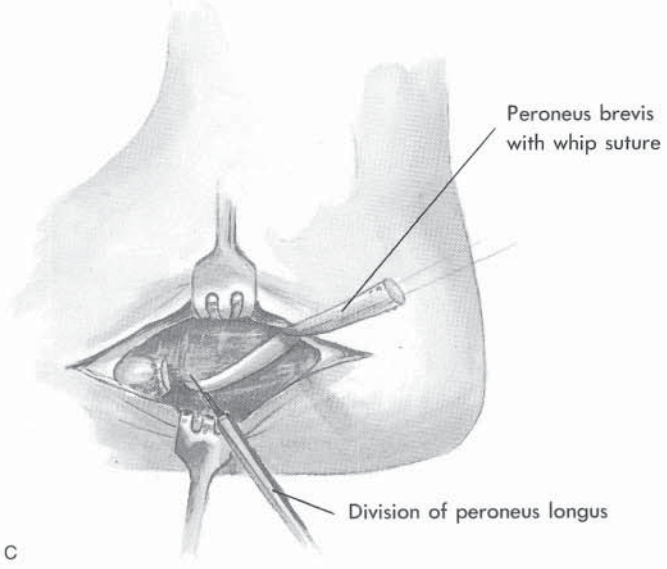

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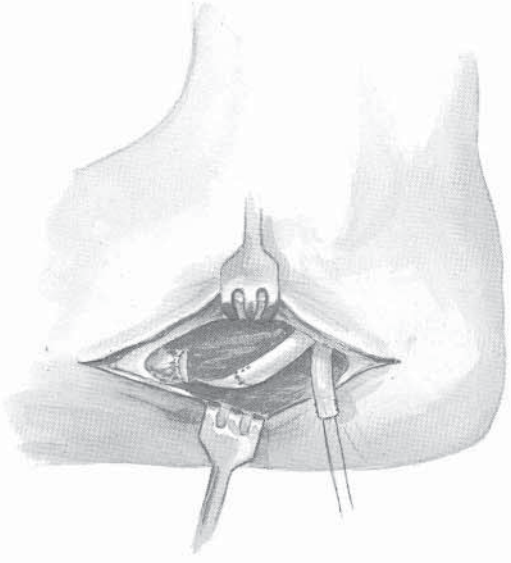
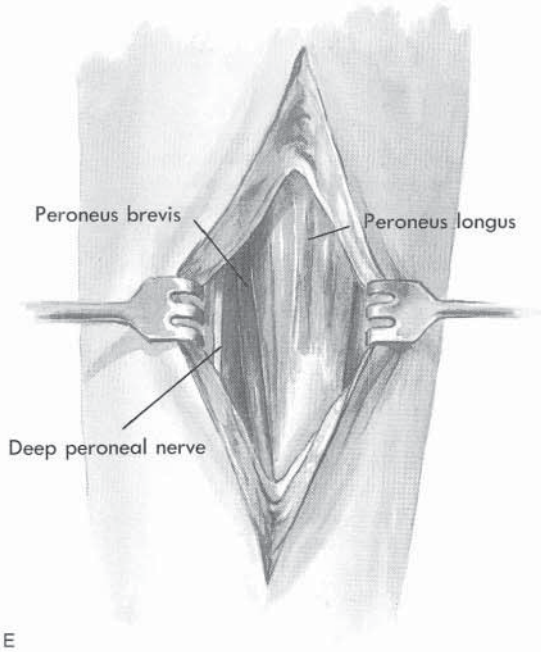


D Stump of longus sutured to brevis

Anterior Transfer of Peroneus Longus Tendon to Base of Second Metatarsal *Continued*

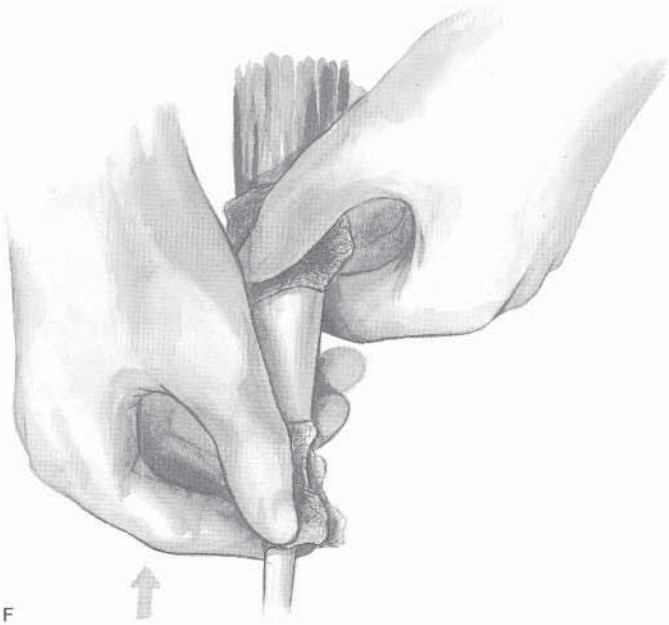
E and **F,** The peroneus longus tendon is mobilized and, with the two-hand technique, gently pulled into the proximal wound in the leg. The origin of the peroneus brevis from the fibula should not be disrupted. An adequate opening is made in the intermuscular septum, taking care not to injure neurovascular structures.

PLATE 26–4. Anterior Transfer of Peroneus Longus Tendon to Base of Second Metatarsal



E

Pulling tendon into proximal wound with two hand technique



F

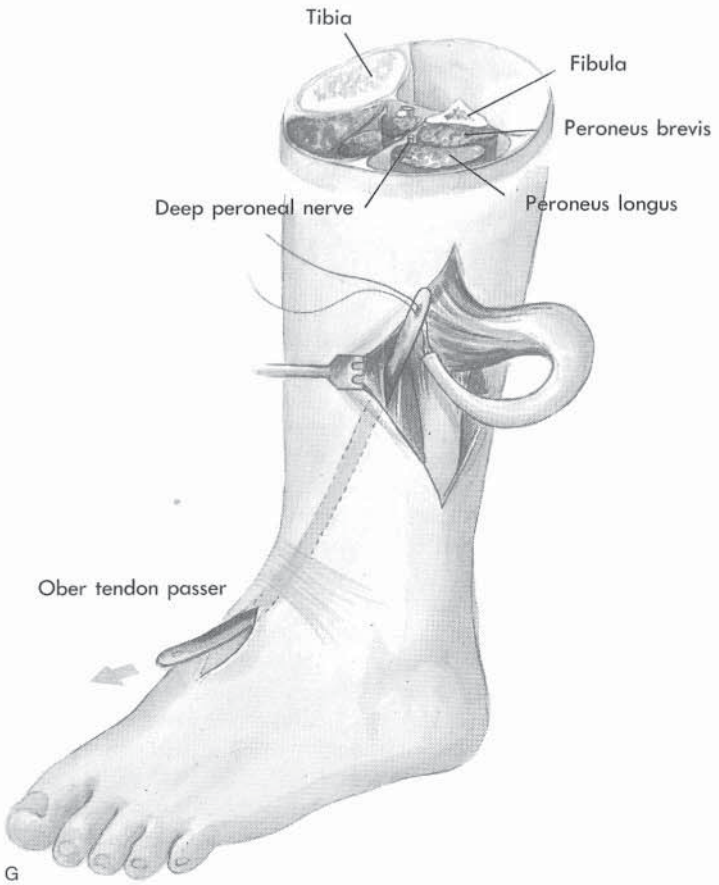
Anterior Transfer of Peroneus Longus Tendon to Base of Second Metatarsal *Continued*

G and **H,** A 2- to 3-cm-long longitudinal incision is made over the dorsum of the foot, centered over the base of the second metatarsal bone. The deep fascia is divided, and the extensor tendons are retracted to expose the proximal one-fourth of the second metatarsal bone. The periosteum is divided longitudinally and the cortex of the recipient bone is exposed.

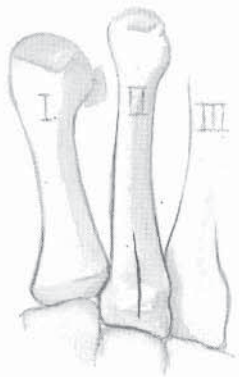
With an Ober tendon passer, the peroneus longus tendon along with its sheath is passed into the anterior tibial compartment, deep to the cruciate crural and tarsal ligaments, and delivered into the incision on the dorsum of the foot. The author does not recommend a subcutaneous route. A direct line of pull of the peroneus longus tendon from its origin to its insertion should be ensured.

I and **J,** A drill hole is made in the base of the second metatarsal. A star-head hand drill is used to enlarge the hole to receive the tendon adequately. The peroneus longus tendon is passed through the recipient hole and sutured on itself under correct tension. If the peroneus longus tendon is not of adequate length, two small holes are made 1.5 cm distal to the large hole at each side of the metatarsal shaft. The silk sutures at the end of the tendon are passed from the large central hole to the lateral distal small holes and the tendon is securely sutured to the bone. The ankle joint should be in neutral position or 5 degrees of dorsiflexion. The pneumatic tourniquet is released and hemostasis is obtained. The wounds are closed in routine manner. A long-leg cast is applied with the ankle in 5 degrees of dorsiflexion and the knee in 45 degrees of flexion. Postoperative care follows the guidelines outlined in the section on principles of tendon transfer (see text).

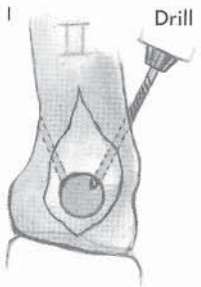
PLATE 26–4. Anterior Transfer of Peroneus Longus Tendon to Base of Second Metatarsal

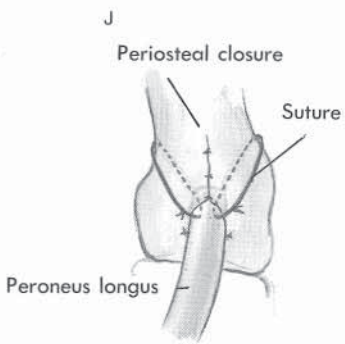


G



H Periosteal incision





Technique of anchoring tendon to bone

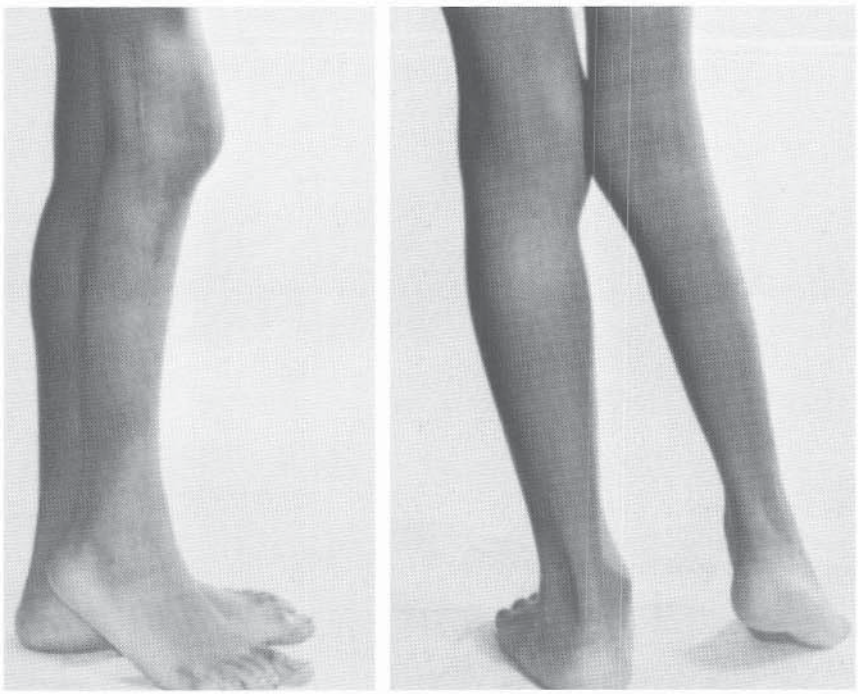


FIGURE 26–16 Equinovarus deformity of the right ankle and foot caused by unopposed action of the triceps surae and posterior tibial muscles.

corrected by subsequent growth if the posterior transfer is successful; however, a subtalar extra-articular arthrodesis may be required later for lateral stability.

Only the peroneus longus and posterior tibial muscles are transferred when the gastrocnemius-soleus muscles are poor in motor strength. The distal stump of the peroneus longus is sutured to the peroneus brevis. If a dorsal bunion tends to develop, lateral transfer of the anterior tibial to the base of the second metatarsal may again be indicated.

When the posterior tibial and peroneal muscles are paralyzed along with the triceps surae, the muscles available for posterior transfer are the anterior tibial and flexor hallucis longus. The flexor digitorum longus may be added if necessary. The interphalangeal joints, particularly that of the great toe, are fused.

When the anterior tibial muscle is paralyzed with the triceps surae, posterior transfer of both peroneals and the posterior tibial is performed; lateral stability of the hindfoot is provided by triple arthrodesis.

The anterior tibial muscle is transferred to the os calcis when all the plantar flexor muscles are paralyzed. The posterior transfer is protected with a plantar flexion-assist ankle-foot orthosis until skeletal maturity. Tendo Achillis tenodesis may be performed to provide posterior stability to the ankle joint. In adolescence, if plantar flexion function of the transferred anterior tibial is adequate, the hindfoot is stabilized by triple arthrodesis.

When the anterior tibial function is inadequate, a Chuinard-type ankle fusion is performed. When only the toe extensors are functioning, no muscles will be available for posterior transfer. An ankle fusion is performed, provided there is adequate stability and muscle control of the knee.

The operative technique and postoperative care for posterior tendon transfer to the os calcis are presented in Plate 26–6.

A dorsal bunion is characterized by dorsiflexion of the first metatarsal and plantar flexion of the great toe (Fig. 26–19). It is caused by muscle imbalance–weakness or absence of the peroneus longus muscle (plantar flexor of the first metatarsal) against normal strength of the anterior tibial muscle (dorsiflexor of the first metatarsal) or flexor hallucis brevis and longus. There are two types of dorsal bunion: one in which there is primary dorsiflexion of the first metatarsal and secondary plantar flexion of the hallux, and another in which there is primary plantar flexion of the hallux with resultant upward displacement of the metatarsal head. The author has treated dorsal bunion successfully by open-up wedge osteotomy of the base of the first metatarsal and transfer of the flexor hallucis longus to the head of the first metatarsal. McKay transfers tendinous insertions of the flexor hallucis brevis and abductor and adductor hallucis to the neck of the first metatarsal (Fig. 26–20). He believes these muscles provide greater mechanical advantage after transfer than the flexor hallucis longus; furthermore, the deforming action of the flexor hallucis brevis is also removed. The operative technique is as follows. A longitudinal incision is made on the medial aspect of the foot, extending from the base of the first metatarsal to the interphalangeal joint of the great toe. The abductor hallucis and medial part of the flexor hallucis brevis are identified, detached from their insertion, and dissected free; as much tendon as possible should be preserved (Fig. 26–21A). Then the lateral tendon of the flexor hallucis brevis and the tendon of the adductor hallucis are sectioned from their insertion and dissected free. The sesamoid bones in the tendinous part of the flexor hallucis brevis are carefully removed. If there is associated hallux valgus, the abductor hallucis is left intact; if hallux varus is present, the adductor hallucis is left attached to its insertion. Next, a circumferential incision is made in the periosteum of the first metatarsal at its neck and elevated for a distance of 1 cm. The abductor hallucis and medial part of the flexor hallucis brevis are transferred medially to

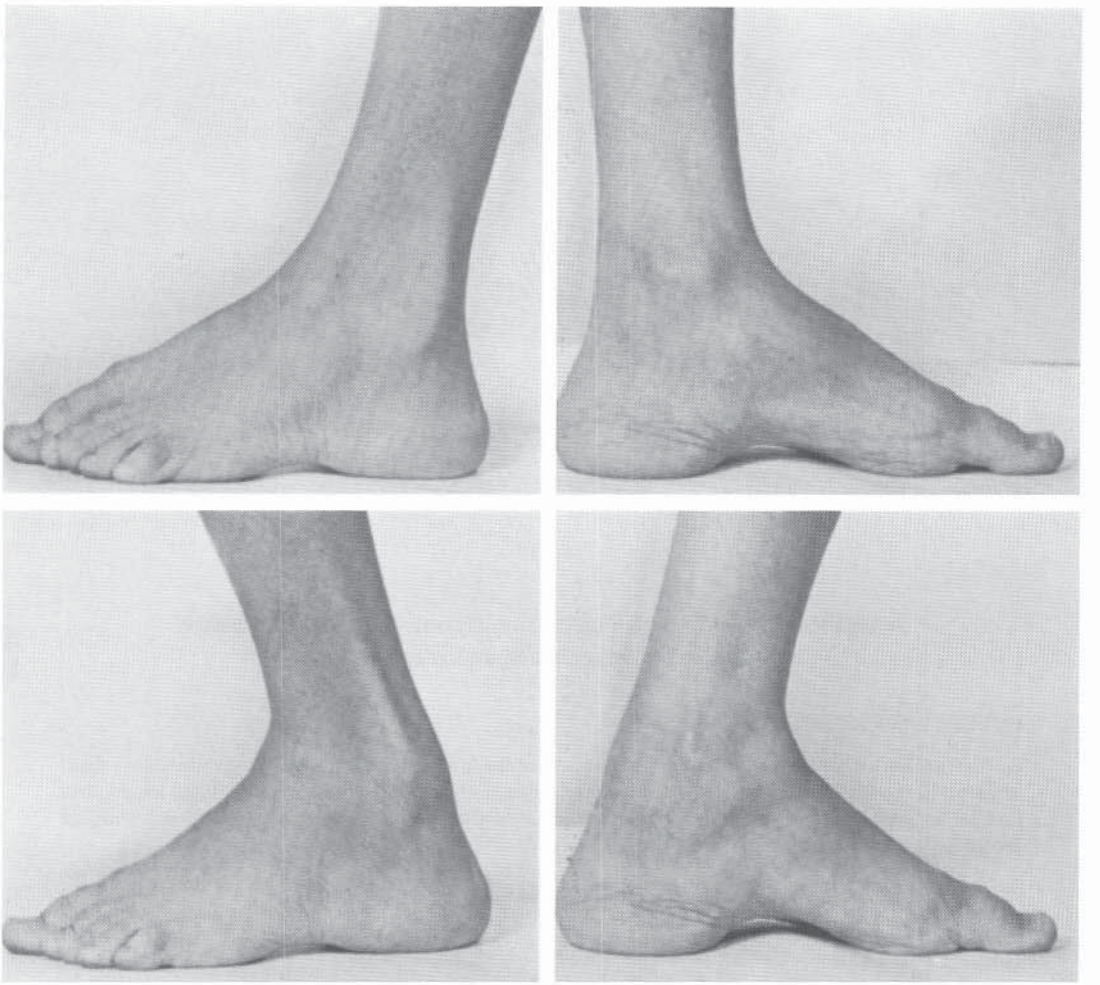


FIGURE 26–17 Calcaneus deformity of the foot and ankle. Note the posterior shift of the tibia over the talus during push-off.

the dorsal aspect of the metatarsal neck; the adductor hallucis and lateral part of the flexor hallucis brevis are transferred dorsally between the first and second metatarsals (Fig. 26–21B). All four tendons are sutured together, creating a myotendinous ring around the neck of the metatarsal (Figs. 26–20C and 26–21C). The collar of periosteum is sutured to the tendon. Next the interphalangeal joint of the great toe is stabilized by tenodesis or arthrodesis. A below-knee walking cast is applied and worn for 3 to 4 weeks. McKay reported complete correction in ten feet (Figs. 26–22 and 26–23).

Westin and associates reported that tenodesis of the tendo achillis to the fibula resulted in improved gait in 66 consecutive patients.[15a] *However, 16 feet (23 percent) required revision because of the development of an equinus contracture.*

Arthrodesis of the Foot and Ankle

In the operative treatment of the paralyzed foot, a multitude of surgical procedures have been developed to provide stability, to correct deformity, and to improve function.

The history of the evolution of stabilizing operations of the foot and ankle is given in Table 26–2. Detailed accounts of these procedures can be found in the original articles and in the comprehensive historical reviews by Hart, Hallgrimsson, and Schwartz.[134,142,284]

In general, stabilizing operations on the foot and ankle can be subdivided into the following: (1) triple arthrodesis, (2) extra-articular subtalar arthrodesis, (3) ankle fusion, and (4) anterior or posterior bone blocks to limit motion at the ankle joint. These procedures may be performed alone or in combination.

TRIPLE ARTHRODESIS

This procedure was devised by Ryerson in 1923 and consists of fusion of the subtalar, calcaneocuboid, and talonavicular joints.[268] The operation is designed to provide lateral stability, and it will also correct deformity if the articular surfaces are resected by pattern. In locomotion, the essential motions of the foot and ankle are plantar flexion and dorsiflexion. In the presence of muscle weakness, a triple arthrodesis will stabilize the hindfoot and diminish the functional demand on the remaining active muscles by reducing the number of joints that they control. Triple arthrodesis is described and illustrated in Plate 26–7.

The subtalar and midtarsal joint motions are particularly

Text continued on page 1378

Anterior Transfer of Posterior Tibial Tendon Through Interosseous Membrane

OPERATIVE TECHNIQUE

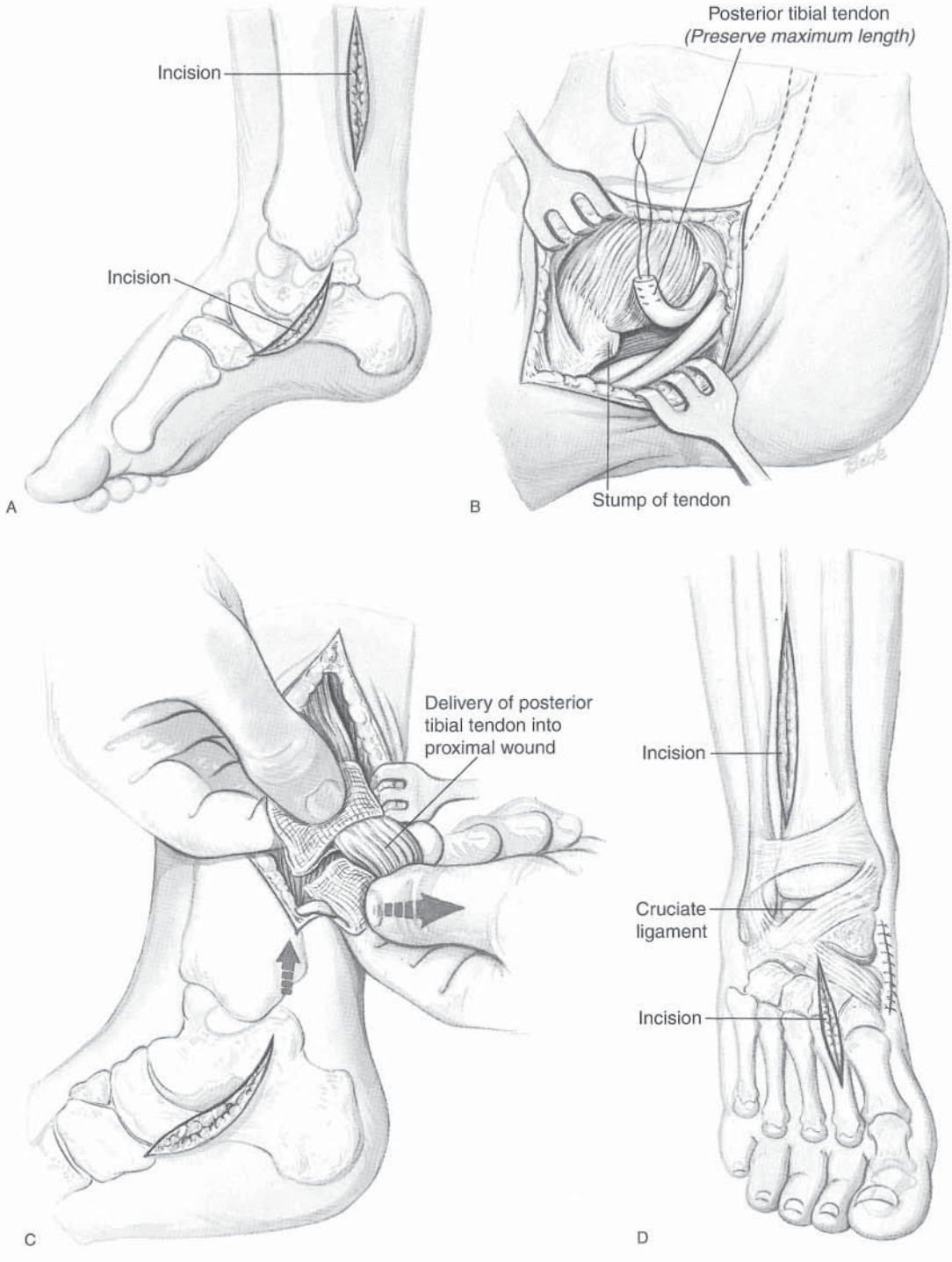
A, A 4-cm long incision is made over the medial aspect of the foot, beginning posterior and immediately distal to the tip of the medial malleolus and extending to the base of the first cuneiform bone. A second longitudinal incision is made 1.5 cm posterior to the subcutaneous medial border of the tibia, beginning at the center of the middle third of the leg and ending 3 cm from the tip of the medial malleolus.

B, The posterior tibial tendon is identified at its insertion and its sheath is divided. The tendon is freed and sectioned at its attachment to the bone, preserving maximal length. The peritenon of the distal 3 cm of the tendon is excised and a 00 silk whip suture is inserted in its distal end.

C, The posterior tibial muscle is identified in the leg incision and its sheath is opened and freed. Traction on the stump in the foot incision will aid in its identification. Moist sponges and the two-hand technique are used to deliver the posterior tibial tendon into the proximal wound. The muscle belly is freed well up the tibia. The operator must be careful to preserve the nerve and blood supply to the posterior tibial muscle.

D, Next, a longitudinal skin incision is made anteriorly, one fingerbreadth lateral to the crest of the tibia, starting at the proximal margin of the cruciate ligament of the ankle and extending 7 cm proximally. Then a 4-cm-long longitudinal incision is made over the dorsum of the foot, centered over the base of the second metatarsal.

PLATE 26–5. Anterior Transfer of Posterior Tibial Tendon Through Interosseous Membrane



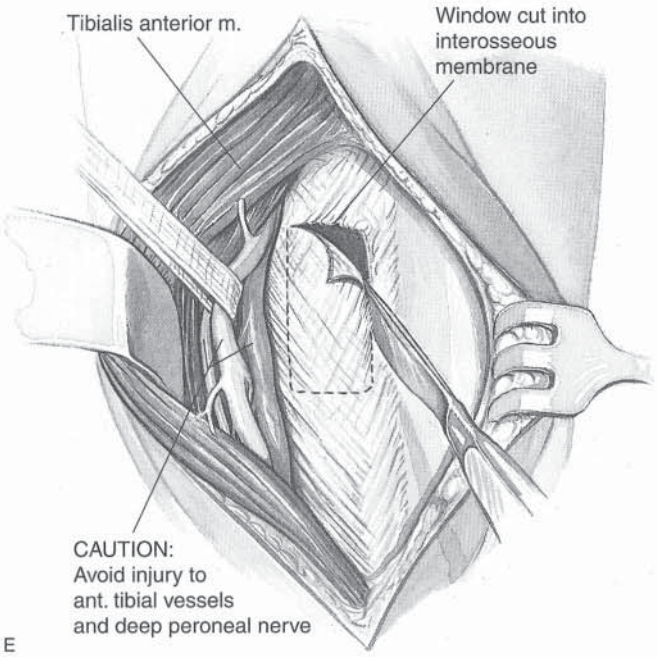
Anterior Transfer of Posterior Tibial Tendon Through Interosseous Membrane *Continued*

E, The anterior tibial muscle is exposed and elevated from the anterolateral surface of the tibia, together with the anterior tibial artery and extensor hallucis longus muscle. It is retracted laterally, exposing the interosseous membrane. Next, a large rectangular window is cut in the interosseous membrane. The surgeon should avoid stripping the periosteum from the tibia or fibula.

F and **G,** Then, with an Ober tendon passer, the posterior tibial tendon is passed through the window in the interosseous membrane from the posterior into the anterior tibial compartment. Care is needed not to twist the tendon or damage its nerve or blood supply. Next, with the aid of an Ober tendon passer, the posterior tibial tendon is passed beneath the cruciate ligament and the extensors and delivered into the wound on the dorsum of the foot. It is anchored to the base of the second metatarsal bone according to the method described in anterior transfer of peroneal tendons (see Plate 26–4). The wounds are closed in layers in the usual manner. A long-leg cast is applied that holds the foot in neutral position at the ankle joint and the knee in 45 degrees of flexion.

The principles of postoperative care are the same as for any tendon transfer.

PLATE 26–5. Anterior Transfer of Posterior Tibial Tendon Through Interosseous Membrane



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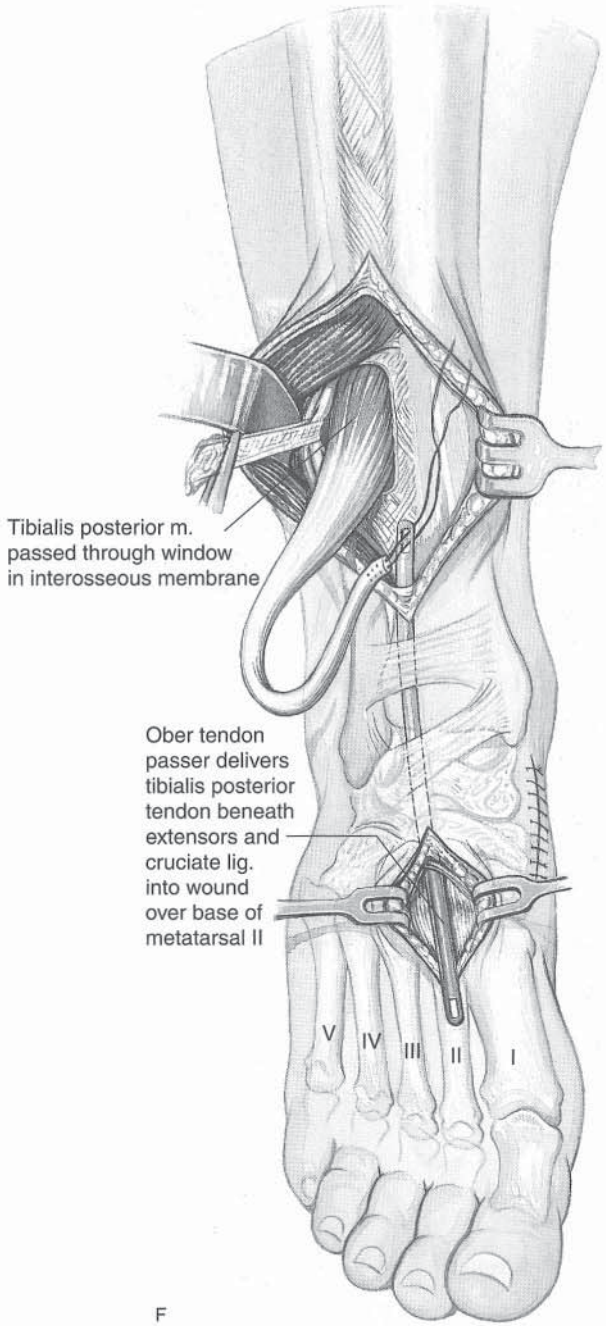

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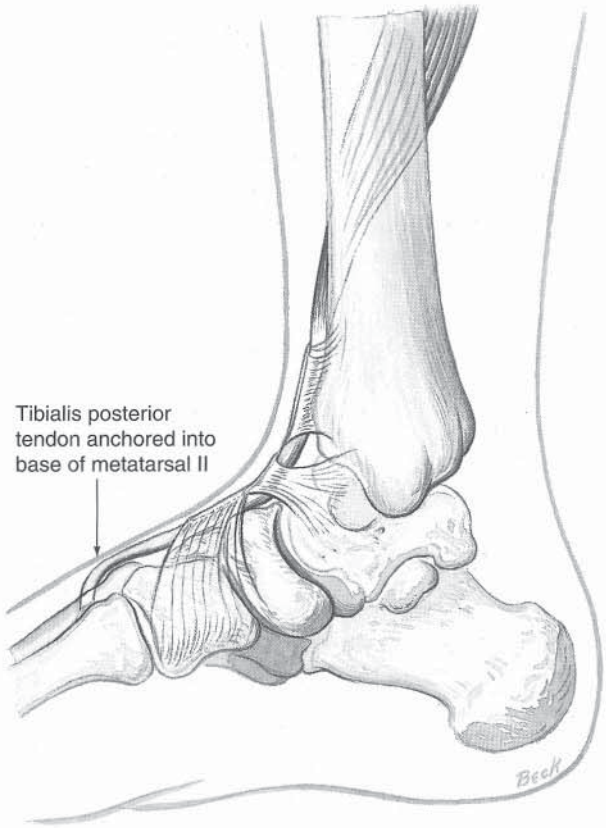

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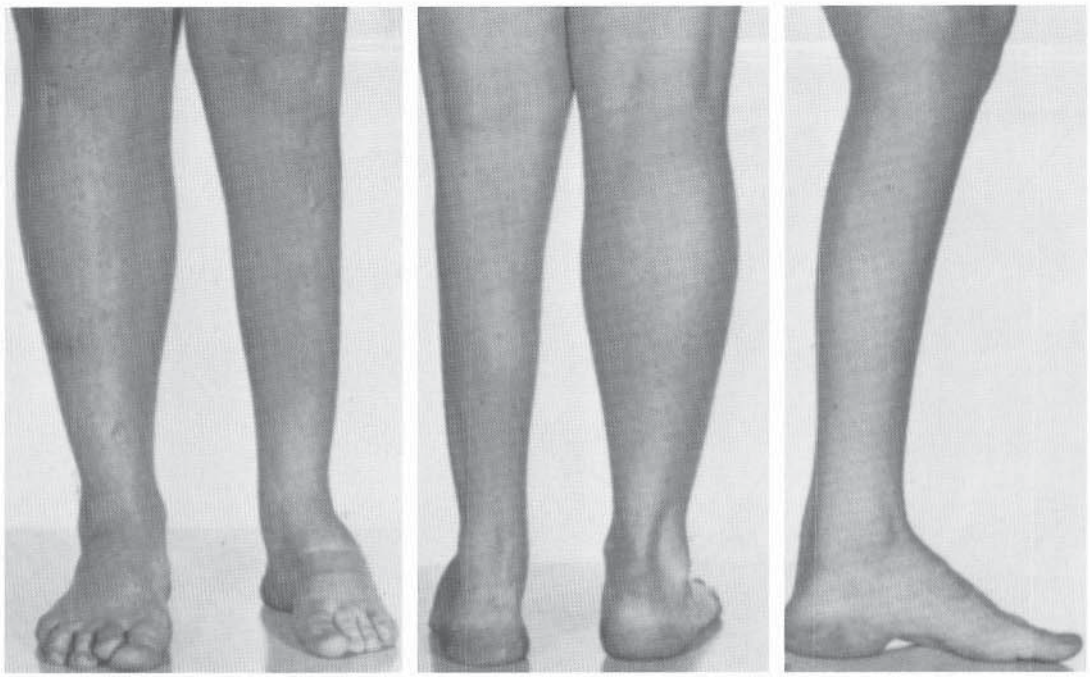


FIGURE 26–18 Calcaneocavus deformity of the left foot and ankle.

important for balance during walking on rough or uneven terrain. The loss of lateral mobility of the hindfoot following triple arthrodesis may result in difficulty in locomotion on an irregular surface.

Triple arthrodesis may exert excessive ligamentous strain on the ankle joint. It is imperative to determine the stability of the body of the talus in the ankle mortise. This is done clinically by testing passive lateral motion of the ankle. If ankle stability is questionable, weightbearing AP radiographs of the ankle with the hindfoot first in forced maximal eversion and abduction and then in forced inversion and adduction are obtained. Normally there is no lateral motion of the body of the talus in the ankle mortise except when the ankle is in marked plantar flexion; then lateral motion may be present in minimal amount. When there is marked instability of the ankle joint, varus or valgus deformity of the hindfoot may recur following triple arthrodesis, and stabilization of the ankle joint may be indicated.

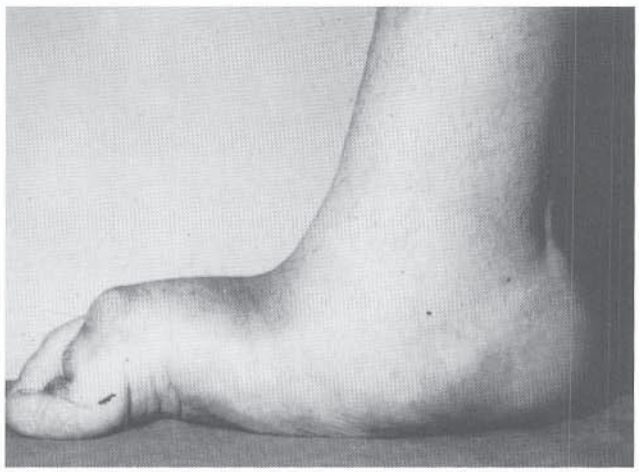


FIGURE 26–19 Dorsal bunion of the left foot. Note the dorsiflexion of the first metatarsal and the plantar flexion of the great toe. (Courtesy of D. W. McKay, M.D.)

Anterior subluxation of the ankle joint may be present in severe equinus deformity; this should be ruled out by obtaining lateral radiographs of the ankle with the foot in maximal plantar flexion and in forced dorsiflexion.

Alignment and weightbearing lines of the lower limb should be carefully studied. The presence of bowleg, knock-knee, or any excessive medial or lateral tibial torsion should be noted. Lateral tibial torsion and genu valgum are common deformities in poliomyelitis. In stance, does the center of gravity of the body fall on the second metatarsal bone? Failure to recognize malalignment of the leg will result in improper positioning of the foot. During surgery, it is mandatory that the knee be draped sterilely in the operative field. The foot should be aligned with the ankle mortise and not with the knee (Fig. 26–24). If there is significant torsional or angular deformity of the leg, it is corrected at a subsequent operation.

The growth of the foot in a young child should not be disturbed. The tarsal bones grow concentrically at their periphery, and resection of their articular surfaces will inhibit their growth. Triple arthrodesis should be deferred until the foot has achieved skeletal maturity, which in girls is 10 to 12 years, in boys 12 to 14 years.

The osseous deformity of the foot should be carefully analyzed on the preoperative radiographs. These images should include AP and mediolateral weightbearing views of the foot and ankle. It is important to obtain the radiographs with the foot held in the positions of maximum correction. Tracings of the foot are made on x-ray negative films. The foot and ankle are divided into three segments, according to function: (1) the talus with the tibia and ankle joint, (2)

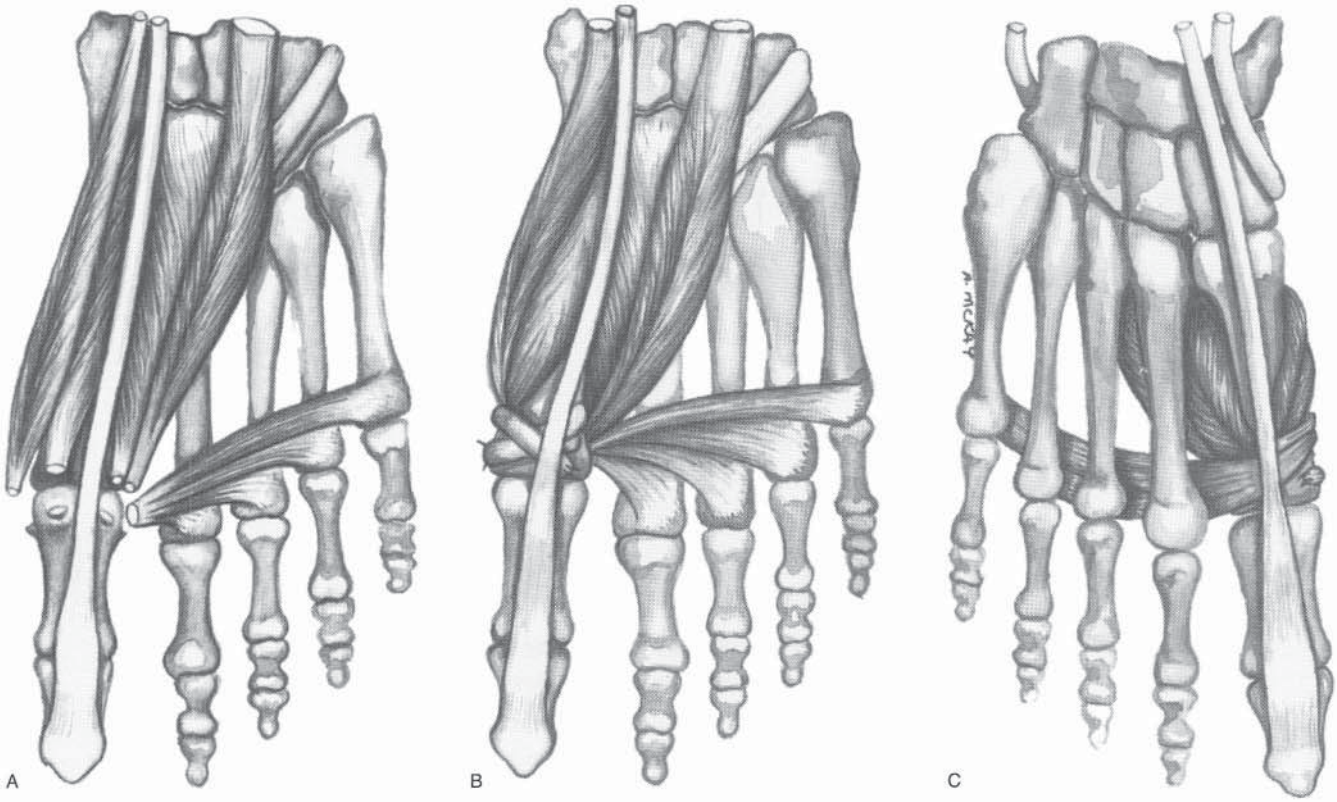


FIGURE 26–20 McKay's technique for correction of dorsal bunion. **A,** Plantar view of right foot showing section of abductor hallucis, adductor hallucis, and flexor hallucis brevis from the base of the proximal phalanx. **B,** Tendinous portions of these muscles transferred to the neck of the first metatarsal, as seen from the plantar aspect. **C,** Dorsal view showing tendons transferred to the dorsum of the neck of the first metatarsal. (Courtesy of D. W. McKay, M.D.)

the os calcis, and (3) the tarsal bones, the joints distal to the midtarsal joint, and the metatarsals and phalanges. The talus is the only tarsal bone that transmits the entire body weight; thus, the importance of double-checking the stability of the body of the talus in the ankle mortise cannot be overemphasized.

The pattern of osteotomies and the plane of resection of the articular surfaces of each joint should be carefully and precisely planned. It is best to draw these lines on tracings of the preoperative lateral radiographs of the foot.

In the correction of varus deformity, a wedge of bone with its base facing laterally is resected from the talonavicular and calcaneocuboid joints (Fig. 26–25). Lateral displacement of the forefoot is often prevented by the "beak" of the navicular bone, which projects posteriorly along the medial side of the head of the talus. It is important to excise this "beak" flush with the main body of the navicular—through a separate incision if necessary. The planes of osteotomies of the talonavicular and calcaneocuboid joints should be parallel to each other in the vertical axis to achieve close apposition of bones. To correct varus deformity of the heel, a laterally based wedge is resected from the subtalar joint. Most of the bone should be removed from the superior surface of the calcaneus. Only a minimal amount of bone should be excised from the talus. A slight valgus position of the heel will provide stability; however, the hindfoot should not be placed in more than 5 to 10 degrees of eversion, as it will cause difficulty in the proper wearing of shoes and is not cosmetically satisfactory. A varus position of the heel should not be accepted.

Valgus deformity of the foot is corrected by excision of a medially based wedge from the midtarsal area and another wedge, also based medially, from the subtalar region. The use of a laminectomy spreader in the subtalar joint will adequately expose the medial side of the hindfoot. Great care should be taken not to injure the posterior tibial nerves and artery, which lie adjacent and superficial to the flexor hallucis longus tendon. In the valgus foot, the os calcis is everted and the head of the talus is plantar flexed over the medial aspect of the foot. The common tendency is to excise a large wedge in order to reduce the calcaneus medially beneath the talus. This should be avoided, as it will reduce the height of the hindfoot and lower the malleoli, resulting in a wide ankle contour and extreme difficulty in fitting shoes. When correcting severe valgus, varus, or calcaneus deformity of the foot, it is best to add bone graft wedges rather than to excise too much bone. Resection of excessive bone from the talus and navicular may also cause avascular necrosis of these tarsal bones with subsequent degenerative arthritis of the ankle and pseudarthrosis of the talonavicular joint.

el-Batouty and associates have described a modified technique of triple arthrodesis to correct paralytic pes valgus.[2a]

For restoration of alignment of the calcaneus foot, a wedge of bone based posteriorly is resected from the subtalar

Text continued on page 1386

Posterior Tendon Transfer to Os Calcis for Correction of Calcaneus Deformity (Green and Grice)

OPERATIVE TECHNIQUE

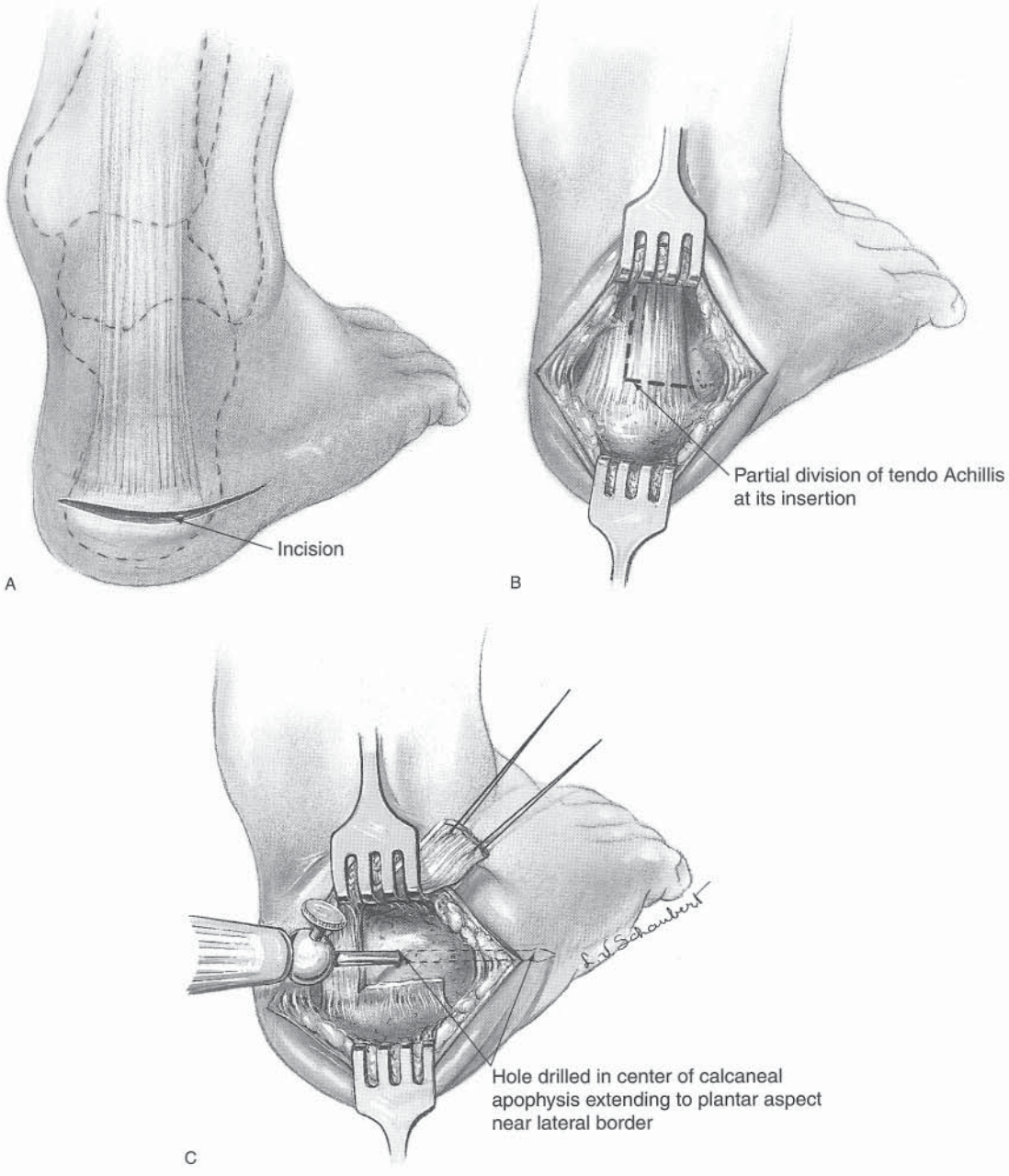
It is best to place the patient in the prone position to facilitate the surgical exposure of the heel. The posterior tibial and peroneus longus and brevis tendons are divided distally at their insertion and delivered into the proximal wound. When the flexor hallucis longus tendon is to be transferred, its distal portion is sutured to the flexor hallucis brevis muscle. The anterior tibial tendon is delivered into the calf and heel through the interosseous route.

A, A 5-cm-long posterior transverse incision is made around the heel along one of the skin creases in the part that neither presses the shoe nor touches the ground.

B, The skin and subcutaneous flaps are undercut and reflected, exposing the os calcis and the insertion of the tendo calcaneus. An L-shaped cut is made in the lateral two-thirds of the insertion of the tendo calcaneus. The divided portion is reflected proximally, exposing the apophysis of the os calcis.

C, Next, with a $\frac{9}{64}$-inch drill, a hole is made through the calcaneus, beginning in the center of the apophysis and coming out laterally at its plantar aspect. With a diamond head hand drill and curet, the hole is enlarged to receive all the transferred tendons.

PLATE 26–6. Posterior Tendon Transfer to Os Calcis for Correction of Calcaneus Deformity (Green and Grice)

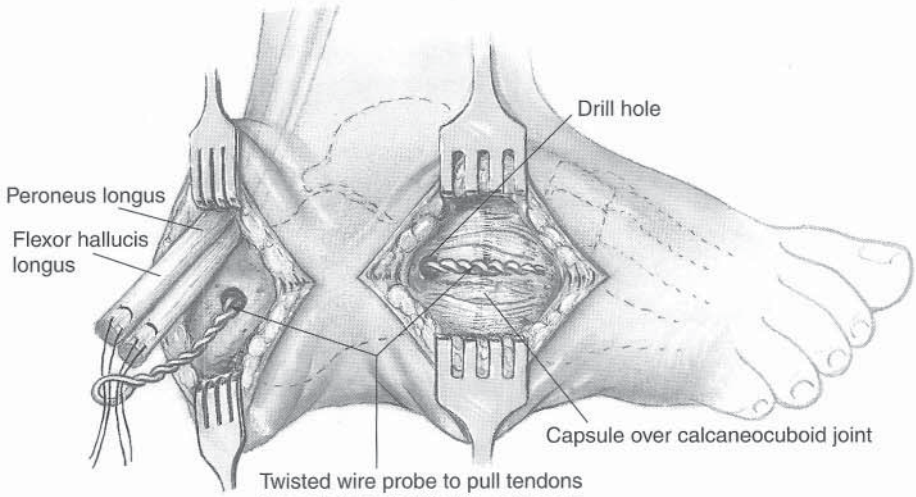


Posterior Tendon Transfer to Os Calcis for Correction of Calcaneus Deformity (Green and Grice) *Continued*

D, Through a lateral incision, the intermuscular septum is widely divided between the lateral and posterior compartments. An Ober tendon transfer is inserted through the wound and directed anterior to the tendo calcaneus into the transverse incision over the os calcis. The threads of the whip sutures at the ends of the peroneal tendons are passed through the hole in the tendon passer and the tendons are delivered at the heel. The posterior tibial tendon is delivered at the heel by a similar route, through an incision in the intermuscular septum between the medial and posterior compartments and anterior to the tendo calcaneus. Next, with a twisted wire probe, the tendons are inserted in the hole and pulled through the tunnel in the calcaneus.

E, At their point of exit on the lateral aspect of the calcaneus the tendons are sutured to the periosteum and ligamentous tissues. The tendons are sutured under enough tension to hold the foot in 15 degrees of equinus when the remaining ankle dorsiflexors are fair in motor strength, and 30 degrees of equinus if they are good or normal. The tendons are sutured to each other and to the periosteum of the apophysis of the calcaneus at the posterior end of the tunnel.

PLATE 26–6. Posterior Tendon Transfer to Os Calcis for Correction of Calcaneus Deformity (Green and Grice)



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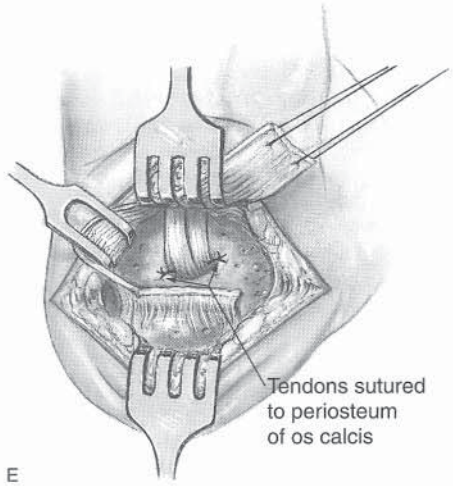


E

Posterior Tendon Transfer to Os Calcis for Correction of Calcaneus Deformity (Green and Grice) *Continued*

F and **G,** The divided portion of the tendo calcaneus is resutured in its original position posterior to the transferred tendons.

The wounds are closed and a long-leg cast is applied to hold the knee in 45 to 60 degrees of flexion and the hindfoot in 15 to 30 degrees equinus, but the forefoot in neutral position. Cavus deformity of the forefoot should be avoided.

POSTOPERATIVE CARE

Three to 4 weeks after surgery the solid cast is removed and a new above-knee bivalved cast is made to protect the limb at all times when exercises are not being performed. It is imperative to prevent forced dorsiflexion of the ankle and stretching of the transferred tendons.

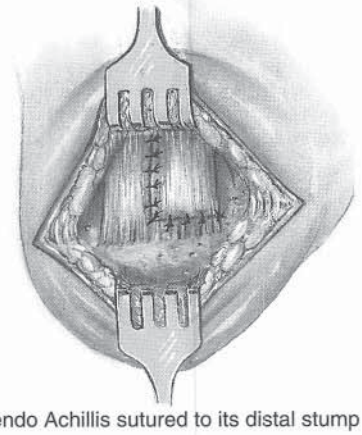
Exercises are first performed in the side-lying position with gravity eliminated, and then in the prone position against gravity. In order to teach the patient the new action of the transferred muscle, the patient is asked to move the foot in the direction of a component of the original action of the muscle and then to plantar flex the foot. For example, when the peroneals are transferred, the patient is asked to evert and plantar flex the foot, or, when the anterior tibial is transferred, to invert and plantar flex the foot. Soon, under supervision, guided dorsiflexion of the foot is performed along with plantar flexion. It is important to develop reciprocal motion and motor strength of agonistic and antagonistic muscles. Weightbearing is not allowed. Ambulation is permitted in the above-knee bivalved cast with crutches.

In about 4 to 6 weeks, when the transferred tendons are fair in motor strength, the patient is allowed to stand on both feet. The heel of the foot that was operated on rests on a 3-cm-thick block to prevent stretching of the transferred tendons. Bearing partial weight on the foot, the patient should rise up on tiptoes while holding on to a table with the hands or using two crutches.

When the transplant functions effectively on tiptoe standing, walking with crutches is begun with three-point gait and partial weightbearing on the affected limb. The heel of the shoe is elevated with a 1- to 1.5-cm lift that tapers in front (toward the toes). Walking periods are gradually increased. When the transplant works effectively in gait and take-off has been developed in walking, standing-tiptoe rising exercises are started without the support of crutches. The knee should not be flexed and the patient should not lean forward while rising up on the toes at least three times. This may take a long time (as much as a year or more), but it is a very important phase of postoperative management.

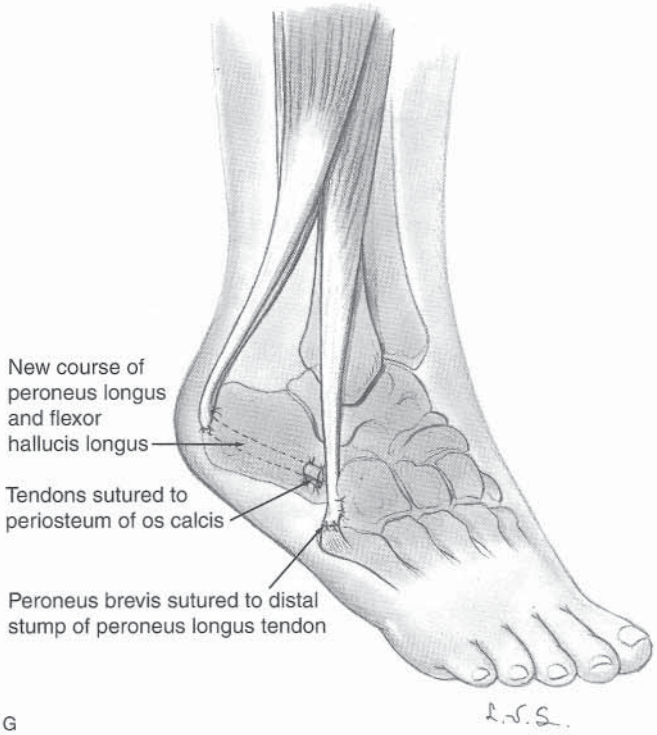
A plantar-flexion spring orthosis or an orthosis with a posterior elastic is worn when the patient is uncooperative in the use of crutches or when muscular control of the knee and hip is poor because of extensive paralysis. A stop at the ankle prevents dorsiflexion of the ankle beyond neutral position.

PLATE 26–6. Posterior Tendon Transfer to Os Calcis for Correction of Calcaneus Deformity (Green and Grice)



Tendo Achillis sutured to its distal stump

F



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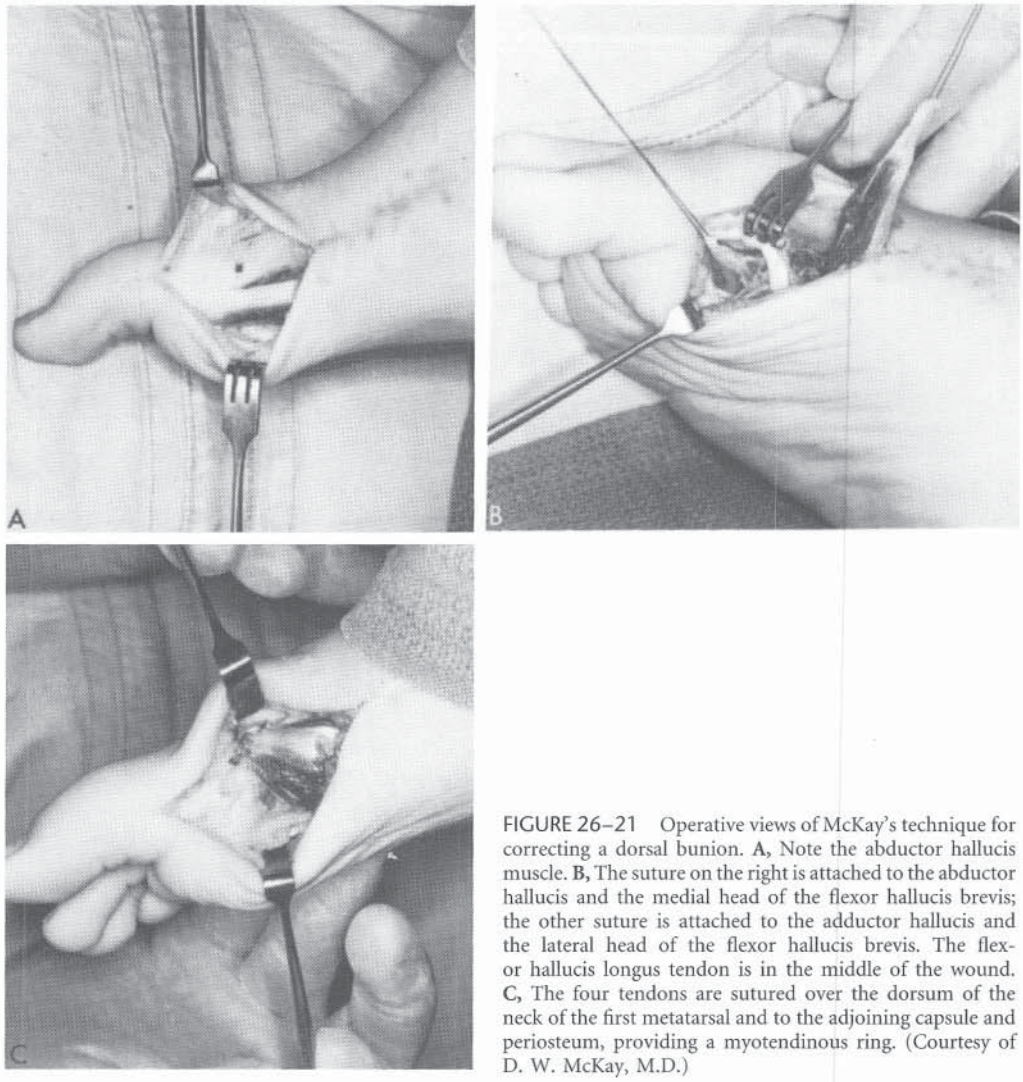


FIGURE 26–21 Operative views of McKay's technique for correcting a dorsal bunion. **A,** Note the abductor hallucis muscle. **B,** The suture on the right is attached to the abductor hallucis and the medial head of the flexor hallucis brevis; the other suture is attached to the adductor hallucis and the lateral head of the flexor hallucis brevis. The flexor hallucis longus tendon is in the middle of the wound. **C,** The four tendons are sutured over the dorsum of the neck of the first metatarsal and to the adjoining capsule and periosteum, providing a myotendinous ring. (Courtesy of D. W. McKay, M.D.)

joint (Fig. 26–26). Often there is associated cavus deformity, which is corrected by excising a dorsally based wedge from the talonavicular and calcaneocuboid joints. It is imperative to displace the os calcis posteriorly to provide a longer lever arm. When contracted, the anterior capsule of the ankle joint is stretched out preoperatively by passive manipulation and corrective casts. Release of soft tissue contracture may be indicated when the contracture is very fixed. It is imperative to obtain normal range of plantar flexion of the ankle.

In severe talipes calcaneus, the bony deformity and soft tissue contracture are rigid, fixing the talus and os calcis in marked dorsiflexion. The associated cavus deformity will be marked, with severe contracture of the plantar fascia and osseous changes in the midtarsal bones. Correction of the deformity by wedge resections will result in an appreciable reduction in the height and length of the foot.

Plantar fasciotomy is performed first. The anterior capsule of the ankle joint is released through an anterolateral approach. Next, the articular surfaces of the subtalar and talonavicular and calcaneocuboid joints are minimally resected, exposing raw cancellous bone. The calcaneus deformity is corrected by inserting an anteriorly based wedge of bone graft in the subtalar joint. Forefoot equinus deformity can be corrected by excising a wedge of bone based dorsally from the talonavicular and calcaneocuboid joints. The author frequently postpones surgical correction of the cavus deformity until solid healing of the triple arthrodesis has taken place. During application of the above-knee cast, however, it is important to immobilize the heel in moderate plantar flexion and the forefoot in maximal dorsiflexion. The common pitfall is to hold the forefoot in equinus position, permitting the cavus deformity to increase. The metatarsal heads should be well padded to prevent skin slough. Three to four months later the metatarsals are osteotomized at their base and elevated into dorsiflexion, correcting the forefoot equinus deformity. In this way, some degree of mobility of the naviculocuneiform and cuneiform metatarsal joints is preserved.

Talectomy will correct the severe calcaneus deformity.[161,206,317,335,336] However, it reduces the height of the foot, lowers the malleoli, and causes great difficulty in fitting shoes. This is particularly disturbing to women. Fibroarthrosis and degenerative arthritis of the ankle joint will often develop in later years. For these reasons, astragalectomy is not recommended.

In the classic triple arthrodesis, it is difficult to displace

the os calcis backward. Dunn described a method of excising the navicular and part of the head and neck of the talus to permit posterior displacement of the calcaneus.[88,89] Hoke had previously resected the navicular and head and neck of the talus; after subtalar joint resection and posterior displacement of the foot, he recommended reshaping and reimplanting the head and neck of the talus.[160] The Hoke and Dunn procedures, however, shorten the foot and increase the likelihood of pseudarthrosis of the talonavicular joint. For these reasons, the author prefers correction of calcaneus deformity by bone graft wedges.

In correction of pes equinus, fixed contracture of the posterior capsule of the ankle and subtalar joints and the triceps surae muscle must be corrected preoperatively. As stated previously, function of the gastrocnemius-soleus muscles should be maintained as much as possible. Wedging casts are tried first, followed by posterior capsulotomy and skeletal traction through the os calcis. In severe equinus deformity, limited heel cord lengthening may have to be performed, but it is preferable to leave some tightness of the triceps surae. It is imperative that the foot be dorsiflexed to neutral position; otherwise a rocker-bottom deformity will result. In patients with cerebral palsy, the author maintains reduction with a large Kirschner wire. One pin is placed through the os calcis into the talus and across the ankle joint into the tibia, while the other transfixes the talonavicular joint. When equinus posture is due simply to drop foot and the foot can be passively dorsiflexed beyond neutral position, it is preferable to perform tendon transfer anteriorly to provide power of active dorsiflexion. If adequate muscles are not available for anterior transfer, the triple arthrodesis may be modified to prevent the foot from dropping down into plantar flexion. Lambrinudi in 1927 described a method of triple arthrodesis in which a wedge of bone is excised from the plantar aspect of the head and neck of the talus and the distal sharp margin of the body of the talus is inserted into a prepared trough in the navicular. In this way the talus is locked in equinus position at the ankle joint while the rest of the foot is maintained in the desired degree of dorsiflexion.[187]

The Lambrinudi operation is not recommended by the author, as his experience with it has been unsatisfactory. For adequate correction of equinus deformity, too much bone has to be resected from the talus, with consequent development of avascular necrosis of the talus, talonavicular pseudarthrosis, flattening of the superior surface of the talus, and painful arthritis of the ankle.[100,144,186,209,213,250]

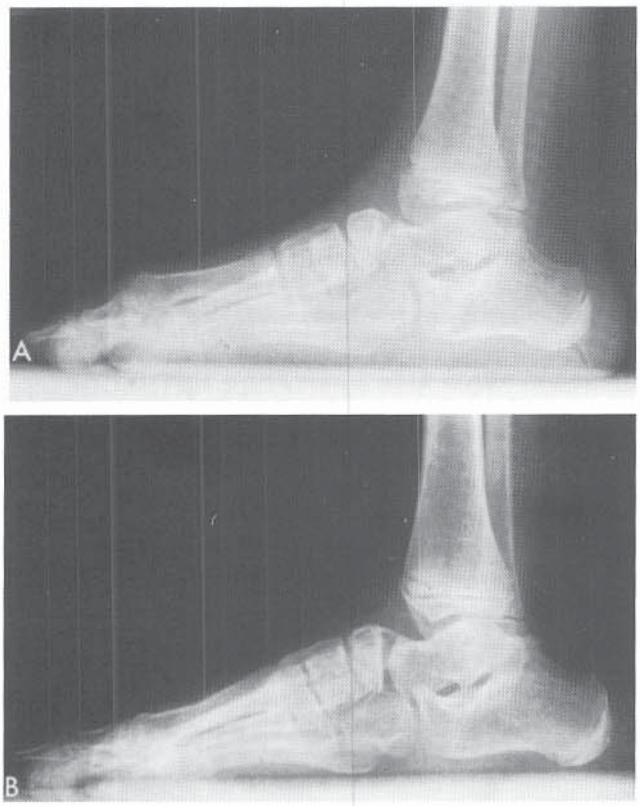


FIGURE 26–22 McKay's technique for correction of dorsal bunion. **A,** Preoperative lateral radiograph of the foot, showing the deformity. **B,** Postoperative lateral radiograph of the foot. Note the excellent correction. (Courtesy of D. W. McKay, M.D.)

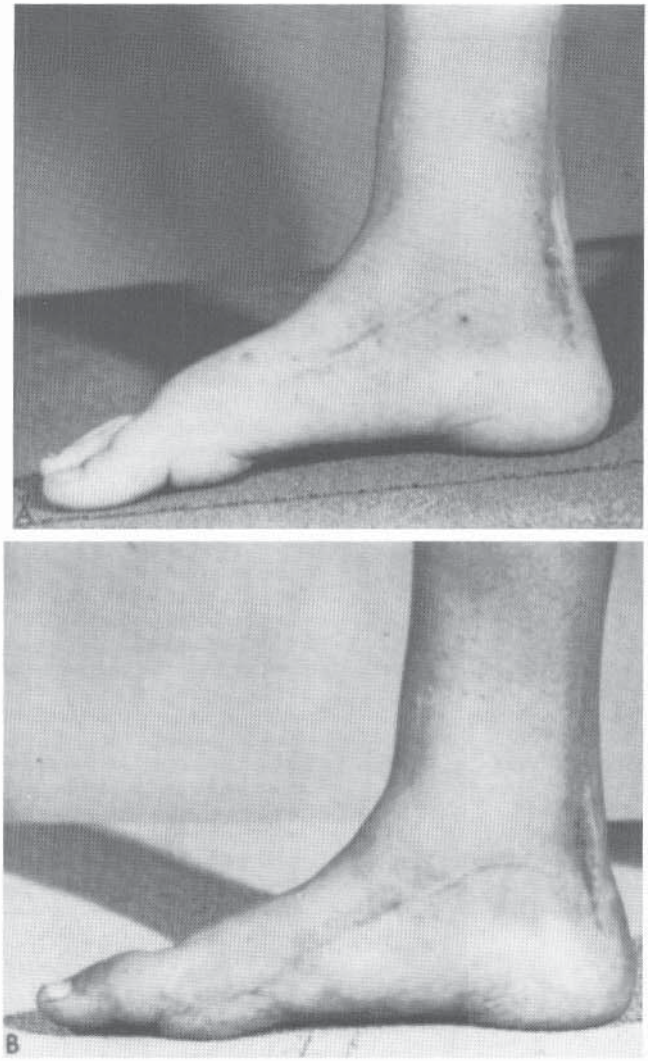


FIGURE 26–23 Dorsal bunion corrected by McKay's technique. **A,** Preoperative photograph. The dorsal bunion developed after a cuneiform open-wedge osteotomy (Fowler's procedure) for cavovarus foot. **B,** Postoperative lateral view of foot showing correction. (Courtesy of D. W. McKay, M.D.)

TABLE 26–2 **History of Stabilizing Operations of the Foot and Ankle**

1879	Albert	Tibiotarsal or ankle joint arthrodesis
1879	Von Lesser	Tibiotarsal or ankle joint arthrodesis
1884	Samster	Ankle and subtalar joint arthrodesis
1901	Whitman	Talectomy and posterior displacement of the foot
1905	Nieny	Talocalcaneal and talocalcaneonavicular or subtalar arthrodesis
1908	Jones	Talocalcaneal and talocalcaneonavicular or subtalar arthrodesis
1908	Goldthwait	Supratalar and infratalar arthrodesis
1911	Lorthioir	Pantalar arthrodesis (temporary removal of the talus)
1911	Ombredanne	Surgical approach for exposure of the subtalar and midtarsal joints
1912	Soule	Talonavicular arthrodesis
1912	Soule	Talonavicular and subtalar arthrodesis
1913	Davis	Subtalar arthrodesis (transverse horizontal section of the tarsus) with posterior displacement of the foot
1915	Albee	Talonavicular arthrodesis with bone graft peg
1916	Davis	Subtalar or calcaneotalar and calcaneotalonavicular arthrodesis
1919	Dunn	Midtarsal tarsectomy and calcaneotalar arthrodesis
1920	Toupet	Posterior bone check
1921	Hoke	Calcaneotalonavicular arthrodesis resection, reshaping and reimplantation of head and neck of the talus, and posterior displacement of the foot.
1922	Dunn	Excision of navicular bone, calcaneotalocuneiform and calcaneocuboid arthrodesis with posterior displacement of the foot
1922	Putti	Anterior bone check
1922	Steindler	Pantalar arthrodesis (talus not temporarily removed)
1923	Ryerson	Triple (subtalar and calcaneocuboid) arthrodesis
1923	Ryerson	Lateral arthrodesis (cuneonavicular, first and fifth tarsometatarsal arthrodesis)
1923	Campbell	Posterior bone block
1925	Smith and von Lackum	Calcaneotalonavicular and calcaneocuboid arthrodesis, excision of head and neck of talus with posterior displacement of the foot
1927	Lambrinudi	Resection of wedge of bone from plantar aspect of head and neck of talus to lock the talus in equinus position at the ankle while the rest of the foot is in the desired degree of dorsiflexion
1933	Brewster	Calcaneonaviculocuneiform arthrodesis, excision of head and neck of the talus, posterior displacement of the foot and countersinking of the body of the talus in the os calcis
1935	Girard	Arthrodesis of subtalar and calcaneocuboid joints; shortening of the neck of the talus; posterior displacement of foot; construction of ankle joint mortise
1952	Grice	Extra-articular arthrodesis of subtalar joint
1963	Chuinard and Peterson	Distraction-compression bone graft arthrodesis of the ankle

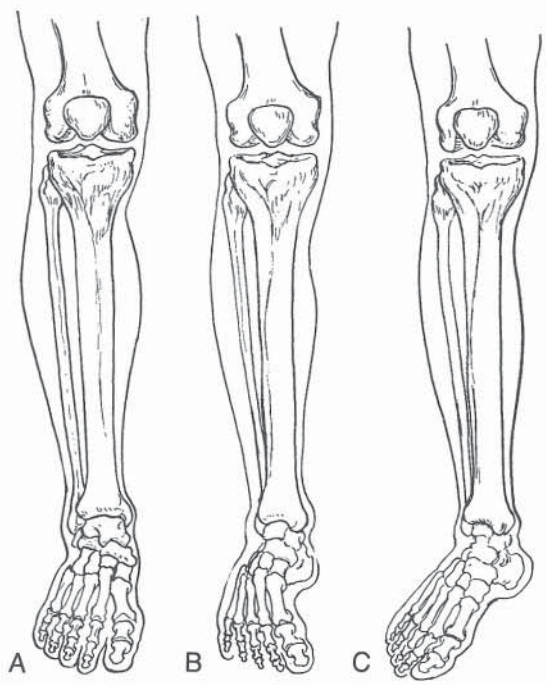


FIGURE 26–24 Alignment of the foot. **A,** Normal foot, ankle, and knee alignment without tibial torsion. **B,** *Incorrect.* Foot is aligned with the knee, not the ankle, in the presence of external tibial torsion. **C,** *Correct.* Foot is aligned with the ankle joint in the presence of external tibial torsion. (From Patterson RL, Parrish FF, Hathaway EN: Stabilizing operations on the foot. J Bone Joint Surg 1950;32-A:3.)

EXTRA-ARTICULAR SUBTALAR ARTHRODESIS

Grice, at the suggestion of William T. Green, developed a method of fusion of the subtalar joint by insertion of autogenous bone grafts in the sinus tarsi in the lateral aspect of the foot for correction of paralytic pes valgus and restoration of the height of the longitudinal arch.[121,123] Any interference with subsequent normal growth of the foot is minimal, at most, because the procedure is extra-articular. The operative technique is described and illustrated in Plate 26–8.

ANKLE FUSION AND PANTALAR ARTHRODESIS

When surgical reconstruction is being considered for a flail foot and ankle, the relationship of the foot and ankle to the lower limb as a whole should be carefully assessed, since there is often associated paralysis of the muscles throughout the lower limb. A variety of techniques have been described in the literature.*

Pantalar arthrodesis is surgical fusion of the joints around the talus—the ankle, subtalar, and talonavicular joints; the calcaneocuboid joint (which is not an articulation of the talus) is also included in the stabilization, thus making the procedure a combination of triple arthrodesis and ankle fusion.

Lorthioir, as he originally reported the procedure in 1911, extirpated and replaced the talus as an autogenous bone graft.[200] In 1923, Steindler advised against the temporary

*See references 4, 14, 27, 49, 57, 114, 138, 165, 181, 198, 250.

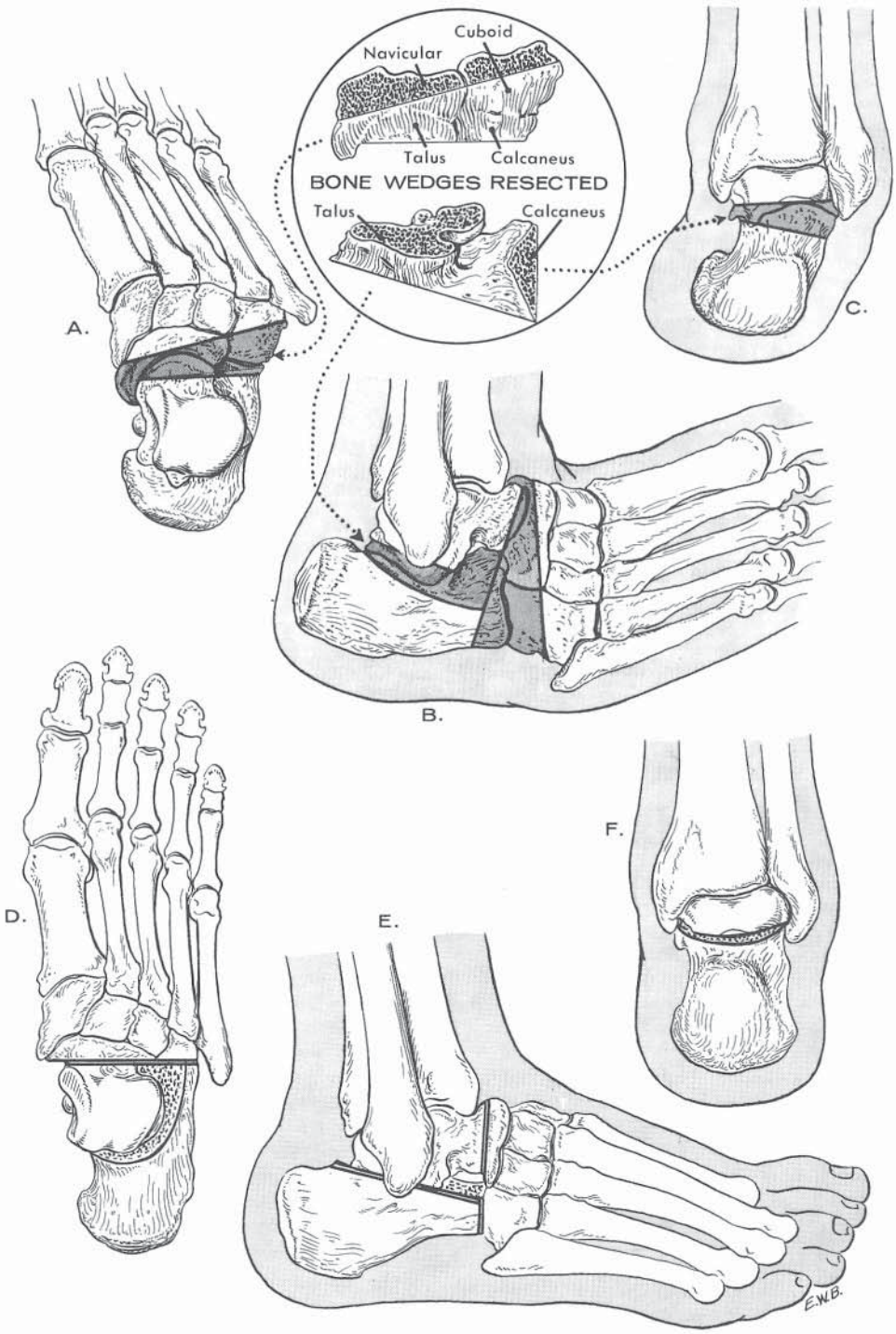


FIGURE 26–25 Wedges of bone to be resected for correction of pes varus. **A** to **C,** Three views of the varus deformity. *Shaded areas* show amount of bone wedges to be resected. **D** to **F,** Corrected positions of the bones postoperatively.

removal of the talus from the wound because of the danger of avascular necrosis; he also included the calcaneocuboid joint in the fusion to provide stability and to maintain correction.[311]

When the muscles below the knee are paralyzed, pantalar arthrodesis will provide stability to the ankle and hindfoot, thus eliminating the need for orthotic support, provided the gluteus maximus is of adequate strength and the knee is stable. Extensor strength of the knee is desirable but not imperative. When the quadriceps muscle is paralyzed, stability of the knee joint is provided by shifting the center of gravity of the body forward anterior to the plane of the knee joint. To lock the knee in extension, the tibia should not be allowed to come forward through a dorsiflexion movement of the ankle, either by a strong triceps surae muscle or by a fixed equinus ankle joint. A good gluteus maximus muscle will transmit push-off power to the ball of the foot when the ankle is rigid and the knee is locked in extension.

Position of ankle fusion is an important consideration. In arthrodesis of the ankle, excessive plantar flexion to stabilize the knee in extension during the stance phase of gait or to

Text continued on page 1394

Triple Arthrodesis

OPERATIVE TECHNIQUE

A pneumatic tourniquet is placed on the proximal thigh, and the patient is positioned semilaterally with a large sandbag under the hip on the affected side.

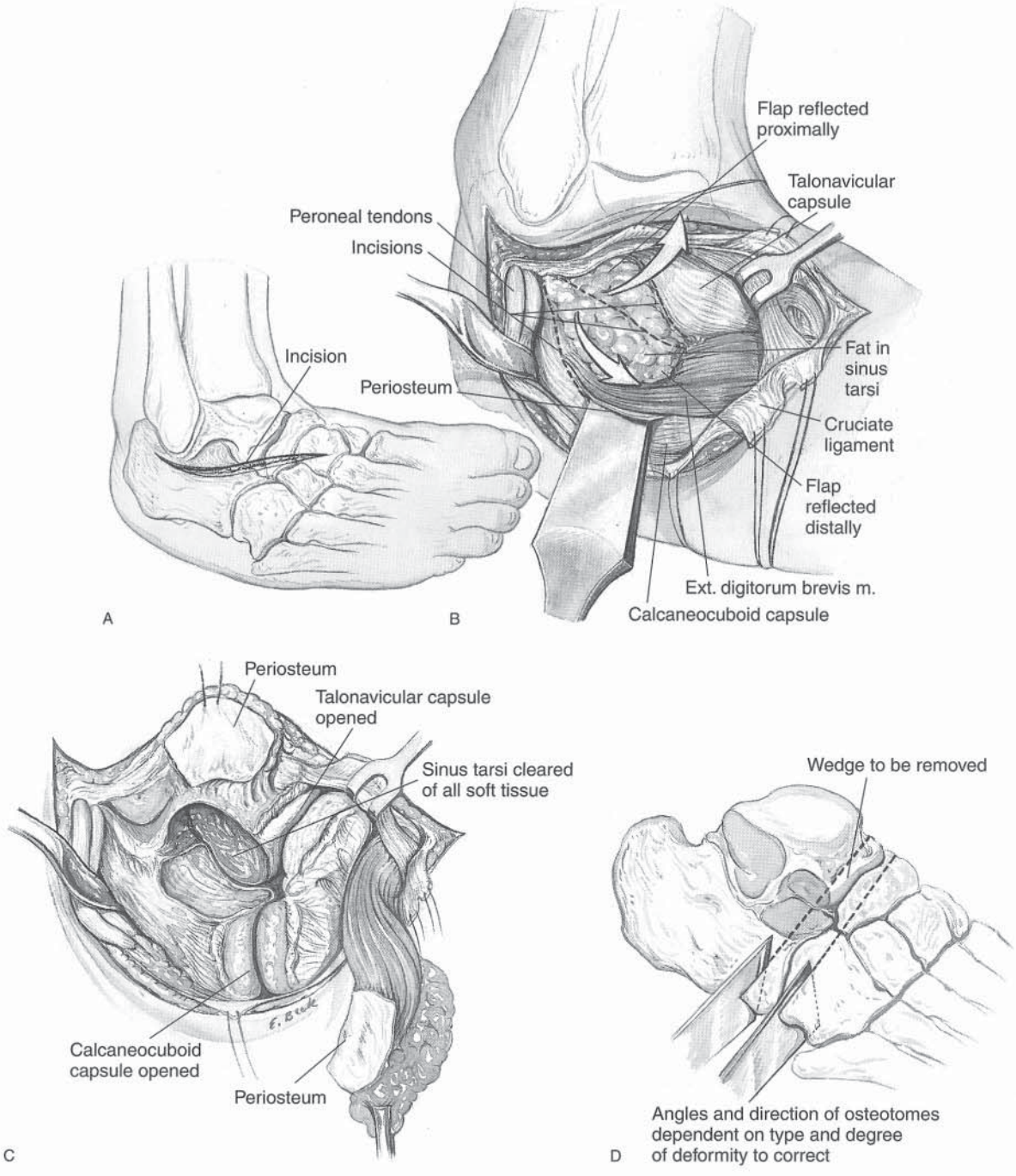
A, A curvilinear incision is made, centered over the sinus tarsi. It starts one fingerbreadth distal and posterior to the tip of the lateral malleolus and extends anteriorly and distally to the base of the second metatarsal bone.

B, Skin flaps should not be developed. The incision is carried to the floor of the sinus tarsi. By sharp dissection, with scalpel and periosteal elevator, the periosteum of the calcaneus, the adipose tissue contents of the sinus tarsi, and the tendinous origin of the exterior digitorum brevis are elevated in one mass from the calcaneus and lateral aspect of the neck of the talus and retracted distally. It is essential to provide a viable soft tissue pedicle to obliterate the dead space remaining at the end of the operation.

Next, an incision is made superiorly over the periosteum of the talus, and the head and neck of the talus are carefully exposed. The upper flap of the skin, subcutaneous tissue, and periosteum should be kept as thick as possible to avoid necrosis. Traction sutures are placed on the periosteum. At no time are the skin edges to be retracted. It is not necessary to divide the peroneal tendons or their sheaths. By subperiosteal dissection, the peroneal tendons are retracted posteriorly to expose the subtalar joint.

C and **D,** The capsules of the calcaneocuboid, talonavicular, and subtalar joints are incised. These joints are opened and their cartilaginous surfaces clearly visualized by turning the foot into varus position. A laminar spreader placed in the sinus tarsi will aid in exposure of the posterior subtalar joint. Before excision of articular cartilaginous surfaces, the surgeon should review the deformity of the foot and decide on the wedges of bone to be removed to correct the deformity. Circulation of the talus and the complications of avascular necrosis of the talus and arthritis of the ankle following triple arthrodesis should always be kept in mind. The height of the foot is another consideration. A low lateral malleolus will cause difficulty with wearing shoes. At times, it is best to add a bone graft rather than resect wedges of bone. With a sharp osteotome, the cartilaginous surfaces of the calcaneocuboid joint are excised. Next, the articular cartilage surface of the talonavicular joint is exposed, the plane of osteotomy being perpendicular to the long axis of the neck of the talus and parallel to the calcaneocuboid joint. When the beak of the navicular is unduly prominent medially, or when, in a varus foot, one cannot obtain adequate exposure of the talonavicular joint without excessive retraction, a second dorsomedial incision may be used to expose the talonavicular joint.

PLATE 26–7. Triple Arthrodesis

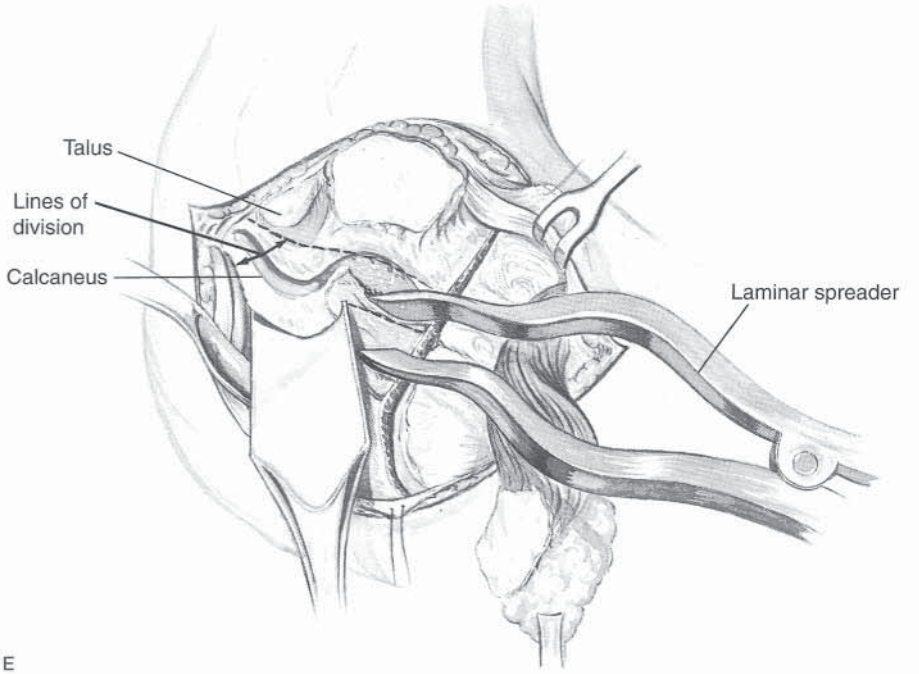


Triple Arthrodesis *Continued*

E to **H,** With a laminar spreader in the sinus tarsi, the subtalar joint is widely exposed and the cartilage of the anterior and posterior joints is excised. The surgeon should keep in mind the neurovascular structures behind the medial malleolus. The wedges of bone that must be removed to correct the deformity are excised in one mass with the articular cartilage. It is of great help to leave the osteotome used on the opposing articular surface in place and held steady by the assistant as a second osteotome or gouge is used to take contiguous cartilage and bone. The divided articular surfaces of the joints to be arthrodesed are "fish-scaled" for maximum raw cancellous bony contact.

The skin is closed with interrupted sutures. A well-molded long-leg cast is applied, holding the foot in the desired position. The author has not found necessary and does not recommend fixation of the joints by staples. In foot stabilization in children with cerebral palsy, especially in the severely athetoid or spastic, secure criss-cross Kirschner wires are used to maintain position. These are removed in 6 to 8 weeks.

PLATE 26–7. Triple Arthrodesis



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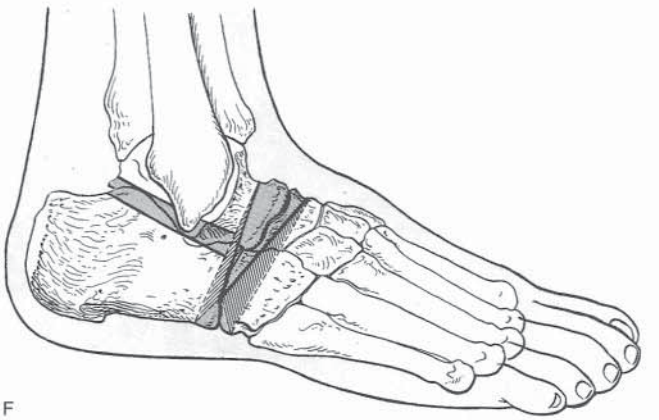

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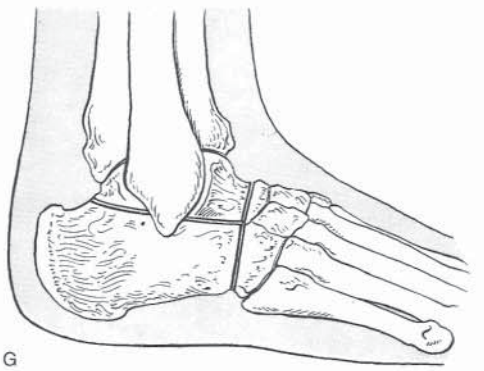

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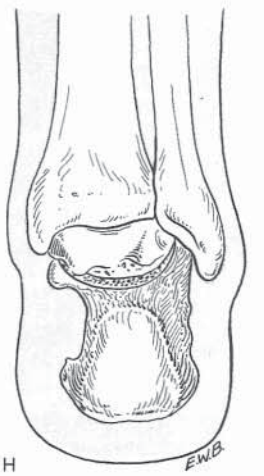

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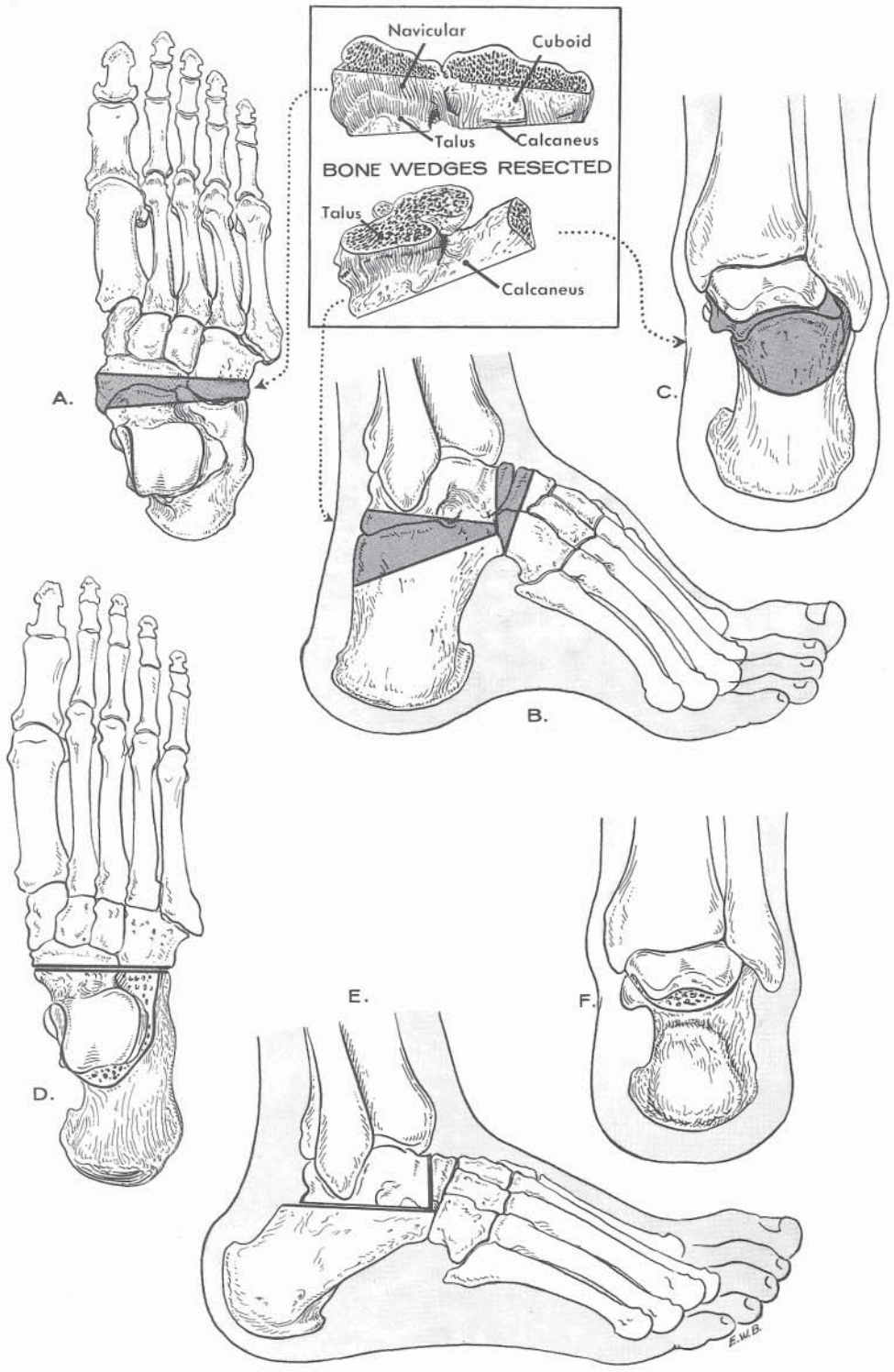


FIGURE 26–26 Wedges of bone to be removed for correction of calcaneocavus deformity. **A** to **C,** Dorsal, lateral, and posterior views of the deformity. *Shaded areas* indicate amount of bone removed. **D** to **F,** Positions of the bones after correction of the deformity by triple arthrodesis. Note the posterior displacement of the hindfoot.

compensate for a short limb will result in increased pressure on the metatarsal heads. Callosities will form, and eventually the skin will ulcerate. Consequently, in later adult life, pain in the forefoot will be a constant complaint. Unequal heel heights are another cause of dissatisfaction; often patients would rather accept shortening and a full-sole build-up. It is imperative that the position of ankle fusion be 5 to 10 degrees equinus. Lateral radiographs of the foot and ankle should be obtained at the time of surgery, and the position of stabilization of the ankle should be accurately measured with a goniometer.

Pronation or supination of the forefoot results in unequal pressure on its sides and may also cause painful callosities and ulceration. When the forefoot is in supination, callosities develop over the fifth metatarsal head, whereas in pronation they develop over the first and second metatarsal heads. In excessive equinus inclination callosities develop over the first and fifth metatarsal heads.

The plantar surface of the foot should be in the normal weightbearing position, with no pronation or supination or uneven pressure under the metatarsal heads. The lateral border of the foot should be straight, with the heel in neutral or slightly valgus position and the ankle in less than 10 degrees of plantar flexion.

Waugh and associates reported the results of 116 pantalar arthrodeses performed in 97 patients after a mean follow-up of 5 years and an average follow-up of 6.9 years. In general, pantalar arthrodesis was found to be an effective and satisfying procedure. About 80 percent of the patients had no complaints referable to their pantalar arthrodesis. Adequate compensatory motion developed in the forepart of the foot, so that rigidity of the feet was not a problem, despite fusion of the ankle and hindpart of the foot. Of the 52 patients who had used an orthosis preoperatively, 47 were able to discard it following fusion. Pseudarthrosis occurred in 14.7 percent of the cases.[331]

When the foot has normal alignment and adequate bony and ligamentous stability, the author recommends ankle fusion only, using the distraction-compression bone graft arthrodesis described by Chuinard and Peterson (Plate 26–9).[63] In the paralyzed limb, there are no compressional forces to maintain the tibia and talus in close apposition, and the weight of the cast pressing on the dorsum of the foot may distract them.

ANTERIOR OR POSTERIOR BONE BLOCKS TO LIMIT MOTION AT ANKLE

In pes calcaneus, construction of a bone buttress anteriorly in the talus will limit dorsiflexion of the ankle by impinging upon the anterior lip of the distal tibia, whereas in equinus deformity of the foot, plantar flexion of the ankle may be restricted by bone block construction on the posterior aspect of the talus.[46,47,109,110] These procedures were developed for use in cases of paralytic calcaneus or drop foot, when there is no musculature available for transfer to provide plantar flexion or dorsiflexion power. Long-term follow-up studies of bone block operations have disclosed a high incidence of recurrence of deformity and fibrous ankylosis or painful degenerative arthritis of the ankle joint. The author does not recommend bone blocks to limit motion at the ankle, as the procedure has all the disadvantages of arthrodesis of the ankle without providing the pain-free stability of the latter.

The only indication for a posterior bone block is in a female patient who desires to wear shoes with heels of varying height and who has fair strength of the triceps surae muscle but no dorsiflexor power. Following triple arthrodesis, small subarticular grafts are placed posteriorly to lift the articular surface of the posterior aspect of the talus and limit plantar flexion. Massive bone grafts that abut the posterior aspect of the tibia should not be used. The small blocks placed beneath the articular surface of the talus are just as effective, heal rapidly, can be performed in combination with triple arthrodesis, and are less likely to cause pain.[117]

The Trunk

The etiology and treatment of pelvic obliquity are discussed in the section on contracture of the iliotibial band. Pelvic obliquity may also be caused by unilateral paralysis of the quadratus lumborum muscle. Paralysis of the abdominal muscles will result in exaggeration of the anterior pelvic tilt and an increase in lumbar lordosis. Lowman has described fascial transplants to substitute for the paralyzed abdominal muscles. For indications and operative technique, the reader is referred to Lowman, Dickson, Clark and Axer, Mayer, and Williamson, Moe, and Basom.[7,65,85,202–204,220,339]

The management of paralytic spine deformity is discussed in Chapter 11, Scoliosis. As with deformity of other etiologies, paralytic deformity in poliomyelitis responds best to combined anterior and posterior fusions with stable instrumentation.[9a] *Instrumentation without fusion has not been successful.*[5a] *Halo femoral traction has also not proved to be beneficial.*[10a] *The role of pelvic obliquity and scoliosis is well discussed by Lee and associates.*[8a]

The Shoulder

The shoulder joint is a multiaxial joint. On full abduction of the shoulder, scapulothoracic motion contributes 60 degrees and glenohumeral motion contributes 120 degrees, in a ratio of 1 : 2. When the arm is abducted to 90 degrees, the arm should rotate externally to allow full abduction. Full 180-degree forward flexion is permitted by internal rotation of the arm. Extension of the shoulder joint is limited to 80 degrees by the mechanical block of the acromion process and the adjacent spine of the scapula. Normal scapulohumeral rhythm is imperative for execution of graceful motions and strength of the shoulder joint.[276]

Saha's functional classification of the muscles acting at the shoulder joint is given below.[274,275]

PRIME MOVERS

The prime movers are the deltoid and clavicular head of the pectoralis major. They provide the greatest amount of active power in shoulder abduction, exerting force in three directions. Their insertion is most distal from the shoulder joint, at the juncture of the middle and upper thirds of the humeral diaphysis. When these prime movers are paralyzed, natural automatic substitution of function with adjacent motors is not feasible. To restore active shoulder abduction, muscle transfer is required.

STEERING GROUP

The steering group consists of the subscapularis, supraspinatus, and infraspinatus muscles. Their force is exerted at the junction of the head-neck and shaft of the humerus, their primary function being to stabilize the humeral head in the glenoid cavity by steering—that is, by rolling and gliding movement. Secondarily, they assist in shoulder abduction.

The infraspinatus muscle acts primarily as a posterior glider of the humeral head during the final stages of full abduction. The supraspinatus and subscapularis muscles are indispensable for shoulder abduction, steering the head of the humerus during abduction in different planes through an arc of 150 degrees. The scapula moves anteriorly and posteriorly and rotates vertically through the extremes of

Text continued on page 1406

Extra-articular Arthrodesis of the Subtalar Joint (Grice Procedure)

OPERATIVE TECHNIQUE

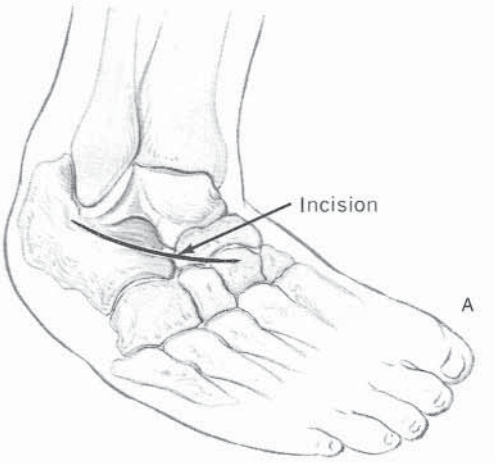
A, A 2½-inch-long, slightly curved incision is made over the subtalar joint, centered over the sinus tarsi.

B, The incision is carried down to the sinus tarsi. The capsules of the posterior and anterior subtalar articulations are identified and left intact. The operation is extra-articular. If the capsule is opened inadvertently, it should be closed by interrupted sutures.

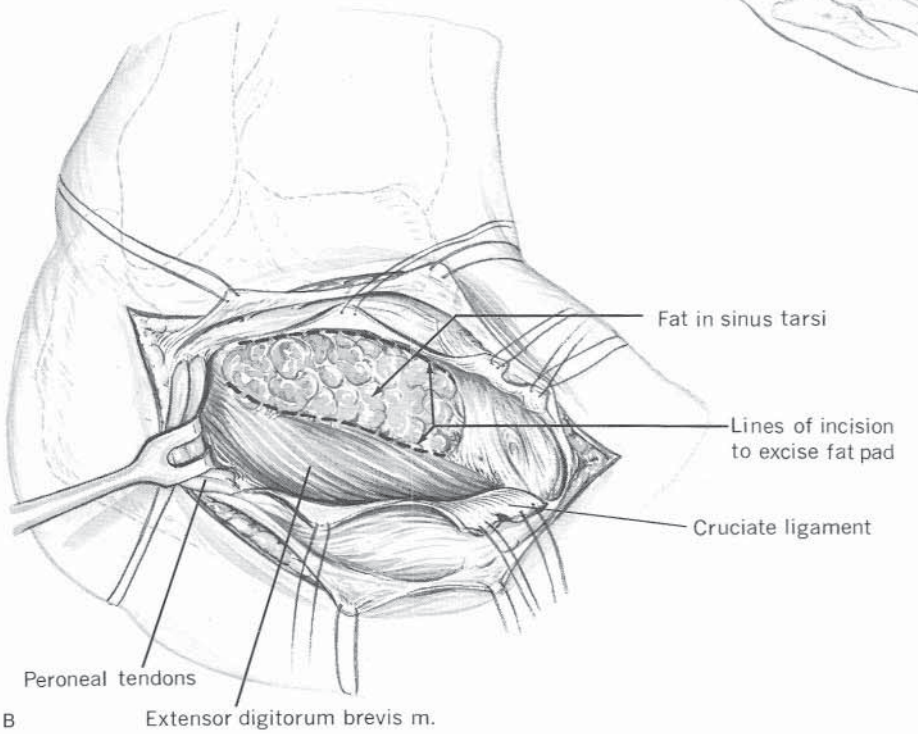
The periosteum on the talus corresponding to the lateral margin of the roof of the sinus tarsi is divided and reflected proximally. The fibrofatty tissue in the sinus tarsi with the periosteum of the calcaneus corresponding to the floor of the sinus tarsi and the tendinous origin of the short toe extensors from the calcaneus is elevated and reflected distally in one mass.

C, The remaining fatty and ligamentous tissue from the sinus tarsi is thoroughly removed with a sharp scalpel and curet.

PLATE 26–8. Extra-articular Arthrodesis of the Subtalar Joint (Grice Procedure)



A



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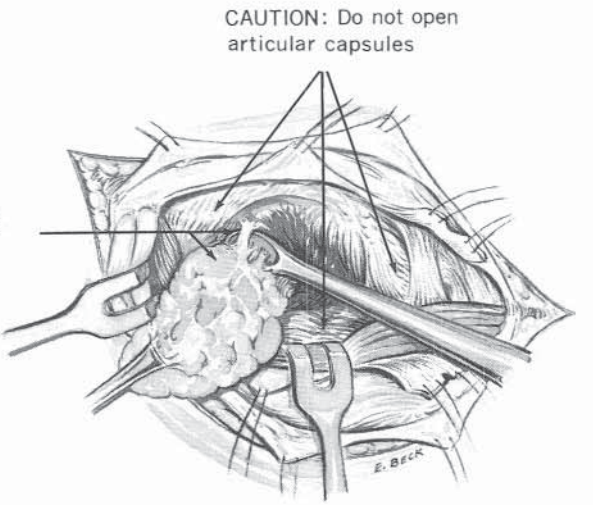


C

Extra-articular Arthrodesis of the Subtalar Joint (Grice Procedure) *Continued*

D, Next, the foot is manipulated into equinus position and inversion, rotating the calcaneus into its normal position beneath the talus and correcting the valgus deformity. Broad straight osteotomes of various sizes ($\frac{3}{4}$ to $1\frac{1}{4}$ inches or more) are inserted into the sinus tarsi, blocking the subtalar joint and determining the length and optimum position of the bone graft and the stability that it will provide. The long axis of the graft should be parallel with that of the leg when the ankle is dorsiflexed into neutral position, and the hindfoot should be 5 degrees valgus or neutral, but never varus. Even a slight degree of varus deformity of the heel seems to increase with growth.

E, The optimum site of the bone graft bed is marked with the broad osteotome. A thin layer of cortical bone ($\frac{1}{8}$ to $\frac{3}{16}$ inch) is removed with a dental osteotome from the inferior surface of the talus (the roof of the sinus tarsi) and the superior surface of the calcaneus (the floor of the sinus tarsi) at the marked site for the bone graft. It is best to preserve the most lateral cortical margin of the graft bed to support the bone block and to prevent it from sinking into soft cancellous bone.

F, A bone graft of appropriate size can be taken from the anteromedial surface of the proximal tibial metaphysis as a single cortical graft, which is then cut into two trapezoidal bone grafts with their cancellous surfaces facing each other. Lately the author prefers to use fibular bone grafts with the cortices intact. The corners of the base of the graft are removed with a rongeur so that it is trapezoidal in shape and can be countersunk into cancellous bone, preventing lateral displacement after operation.

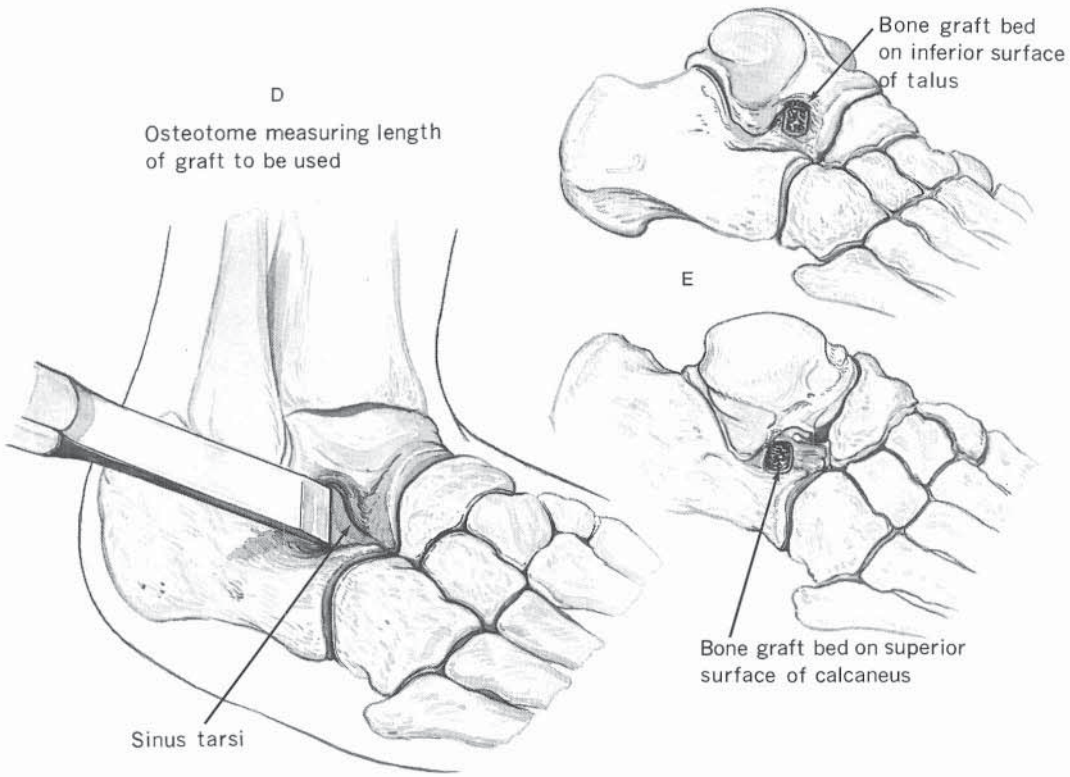
The bone graft is placed in the prepared graft bed in the sinus tarsi by holding the foot in varus position. An impacter may be used to fix the cortices of the graft in place. The longitudinal axis of the graft should be parallel with the shaft of the tibia with the ankle in neutral position.

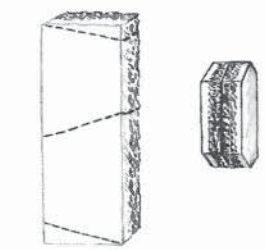
With the foot held in the desired position, the distal soft tissue pedicle of fibrofatty tissue of the sinus tarsi, the calcaneal periosteum, and the tendinous origin of the short toe extensors are sutured to the reflected periosteum from the talus. The subcutaneous tissue and skin are closed with interrupted sutures, and an above-knee cast is applied.

POSTOPERATIVE CARE

The cast is removed 6 to 10 weeks after operation and radiographs are taken. If there is solid healing of the graft, gradual weightbearing is allowed with the protection of crutches. Active and passive exercises are performed to strengthen the muscles and to increase the range of motion of the ankle and the knee.

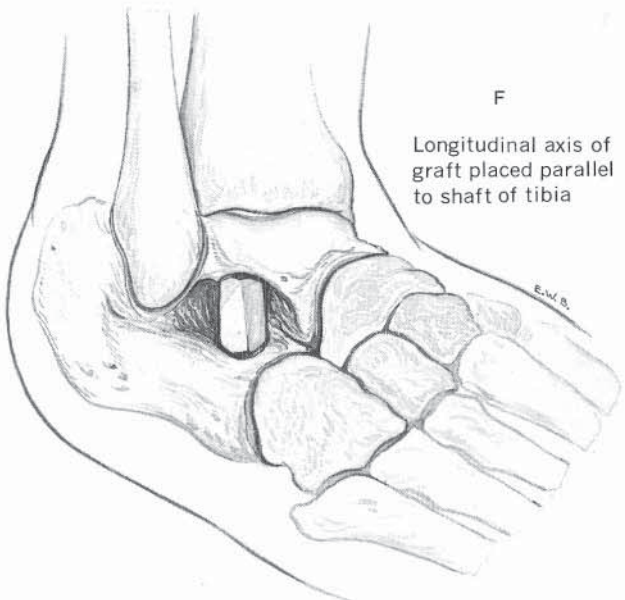
PLATE 26–8. Extra-articular Arthrodesis of the Subtalar Joint (Grice Procedure)





Shaping of bone graft from tibia

Shaped fibular graft
(Author's preferred method)



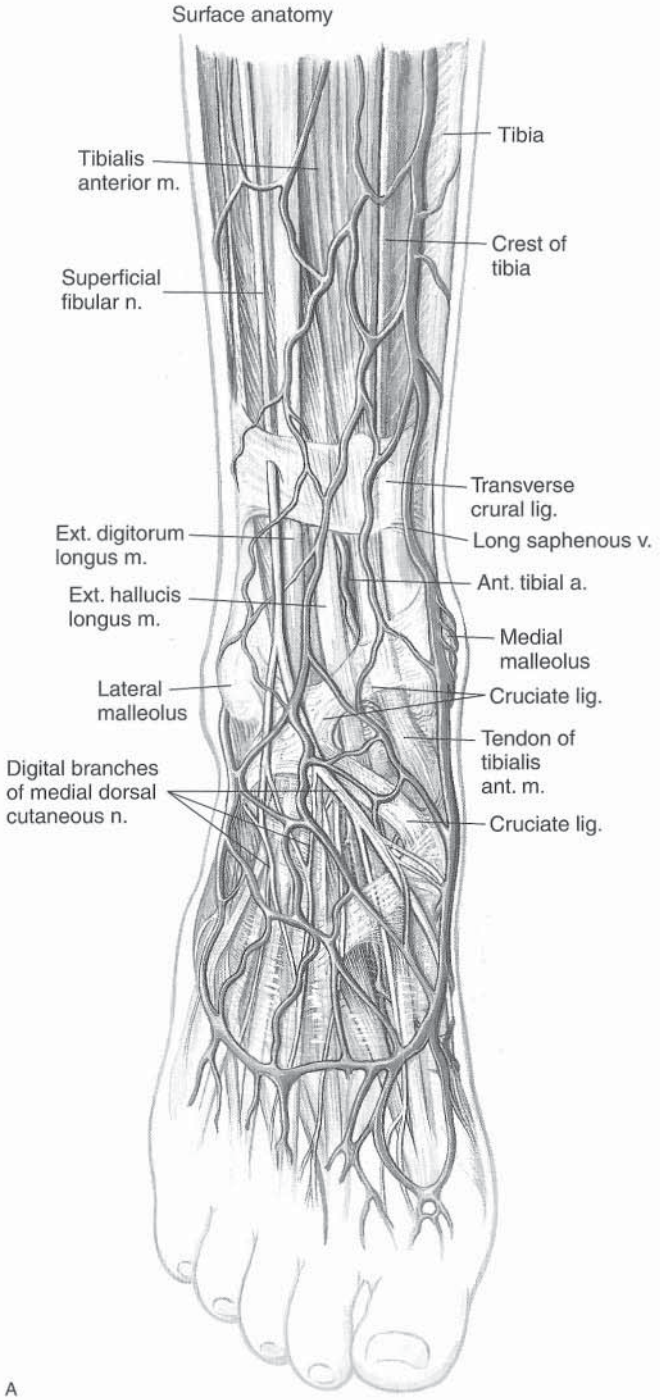
Arthrodesis of the Ankle Joint Through the Anterior Approach Without Disturbing the Distal Tibial Growth Plate

OPERATIVE TECHNIQUE

A and **B,** A longitudinal skin incision is made beginning 7 cm proximal to the ankle joint between the extensor digitorum longus and extensor hallucis longus tendons and extended distally across the ankle joint in line with the third metatarsal, ending 4 cm distal to the ankle joint.

The subcutaneous tissue is divided and the skin flaps are mobilized and retracted to their respective sides. The veins crossing the field are clamped, divided, and coagulated. The intermediate and medial dorsal cutaneous branches of the superficial peroneal nerve are identified and protected by retraction to one side of the wound.

PLATE 26–9. Arthrodesis of the Ankle Joint Through the Anterior Approach Without Disturbing the Distal Tibial Growth Plate



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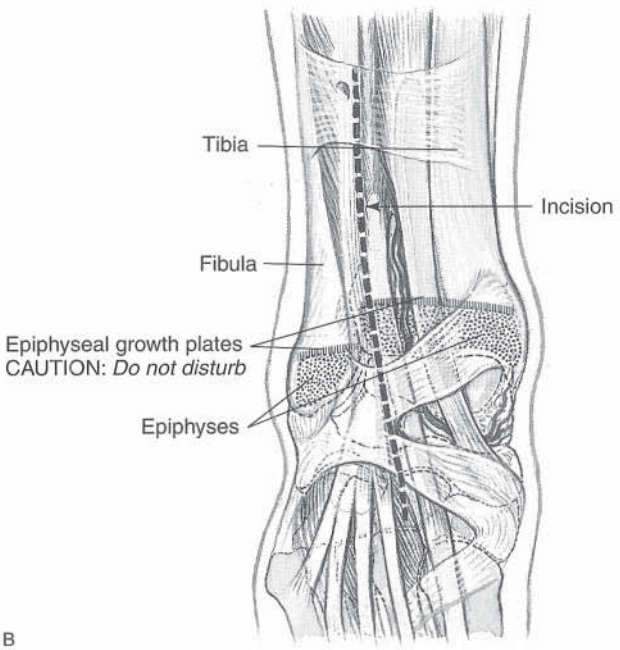


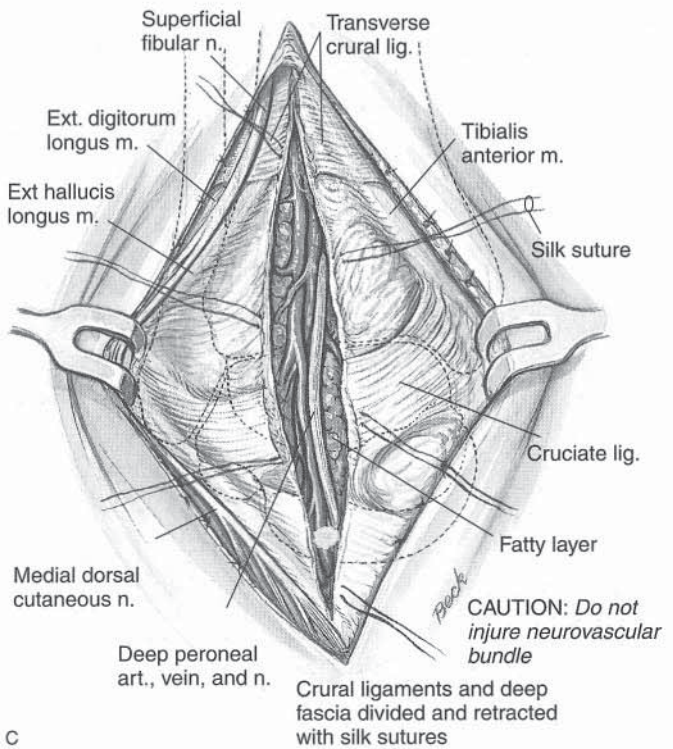
B

Arthrodesis of the Ankle Joint Through the Anterior Approach Without Disturbing the Distal Tibial Growth Plate *Continued*

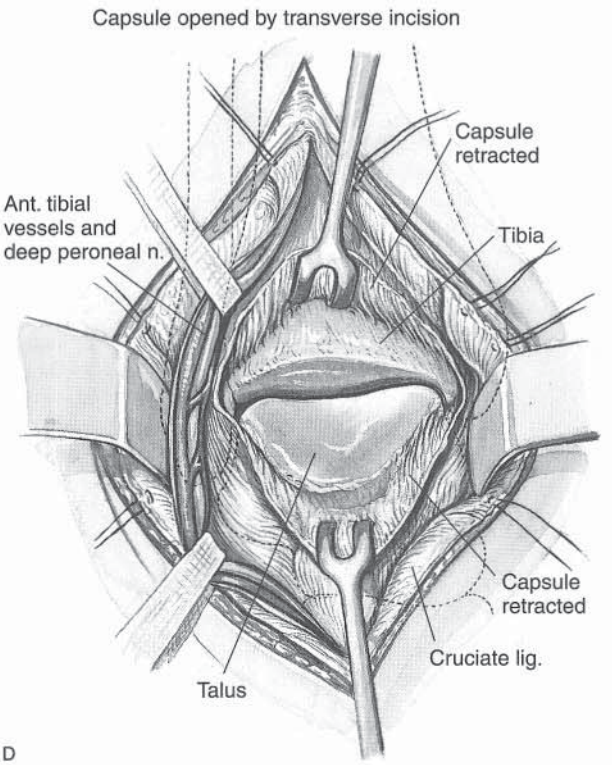
C, The deep fascia and transverse crural and cruciate crural ligaments are divided in line with the skin incision. The ligaments are marked with 00 silk suture for accurate closure later.

D, The neurovascular bundle (deep peroneal nerve, anterior tibial–dorsalis pedis vessels) is identified, isolated, and retracted laterally with the extensor hallucis longus, extensor digitorum longus, and peroneus tertius tendons. The anterolateral malleolar and lateral tarsal arteries are isolated, clamped, divided, and ligated. The distal tibia, ankle joint, and talus are identified. A transverse incision is made in the capsule of the talotibial joint from the posterior tip of the medial malleolus to the lateral malleolus. The edges of the capsule are marked with 00 silk suture for meticulous closure later.

PLATE 26–9. Arthrodesis of the Ankle Joint Through the Anterior Approach Without Disturbing the Distal Tibial Growth Plate



C



D

Arthrodesis of the Ankle Joint Through the Anterior Approach Without Disturbing the Distal Tibial Growth Plate *Continued*

E to **G,** The capsule is reflected and retracted distally on the talus and proximally on the tibia. The periosteum of the tibia should not be divided. The distal tibial and fibular epiphyseal plates should not be disturbed in growing children. With thin curved and straight osteotomes, the cartilage and subchondral bone are removed from the opposing articular surfaces of the distal tibia and proximal talus down to raw bleeding cancellous bone. Cartilage chips should not be left posteriorly.

H, Next, a large piece of bone for grafting is taken from the ilium and fashioned to fit snugly in the ankle joint. The graft should have both cortices intact and should be thicker at one end and wedge-shaped. The cortices of the graft are perforated with multiple tiny drill holes. The ankle joint is held in the desired position, and the bone graft is firmly fitted into the joint with an impacter. If any space is left on each side of the graft, it is packed with cancellous bone from the ilium. The graft in the ankle joint gives compressional force to the arthrodesis and adds to the height of the foot and ankle. The capsule of the ankle joint and the transverse crural and cruciate crural ligaments are closed carefully in layers. The deep fascia and the wound are closed in the usual manner. Radiographs are obtained in AP and lateral views to ensure that the ankle joint is in the desired position.

I, A long-leg cast is applied with the ankle joint in the desired position of plantar flexion (boys, 10 degrees; girls, 15 to 20 degrees) and the knee in 45 degrees of flexion.

POSTOPERATIVE CARE

Periodic radiographs are obtained to determine the position of the graft and the extent of healing. Eight to 10 weeks after surgery, the solid cast is removed and radiographs are obtained with the cast off. Ordinarily, by this time, the fusion is solid and the patient is gradually allowed to be ambulatory. Full weightbearing is begun 2 to 3 weeks later.

PLATE 26–9. Arthrodesis of the Ankle Joint Through the Anterior Approach Without Disturbing the Distal Tibial Growth Plate

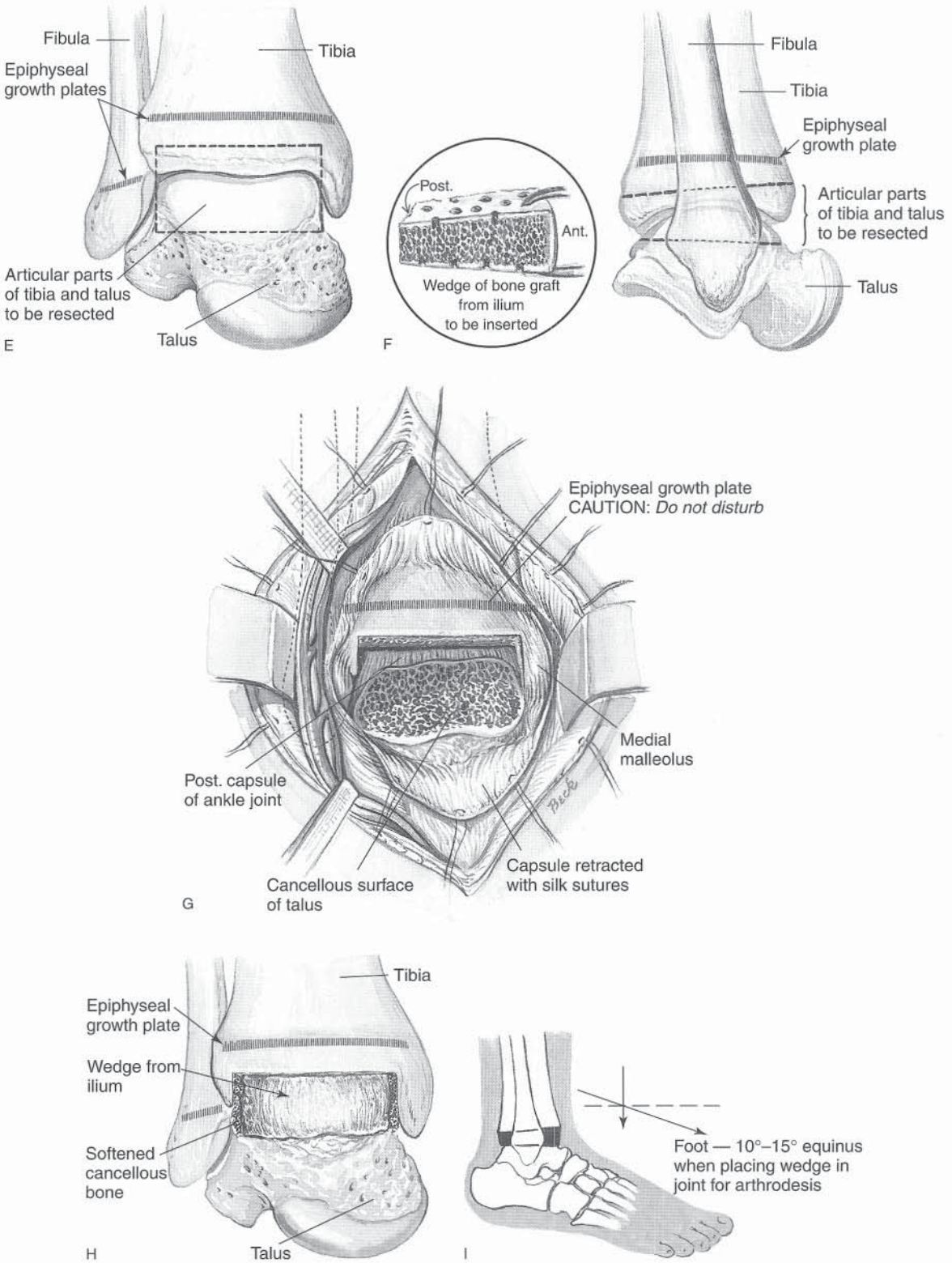


TABLE 26–3 **Possible Tendon Transfer to Restore Power at the Shoulder Joint**

Muscle Requiring Replacement or Reinforcement	Action	Choices of Muscles for Transfer (in Order of Preference)
Deltoid and clavicular head of pectoralis major	Prime mover (abduction)	1. Trapezius (as far down as possible on shaft)
Supraspinatus	Superior glider	1. Levator scapulae (first choice because of direction and length of its fibers) 2. Sternocleidomastoid 3. Scalenus anterior 4. Scalenus medius 5. Scalenus capitis (All act from above and are good substitutes.)
Infraspinatus	Posterior glider (acting from behind)	1. Latissimus dorsi 2. Teres major
Subscapularis	Posterior glider	1. Upper two digitations of serratus anterior 2. Pectoralis minor 3. Pectoralis major (whole or part) (These muscles act in almost same direction as fibers of subscapularis.)

Modified from Saha AK: Surgery of the paralyzed and flail shoulder. Acta Orthop Scand Suppl 1967;97:40.

the arc (about 30 degrees on either side). In general, vertical gliding of the humeral head is accomplished by the muscle fibers acting in the plane of motion, whereas the muscle fibers that are anterior and posterior to these act to glide the humeral head in the horizontal plane at succeeding stages of shoulder abduction.

DEPRESSOR GROUP

The depressor group consists of the sternal head of the pectoralis major, the latissimus dorsi, and the teres major and teres minor. (Teres minor is included in this group, as electromyographic evidence has shown that it participates in this motion, although anatomically it is classified as belonging to the short rotator group.) The function of these muscles is to rotate the shaft of the humerus during abduction and to depress the humeral head, assisting in the last few degrees of full abduction. The steering action that they exert on the humeral head is minimal, however.

Scapular rotation takes place through its body in an AP axis and contributes about 60 degrees of total shoulder abduction. Fixation of the scapula is equally important during abduction provided by gravity and the lower fibers of the serratus.

When the deltoid and clavicular head of the pectoralis major are paralyzed, it is important to determine the action of the steering group of muscles when performing tendon transfers to restore shoulder abduction. If the latter are paralyzed, transfer of a single muscle (such as the trapezius) or of several muscles to a common attachment to restore function will give at best only 90 degrees of shoulder abduction, and scapulohumeral rhythm will still be disturbed. According to Saha, if there is paralysis of any two of the steering group of muscles, appropriate tendon transfers should be performed to restore their function. This is as imperative as trapezius transfer for paralysis of the deltoid. Table 26–3 gives Saha's recommendations for possible tendon transfers to restore power at the shoulder joint.

The Saha trapezius transfer for paralysis of the deltoid is shown in Figure 26–27. The upper and middle trapezius are completely mobilized from its insertion, providing an additional 5 cm of length without disturbing the nerve and blood supply to the muscle. The detached portion of the

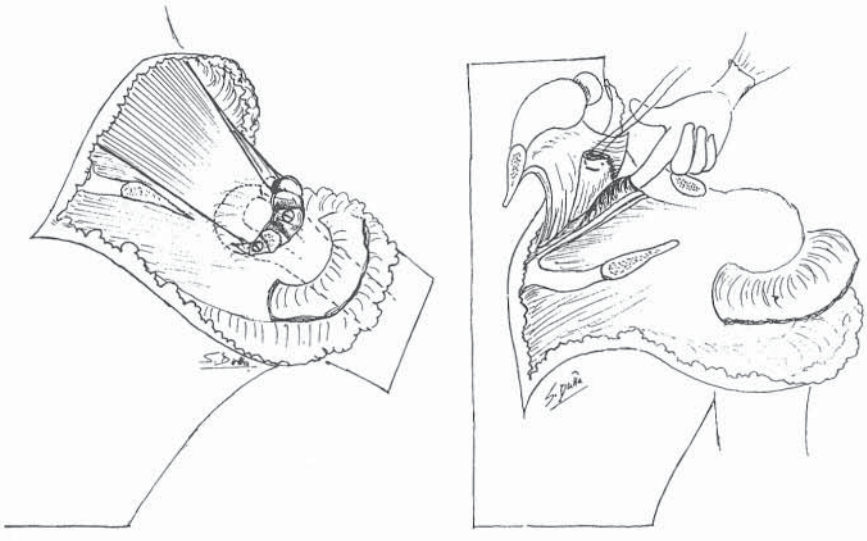


FIGURE 26–27 Trapezius muscle transfer (Saha) for paralysis of the deltoid muscle. (From Saha AK: Surgery of the paralyzed and flail shoulder. Acta Orthop Scand Suppl 1967;97:57.)

trapezius with the distal end of the clavicle, the capsule of the acromioclavicular joint, the acromion process, and the adjoining portion of the posterior border of the spine of the scapula are rerouted and attached by two screws to the humeral shaft as far distally as possible. The acromion is crushed to aid coaptation with the curve of the shaft of the humerus.

Transfer of the upper two digitations of the serratus anterior for paralysis of the subscapularis is shown in Figure 26–28. Levator scapulae transfer for paralysis of the supraspinatus is shown in Figure 26–29, pectoralis minor transfer for paralysis of the subscapularis in Figure 26–30, sternocleidomastoid transfer for paralysis of the supraspinatus in Figure 26–31, and either latissimus dorsi or teres major transfer, or both, for paralysis of the subscapularis in Figure 26–32. For technical details the reader is referred to Saha's original article.[274]

ARTHRODESIS OF THE SHOULDER

Arthrodesis of the shoulder is indicated when there is paralytic subluxation or dislocation of the shoulder and extensive paralysis of the scapulohumeral muscles. Because scapulothoracic motion will serve as a substitute for glenohumeral joint motion, it is important that the motor strength of the trapezius and serratus anterior be normal. The direct action of the scapula will move the arm. Normal function of the hand, however, is a primary requisite. It is best to delay shoulder arthrodesis until after epiphyseal closure has taken place.

The optimum position for shoulder fusion, as recommended by the Research Committee of the American Orthopedic Association, is 50 degrees of abduction, 20 degrees of flexion, and 25 degrees of internal rotation. This position is very functional, allowing the patient to reach the face and the top of the head with the elbow flexed.[13]

It is wise, however, to consider the sex and occupation of the patient, and the regional muscle power and functional status of the opposite limb. In general, office workers require more abduction than do laborers. In females, the degree of abduction should be less, since this permits the scapula a better resting position in relation to the trunk. For cosmetic reasons, females strongly object to the winging of the scapula. The lesser degree of abduction is functionally compensated for by fusing it in greater internal rotation. The most acceptable position of shoulder arthrodesis in females is 30 degrees of glenohumeral abduction, 5 to 10 degrees of flexion, and 45 degrees of internal rotation. The shoulder should never be fused in external rotation, as the limb will be positioned in an awkward and functionally poor position.

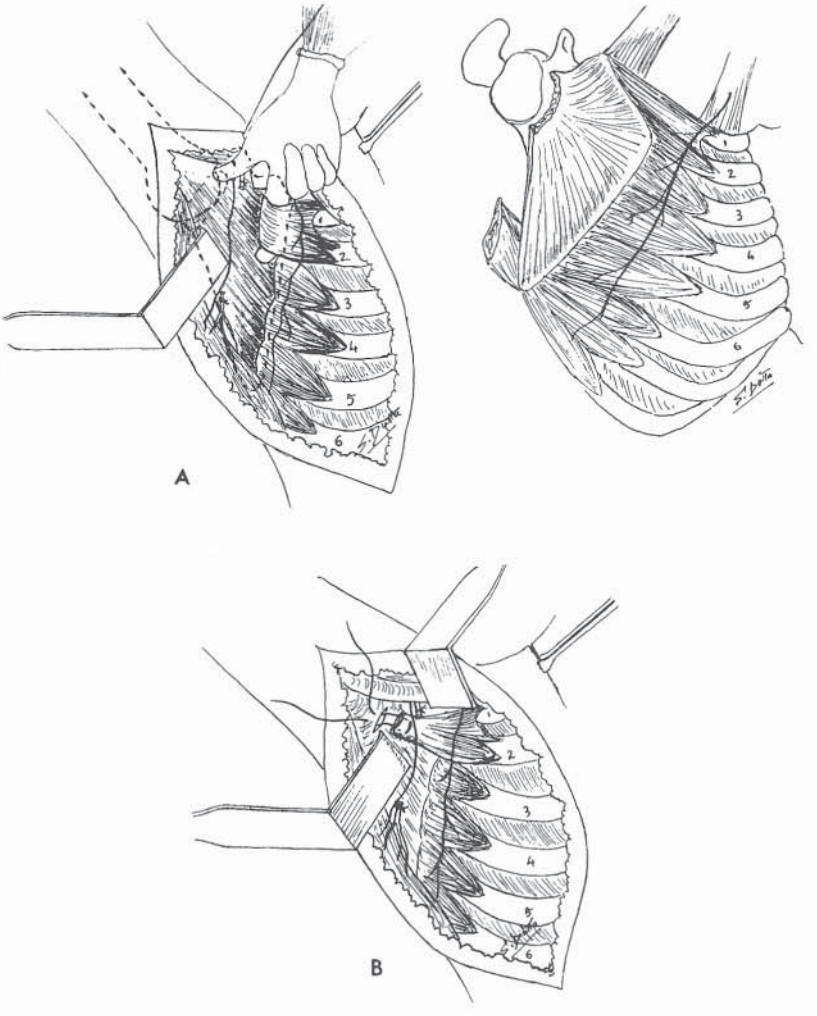


FIGURE 26–28 **A** and **B,** Transfer of upper two digitations of serratus anterior for paralysis of the subscapularis muscle. (From Saha AK: Surgery of the paralyzed and flail shoulder. Acta Orthop Scand Suppl 1967;97:59.)

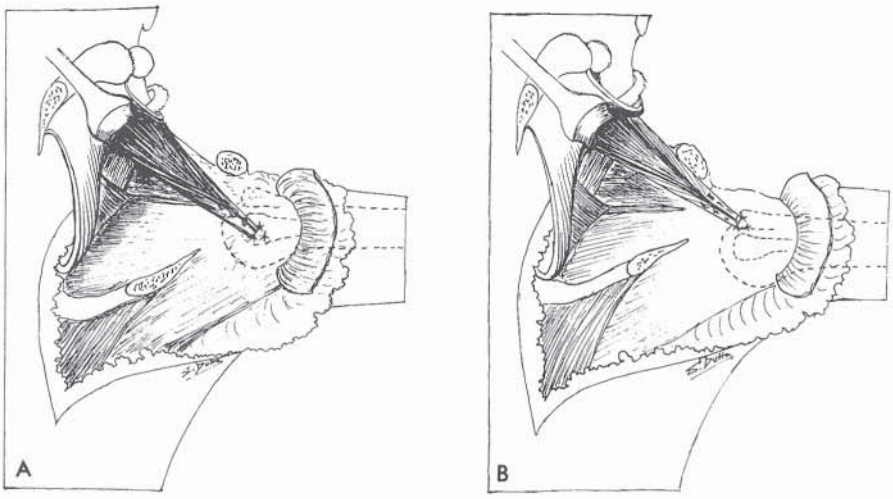


FIGURE 26–29 **A** and **B,** Levator scapulae transfer for paralysis of the supraspinatus. (From Saha AK: Surgery of the paralyzed and flail shoulder. Acta Orthop Scand Suppl 1967;97:60.)

It should be explained to the patient that following surgery, extension and rotation of the shoulder will be limited and that the patient will have difficulty lying on the side of the arthrodesis to sleep. Fusion of both shoulders should never be performed because of the loss in range of motion. Arthrodesis of the shoulder will increase the power of both flexion and extension of the elbow and will provide adduction power to the shoulder, enabling the patient to grip an object between the arm and the body.

For technical details of shoulder fusion, the reader is referred to the original descriptions in the literature.* It should be emphasized, however, that the position of fusion should be calculated according to the angle between the humerus and scapula rather than that of the arms and thorax. Internal fixation is imperative; otherwise, the angle will be changed in the shoulder spica cast.

The Elbow

Loss of elbow flexion results from paralysis of the biceps brachii and the brachialis muscles. The resultant functional deficit is considerable, as the patient is unable to bring the hand to the head, mouth, or trunk.

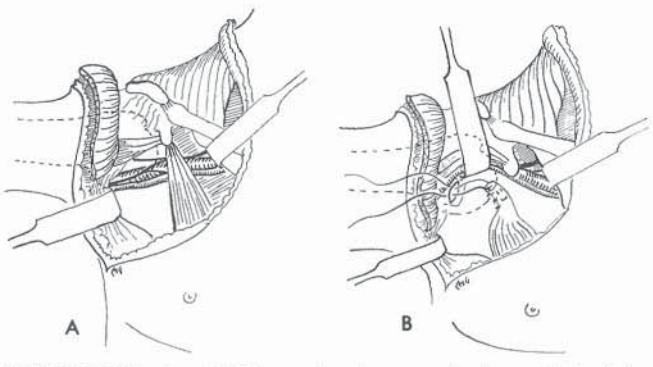


FIGURE 26–30 **A** and **B,** Pectoralis minor transfer for paralysis of the subscapularis. (From Saha AK: Surgery of the paralyzed and flail shoulder. Acta Orthop Scand Suppl 1967;97:61.)

*See references 13, 20, 34, 39, 40, 56–58, 108, 179, 330.

A number of operative procedures have been devised to restore elbow flexion. These include (1) the Steindler flexorplasty,[303] (2) transfer of the distal third of the pectoralis major muscle (Clark),[64] (3) transfer of the pectoralis major tendon (Brooks and Seddon),[38] (4) transfer of the sternocleidomastoid muscle (Bunnell),[41] (5) transfer of the latissimus dorsi (Hovnanian),[162] (6) transfer of the pectoralis minor (Spira),[298] and (7) anterior transfer of the triceps brachii tendon to the biceps insertion on the radial tuberosity (Bunnell[41] and Carroll[51]).

Before a specific procedure is selected it is imperative to carefully assess the motor strength of the muscles of the entire upper limb and the functional status of the opposite limb. With paralysis of the elbow flexors, there is often a varying degree of paresis of the muscles of the scapulohumeral joint, forearm, and hand.

Function of the hand is of primary concern. Restoration of elbow flexion is only one step in total functional reconstruction of the upper limb, and procedures on the hand should precede those on the elbow. In the absence of a functional hand, flexorplasty of the elbow should not be performed.

STEINDLER FLEXORPLASTY

Steindler in 1918 described a procedure in which the humeral origins of the flexor carpi radialis, the palmaris longus, the pronator teres, the flexor digitorum sublimis, and the flexor carpi ulnaris were transferred to a more proximal site on the humerus, thereby changing the leverage of these muscles across the elbow joint and enhancing their flexor action at the elbow.[303] Later he reviewed the results of flexorplasties of the elbow and pointed out that the disadvantage of this transfer was an increase in the pronatory action of these muscles on the forearm.[304,307,309,310]

Bunnell modified the Steindler flexorplasty by attaching the transferred muscles to the lateral border of the humerus to decrease their tendency to cause pronation. To reach the lateral border of the humerus, the common flexor muscles had to be elongated with a fascial graft (Fig. 26–33).[41] This method of fascial lengthening is technically difficult if one is to obtain secure fixation and maximum strength of the flexorplasty. Mayer and Green attached the transferred mus-

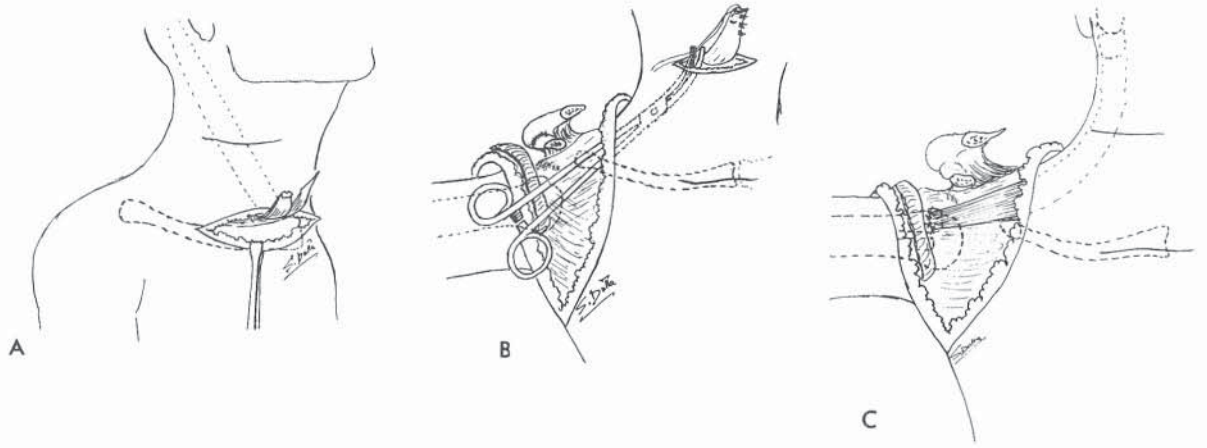


FIGURE 26–31 A to C, Sternocleidomastoid transfer for paralysis of the supraspinatus muscle. (From Saha AK: Surgery of the paralyzed and flail shoulder. Acta Orthop Scand Suppl 1967;97:62.)

cles on the anterior surface of the humerus through holes drilled in bone.[222] This direct fixation to bone ensured firm healing of the transferred muscle. The operative technique of Steindler flexorplasty as modified by Mayer and Green is described and illustrated in Plate 26–10.

Carroll and Gartland reported the results of Steindler flexorplasty in 27 patients.[52] Kettelkamp and Larson evaluated the results of Steindler flexorplasty in 15 patients, determining the maximum strength of the flexorplasty through a useful range of flexion. Nine of the 15 patients were able to lift a weight of 1 pound or more to 110 degrees of flexion. On correlating the strength of the flexorplasty with the degree of flexion contracture of the elbow, they found the average strength of the flexorplasty to be 2 pounds to 110 degrees of flexion when the degree of flexion contracture was between 30 and 60 degrees. The mechanical advantage and strength of the transfer were increased in the presence of flexion contracture of the elbow.[178] When there is marked paralysis of the opposite limb, the strength restored by flexorplasty is a greater consideration than is cosmetic appearance.

Mayer and Green, however, emphasize the importance of the appearance of the arm, preferring that the flexion position of the elbow not exceed 15 degrees. They recommend that a turnbuckle extension orthosis be worn at night to prevent the development of flexion contracture of the elbow. As soon as the strength of the transfer is fair or fair plus, a night splint is used and passive stretching exercises of the elbow flexors are performed.[222]

The motor strength of the triceps is another important factor in the development of flexion contracture of the elbow, as this contracture represents the residuum of flexorplasty and the end result of dynamic imbalance between elbow flexors and extensors over the years. A flexion defor-

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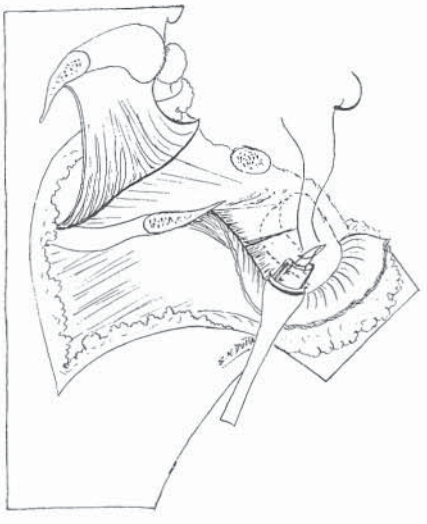


FIGURE 26–32 Latissimus dorsi or teres major transfer for paralysis of the subscapularis muscle. (From Saha AK: Surgery of the paralyzed and flail shoulder. Acta Orthop Scand Suppl 1967;97:64.)

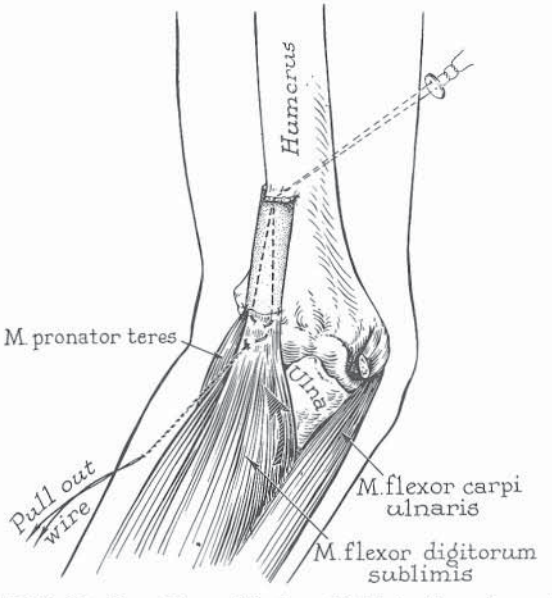


FIGURE 26–33 Bunnell's modification of Steindler flexorplasty of the elbow. The common flexor muscles are elongated by a fascial graft. (From Bunnell S: Restoring flexion to the paralytic elbow. J Bone Joint Surg 1951;33-A:566.)

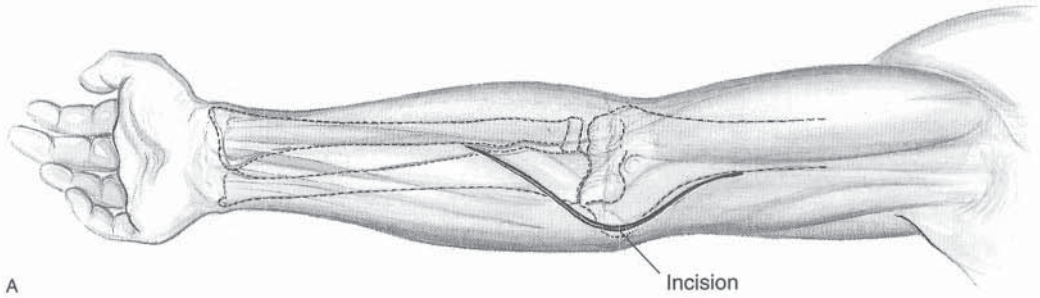
Flexorplasty of the Elbow (Steindler)

OPERATIVE TECHNIQUE

A, With the elbow in extension, a curved longitudinal incision is made over the anteromedial side of the elbow, beginning approximately 3 inches above the flexion crease of the elbow joint over the medial intermuscular septum and extending distally to the anterior aspect of the medial epicondyle. At the joint level it turns anterolaterally on the volar surface of the forearm along the course of the pronator teres muscle for a distance of approximately $2\frac{1}{2}$ inches.

B, The subcutaneous tissue and fascia are divided in line with the skin incision and the skin flaps are widely mobilized and retracted. Next, the ulnar nerve is located posterior to the medial intermuscular septum and lying in a groove on the triceps muscle. It is isolated, and a moist hernia tape is passed around it for gentle handling. The ulnar nerve is traced distally to its groove between the posterior aspect of the medial epicondyle of the humerus and the olecranon process. The fascial roof over the ulnar nerve is carefully divided under direct vision over a grooved director.

PLATE 26–10. Flexorplasty of the Elbow (Steindler)



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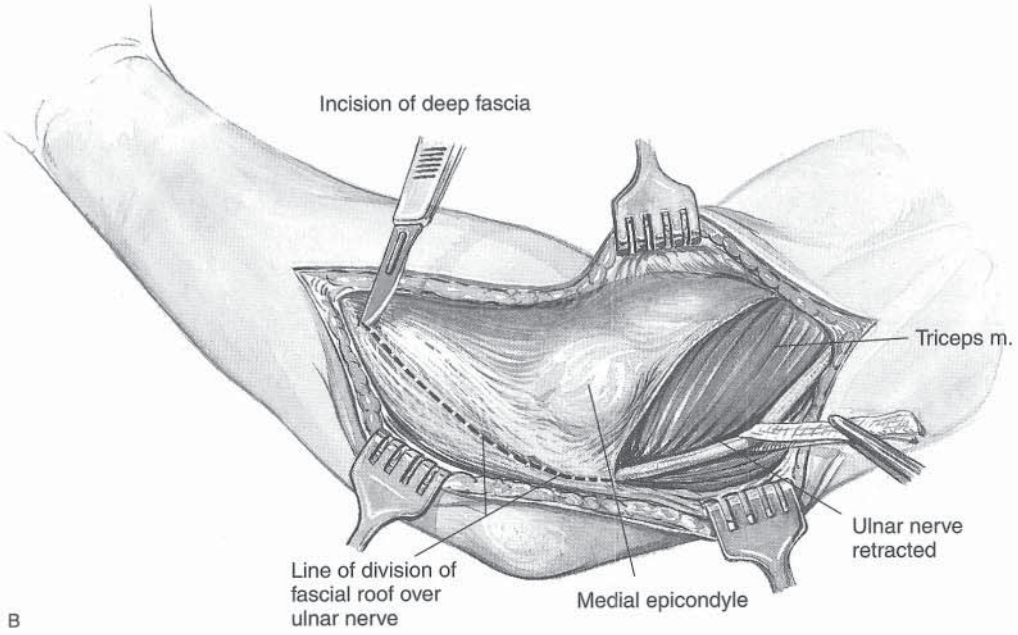


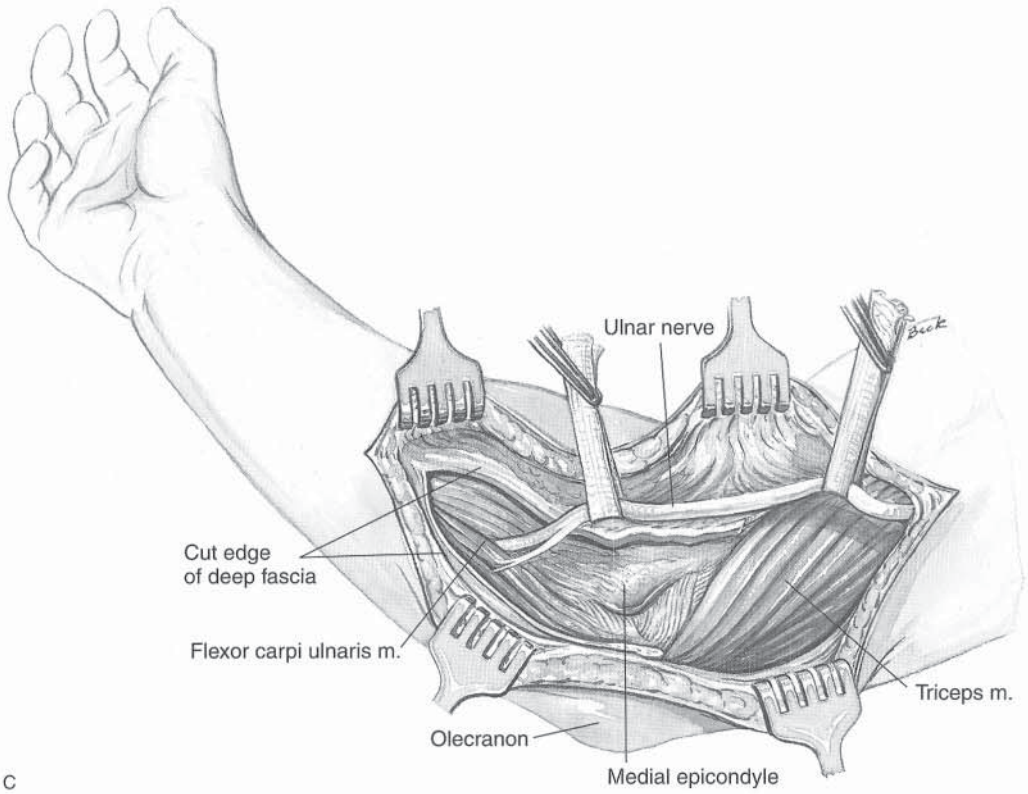
B

Flexorplasty of the Elbow (Steindler) *Continued*

C, The ulnar nerve is dissected free distally to the point where it passes between the two heads of the flexor carpi ulnaris muscle. Inadvertent damage to the branches of the ulnar nerve to the flexor carpi ulnaris muscle should be avoided. A second hernia tape is passed around the ulnar nerve in the distal part of the wound, and the nerve is retracted posteriorly.

D, Next, the biceps tendon is identified over the anterior aspect of the elbow joint. The deep fascia and the lacertus fibrosus are divided along the medial aspect of the biceps tendon.

PLATE 26–10. Flexorplasty of the Elbow (Steindler)



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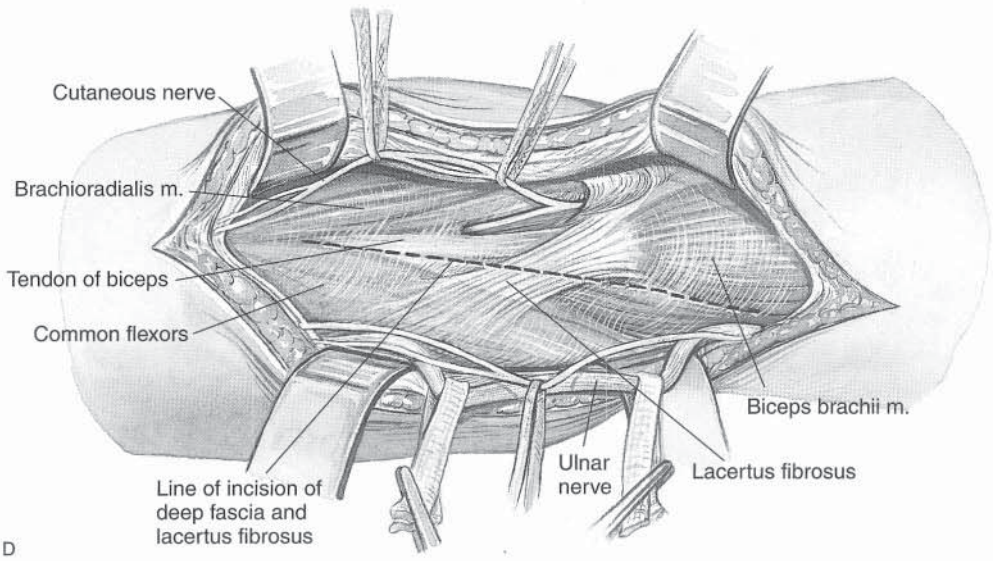


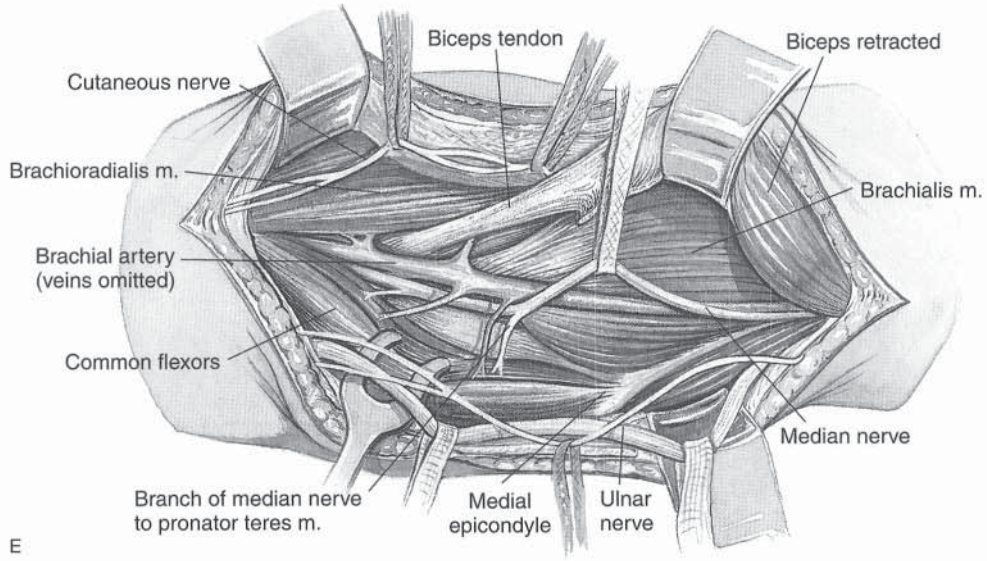
D

Flexorplasty of the Elbow (Steindler) *Continued*

E, By digital palpation, the interval between the biceps and pronator teres muscle is developed. The brachial artery with its accompanying veins runs along the medial side of the biceps tendon. The median nerve, lying medial to the brachial artery, is dissected free of the surrounding tissues and gently retracted anteriorly with a moist hernia tape. The branches of the median nerve to the pronator teres muscle must be identified and protected from injury.

F, Next, with an osteotome, the common flexor origin of the pronator teres, flexor carpi radialis, palmaris longus, flexor digitorum sublimis, and flexor carpi ulnaris is detached en bloc with a flake of bone from the medial epicondyle.

PLATE 26–10. Flexorplasty of the Elbow (Steindler)



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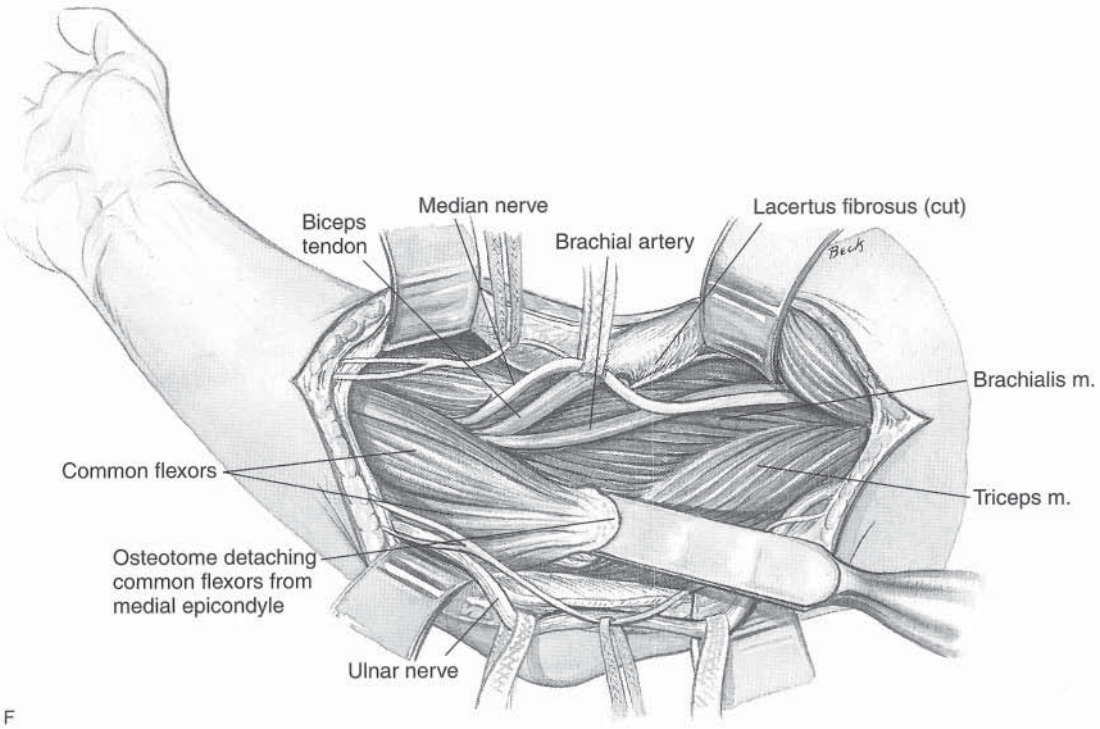


F

Flexorplasty of the Elbow (Steindler) *Continued*

G, By sharp and blunt dissection, the flexor muscle mass is freed and mobilized distally away from the joint capsule and the ulna as far as the motor branches of the median nerve and ulnar nerve will permit. A No. 1 silk whip suture is placed in the proximal end of the common flexors.

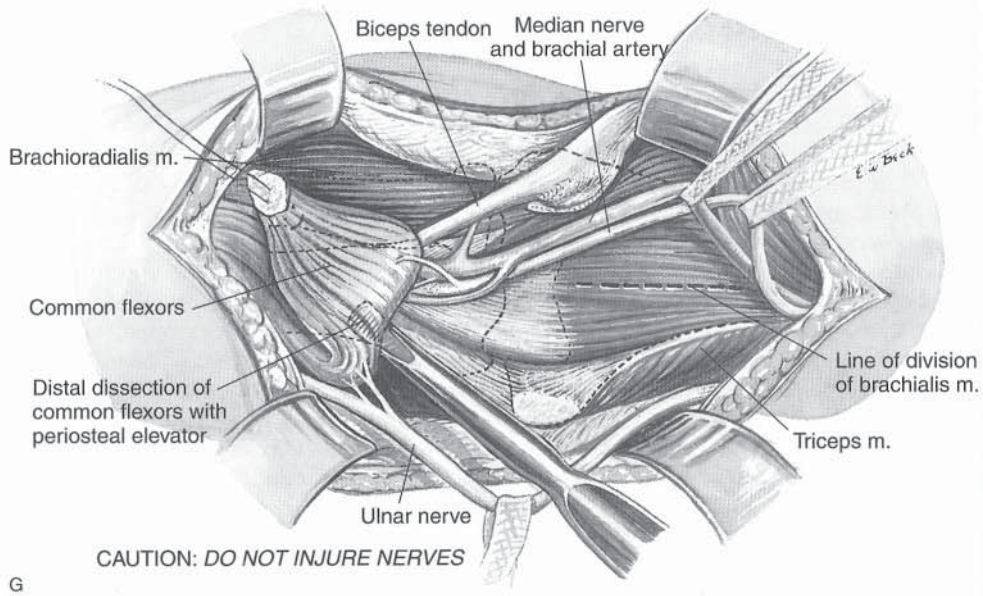
H, The biceps muscle, brachial vessels, and median nerve are retracted laterally, and the atrophied branchial muscle is split longitudinally. The periosteum is incised and stripped, exposing the anterior aspect of the distal humerus.

The elbow is then flexed to 120 degrees to determine the site of attachment of the transfer (usually 2 inches proximal to the elbow). With a drill, a hole is made on the anterior surface of the humerus. The opening is enlarged with progressively larger diamond-head hand drills to receive the transferred muscle. The action of the transfer as a pronator of the forearm is decreased by transferring it laterally on the humerus. With smaller size drill points, two tunnels are made from the lateral and medial cortices of the humerus and are connected to the larger hole for passing the suture.

I and **J,** Because the elbow will be immobilized in acute flexion, it is best to close the distal half of the wound before anchoring the transplant to the humerus. The ends of the whip suture are brought out through the tunnels, and the common flexors and the origin are firmly secured in the larger hole. The periosteum is closed with interrupted sutures over the transferred tendon, thus reinforcing its anchorage. The proximal half of the wound is closed, and a long-arm cast is applied with the elbow in acute flexion and the forearm in full supination.

For postoperative care, see the guidelines outlined in the text on principles of tendon transfer.

PLATE 26–10. Flexorplasty of the Elbow (Steindler)



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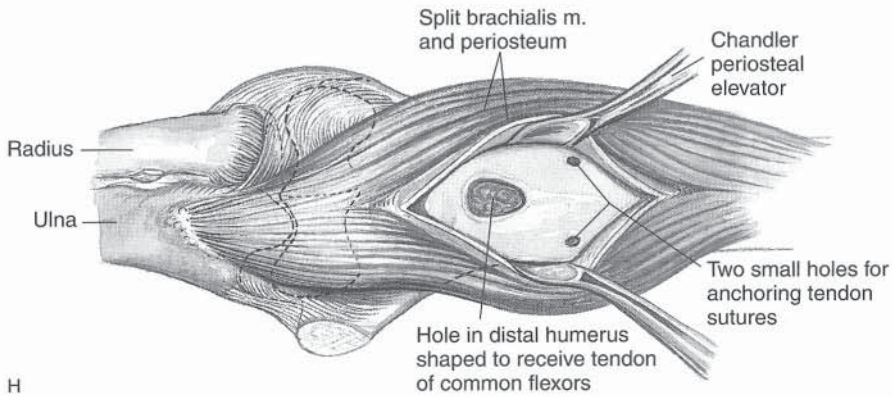

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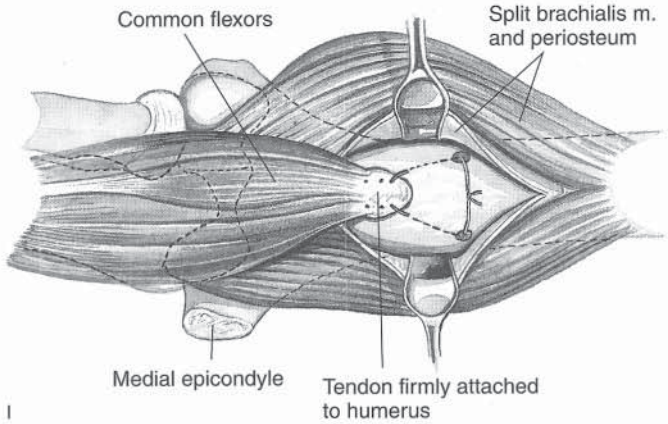

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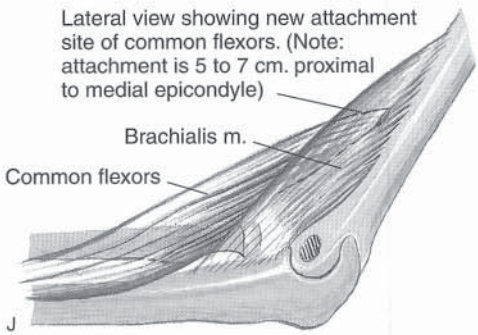


J

mity of the elbow greater than 30 degrees will usually develop when the triceps brachii is less than fair in motor strength. In the presence of a weakened triceps brachii, the period of postoperative immobilization should be short, and assisted active exercises should be started on the fifth to seventh postoperative day, with precautions taken to prevent the transfer from being stretched away from the bone. There should be an extensive period of postoperative splinting at night with the elbow in extension.

Loss of supination is an inherent complication of the Steindler flexorplasty. The attachment of the transferred muscles as far laterally on the humerus as possible minimizes the severity of pronation contracture; however, even with this precaution a varying degree of pronation deformity of the forearm results in a dynamic imbalance between a strong pronator teres and a paralyzed biceps brachii—its chief supinator opponent. The strength of the flexorplasty is not influenced by the degree of pronation contracture; however, it does impair the position of the hand and impedes its functional use. If the motor strength of the supinators of the forearm is poor, flexor carpi ulnaris transfer to the dorsum of the radius is performed to enhance supinator function and to prevent the development of pronation contracture of the forearm.

In poliomyelitis, paralysis of the elbow without paralysis of the muscles that control the scapulohumeral joint is rarely seen. A flail shoulder will greatly impede the effectiveness of the flexorplasty. In the abducted position of the shoulder, the force of gravity assists elbow flexion and the amount of strength required to flex the elbow is decreased. Arthrodesis of the shoulder will markedly improve the results of flexorplasty.

Overestimation of the motor strength of the common flexor muscle group is a frequent pitfall and accounts for poor results in some patients. For the transfer to function effectively, these muscles must rate at least good in motor strength. If they are weaker, other reconstructive methods must be employed. The flexor digitorum sublimis may be strong and the flexor carpi ulnaris and carpi radialis weak; in such an instance, following Steindler flexorplasty, flexion of the elbow is accomplished only by clenching the fingers, and any relaxation of the grip will allow the elbow to extend. This interferes with function of the hand, which is the primary interest of the patient.

PECTORALIS MAJOR TRANSFER TO RESTORE ELBOW FLEXION

A portion of the pectoralis major transfer was used by Clark to restore active elbow flexion.[64] The nerve supply of the distal third of the pectoralis major muscle (from branches of the medial thoracic nerve) is separate from that of its proximal part. This inferior strip of the pectoralis major muscle, which is 5 to 7 cm in width, is freed from the chest wall and mobilized toward the axilla as far as its nerve and blood supply will allow. The muscle is then passed subcutaneously down the arm and sutured to the biceps tendon. The reader is referred to Clark's original description for the specific technical details.[64] Clark's operation is particularly indicated in patients with traction injury of the upper trunk of the brachial plexus, in whom the clavicular head of the pectoralis major is paralyzed but the sternal head is normal in motor strength. Seddon in 1949 reported satisfactory results in 15 of 16 pectoralis major transfers performed by Clark's method.[286] In a 1959 report by Segal and associates, the results of Clark's operation in 17 patients were graded excellent or good in 47 percent and fair or failure in 53 percent.[287]

Brooks and Seddon devised a technique in which the entire pectoralis major muscle is transferred to restore elbow flexion.[38] The gap between the distal end of the pectoralis major tendon and the tuberosity of the radius is bridged by the long head of the biceps, which is detached from its origin and completely mobilized. Reducing the blood supply of the biceps brachi induced conversion of the muscle into tendon. The procedure was recommended by Brooks and Seddon in those patients in whom either the lower part of the pectoralis major is paralyzed (or too weak for the Clark transfer) but the clavicular head is strong, or the entire muscle is of such weakness that it is desirable to use all the active muscle. They reported the results as excellent or good in six, fair in two, and failure in two (one patient had arthrogryposis multiplex congenita and the other poliomyelitis).

The operative technique of the Brooks-Seddon procedure is described and illustrated in Plate 26–11. The author recommends its use when the Steindler flexorplasty is not applicable; of course, it should be employed only when the biceps brachii is completely and permanently paralyzed. The procedure does restore some degree of active supination of the forearm and rarely limits passive extension of the elbow.

One disadvantage of the pectoralis major transfer is that in the presence of weak scapulohumeral muscles, active flexion of the elbow is often accompanied by shrugging, adduction, and internal rotation of the shoulder. These undesirable motions, with the hand hitting the chest wall, seriously impair the result of the operation. If there is appreciable paralysis of the shoulder muscle, the pectoralis major transfer must be followed by arthrodesis of the scapulohumeral joint.

PECTORALIS MINOR TRANSFER

Spira in 1957 reported the successful use of the pectoralis minor as a motor to restore elbow flexion. The patient had complete paralysis of the pectoralis major, biceps brachii, and brachialis muscles. A tube of fascia lata was used to bridge the gap between the detached origin of the pectoralis minor and the paralyzed biceps tendon.[298]

STERNOCLEIDOMASTOID TRANSFER

Bunnell utilized the sternocleidomastoid muscle as a motor to restore active elbow flexion.[41] The sternoclavicular insertion of the sternocleidomastoid muscle is detached and the distal half of the muscle is mobilized by gentle blunt dissection. A long tube of fascia lata is used to bridge the gap, extending from the distal end of the sternocleidomastoid muscle and then passing forward subcutaneously in the arm to the elbow, where the graft is attached to the tuberosity of the radius (Fig. 26–34).

Carroll reported results in 15 cases of sternocleidomastoid transfer, with satisfactory results in 80 percent. He

stressed the importance of placing the transferred muscle under maximal tension at the time of operation.[50]

In the personal experience of the author with seven patients, the strength of elbow flexion following Bunnell's sternocleidomastoid transfer is excellent; however, the aesthetic appearance of the procedure is very grotesque and objectionable, particularly in females. It should be used in cases in which it is the only method available, but it must be limited to male patients, in whom function is the primary consideration and the resultant deformity can be hidden by the buttoned collar of a shirt.

ANTERIOR TRANSFER OF THE TRICEPS BRACHII

Anterior transfer of the triceps brachii was described by Bunnell in 1948 and in 1951, and later by Carroll, who showed the feasibility and effectiveness of the procedure without the use of the fascial graft that was recommended by Bunnell.[41,53] In 1970 Carroll and Hill reported the results of triceps transfer in 15 patients (8 with arthrogryposis multiplex congenita and 7 with posttraumatic and paralytic loss of elbow flexion). The criteria of success were preoperative inability to flex the elbow against gravity and bring the hand to the mouth, and postoperative ability to do so. Carroll and Hill's results were as follows: in the posttraumatic and paralytic group there were five successes, one limited result, and one failure; in the arthrogrypotic group there were five successes, one limited result, and two failures.[53]

The operative technique described by Carroll is as follows. The patient is placed in lateral position. A midline incision is made on the posterior aspect of the arm, beginning in its middle half and extending distally to a point lateral to the olecranon process; then the incision is carried over the subcutaneous surface of the shaft of the ulna for a distance of 5 cm. The subcutaneous tissue is divided and the wound flaps are mobilized. The ulnar nerve is identified and mobilized medially to protect it from injury. The intermuscular septum is exposed laterally. The triceps tendon is detached from its insertion with a long tail of periosteum. Then the triceps muscle is freed and mobilized proximally as far as its nerve supply permits. The motor branches of the radial nerve to the triceps enter the muscle in the interval between the lateral and medial heads as the radial nerve enters the musculospiral groove. The distal portion of the detached triceps is then sutured to itself to form a tube. Through a curvilinear incision in the antecubital fossa, the interval between the brachioradialis and the pronator teres is developed. With an Ober tendon passer, the triceps tendon is passed into the anterior wound subcutaneously, superficial to the radial nerve. With the elbow in 90 degrees of flexion and the forearm in full supination, the triceps tendon is either sutured to the biceps tendon or anchored to the radial tuberosity by a suture passed through a drill hole (Fig. 26–35). The wound is closed in routine fashion. An above-elbow cast is applied with the elbow in 90 degrees of flexion and full supination for four weeks, at which time immobilization is discontinued and active exercises are begun. Gravity provides extension to the elbow.[53]

Loss of active extension against gravity is a definite disadvantage of anterior transfer of the triceps. The operation should be restricted to exceptional cases in which restoration of elbow flexion is imperative and in which no other tendon transfer is possible. Another indication for anterior transfer of the triceps is that of brachial plexus injuries, in which simultaneous contraction of the triceps (the antagonistic muscle) occurs on active flexion of the elbow, and the action of the pectoralis major transfer is impaired. This simultaneous flexion-extension mass action can be successfully overcome by anterior transfer of the triceps into the flexor apparatus.

LATISSIMUS DORSI TRANSFER

Hovnanian described a method of transfer of the origin and belly of the latissimus dorsi muscle into the arm.[162] This is feasible because the nerve supply of the latissimus dorsi (the thoracodorsal nerve) is a long nerve (12 to 17 cm in length) and is highly mobile and easily identified; also, the blood supply of the latissimus dorsi muscle enters from a wide zone in its proximal third. Thus the latissimus dorsi can be mobilized without denervating or devascularizing the muscle. Active elbow flexion is restored by anchoring the origin of the latissimus dorsi muscle into the biceps tendon near the radial tuberosity; active extension of the elbow is obtained by suturing it to the olecranon (Fig. 26–36).

PARALYSIS OF THE TRICEPS BRACHII MUSCLE

Loss of active extension of the elbow due to paralysis of the triceps muscle seldom causes significant disability because the elbow will extend passively under the force of gravity. A strong triceps is not essential for crutch walking, provided good shoulder depressors are present. A triceps strap or band is added to the crutch, and with the elbow locked in slight hyperextension, the patient can ambulate quite well. If there is marked paralysis of both lower limbs and trunk, however, a functional triceps is desirable in order to lock the elbow in extension for daily activities such as arising from a bed or chair or reaching for objects overhead.

Various operative procedures have been devised to restore active extension of the elbow. Trapezius muscle transfer was used in 1930 by Lange, who detached it from the acromion and joined it by long silk sutures to the olecranon.[189] Ober and Barr described a technique for transferring the brachioradialis muscle by rerouting it at the elbow to a more posterior position.[246] Extensor carpi radialis longus muscle was added to the brachioradialis muscle transfer if greater strength was necessary. The transfer of flexor carpi radialis and ulnaris muscles was proposed by Hohmann.[158,159] Friedenberg transferred the biceps brachii for triceps paralysis.[106] The posterior part of the deltoid was proposed by d'Aubigné.[5] The latissimus dorsi was used for transfer to restore elbow extension by Hohmann,[158,159] Lange,[189] Harmon,[139] Schottstaedt and associates,[283] Hovnanian,[162] and duToit and Levy.[92]

The relative merits and shortcomings of the various procedures are not discussed here as the author has had no personal experience with them. The Hovnanian transfer of the origin of the latissimus dorsi to the olecranon, leaving its insertion intact, is recommended, as the procedure is physiologically sound and has been successful in the author's experience.

Text continued on page 1424

Pectoralis Major Transfer for Paralysis of Elbow Flexors

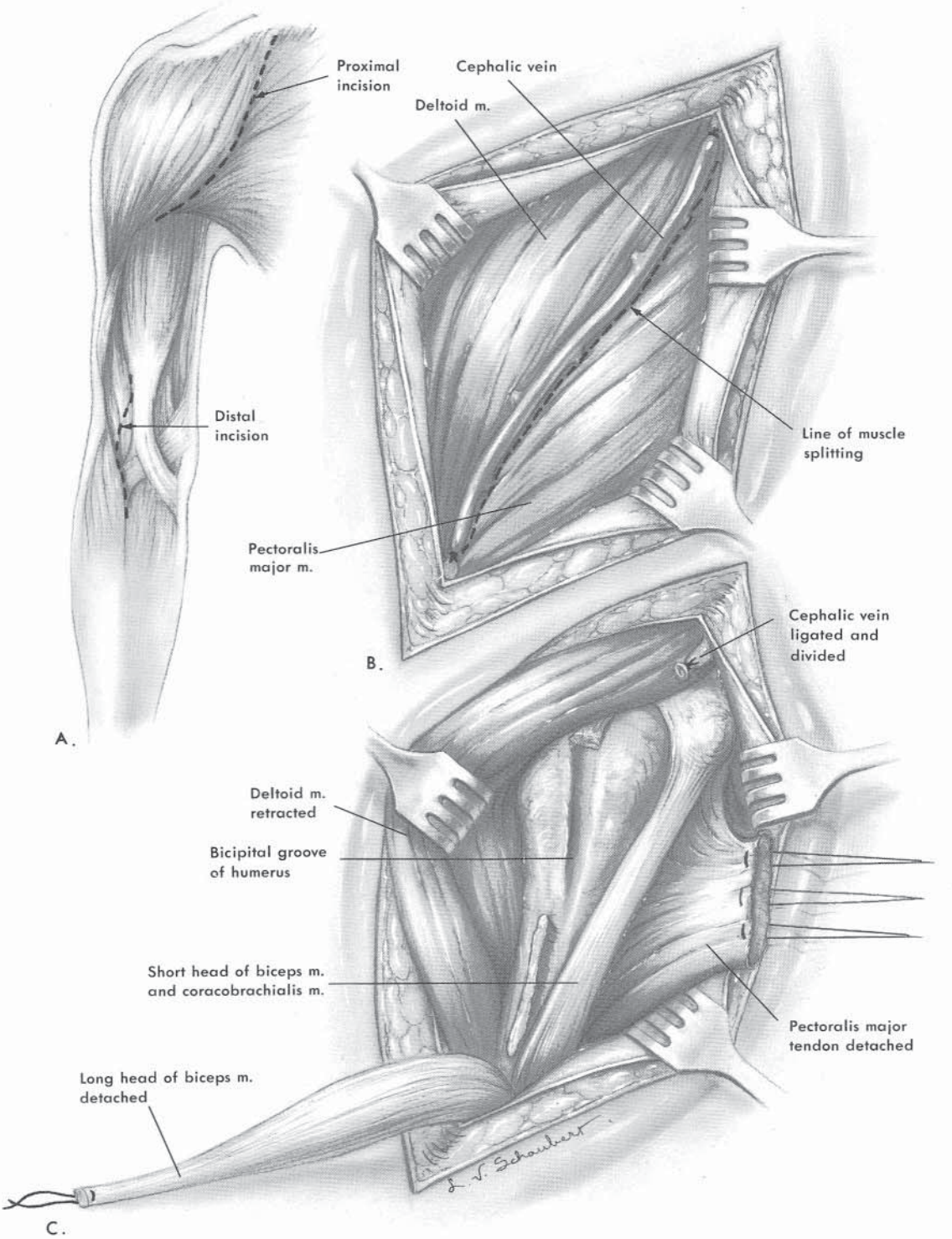
OPERATIVE TECHNIQUE

A, The patient is positioned supine with the upper limb supported on a hand table with the shoulder in 45 degrees of abduction and 30 degrees of external rotation. Two incisions are made, the first one following the deltopectoral groove and extending from the clavicle down to the junction of the upper and middle thirds of the arm. The second incision is centered over the anteromedial aspect of the elbow.

B, Through the first incision the subcutaneous tissue and deep fascia are divided, and the cephalic vein is ligated if necessary.

C, The pectoralis major tendon is identified and divided at its insertion, as close to the bone as possible. By blunt dissection, the muscle is mobilized from the chest wall toward the clavicle. The deltoid muscle is then retracted laterally and the tendon of the long head of the biceps is exposed running upward toward the shoulder joint. It is severed at the upper end of the bicipital groove and pulled distally into the wound.

PLATE 26–11. Pectoralis Major Transfer for Paralysis of Elbow Flexors



Pectoralis Major Transfer for Paralysis of Elbow Flexors *Continued*

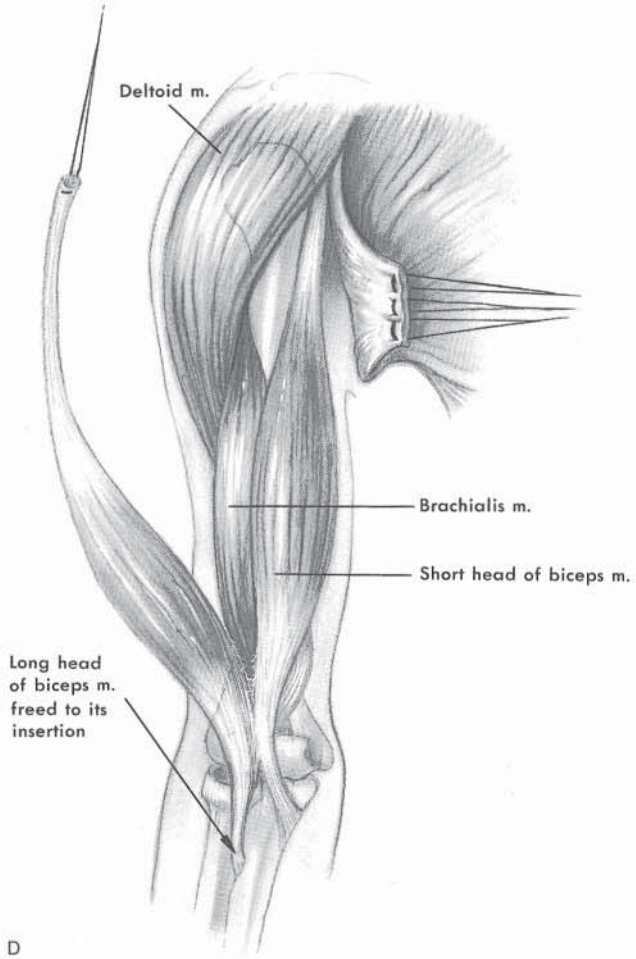
D, By blunt and sharp dissection, the muscle belly of the long head of the biceps is mobilized to the lowest third of the arm by freeing it from the short head. The vessels and nerves entering the muscle belly are divided and ligated as necessary. The tendon and muscle of the long head are delivered into the distal second incision and freed down to the tuberosity of the radius. Often, freeing the muscle from adhesions to the overlying fascia requires sharp dissection. After complete mobilization of the long head of the biceps by traction on its proximal end, the operator should be able to flex the elbow.

E, The long head of the biceps is pulled into the upper wound. Two slits are made in the tendon of the mobilized pectoralis major through which the tendon of the long head is passed, looped on itself, and brought down again into the distal wound. With the elbow acutely flexed, the proximal end of the tendon is sutured to its own tendon of insertion through a slit in the distal tendon. Silk sutures are also inserted at the level of the tendon of the pectoralis major. The incisions are then closed in routine manner. A plaster of Paris reinforced Velpeau bandage is applied with the elbow acutely flexed.

POSTOPERATIVE CARE

Plaster of Paris immobilization is continued for 3 weeks. At the end of this time active flexion and extension exercises of the elbow are started, first with gravity eliminated and then against gravity. A sling is used to protect the transferred tendon from stretching. Care should be taken to extend the elbow gradually so that active flexion above the right-angle position is maintained. Extension of the elbow is regained slowly.

PLATE 26–11. Pectoralis Major Transfer for Paralysis of Elbow Flexors



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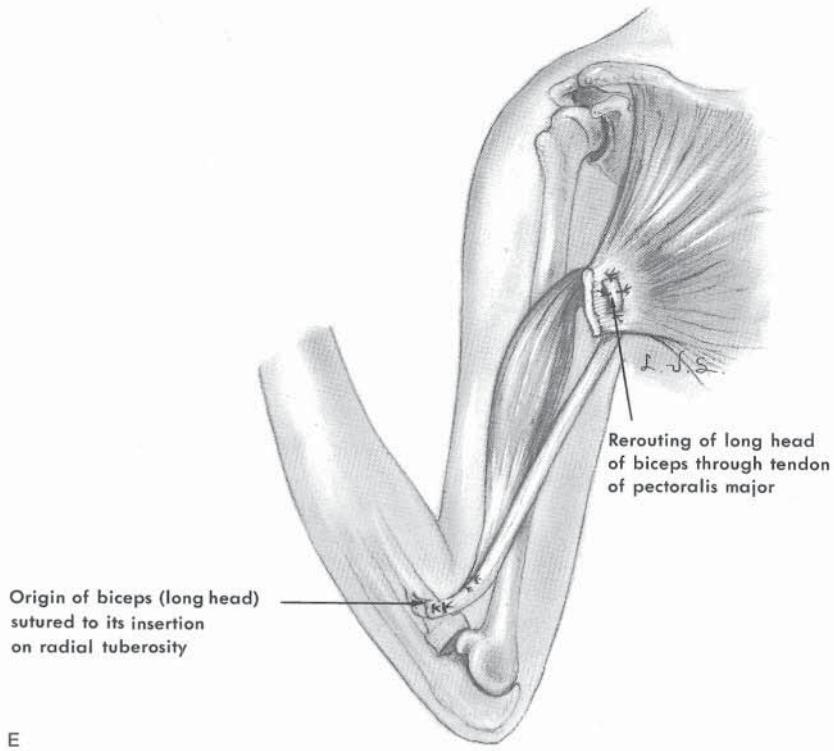

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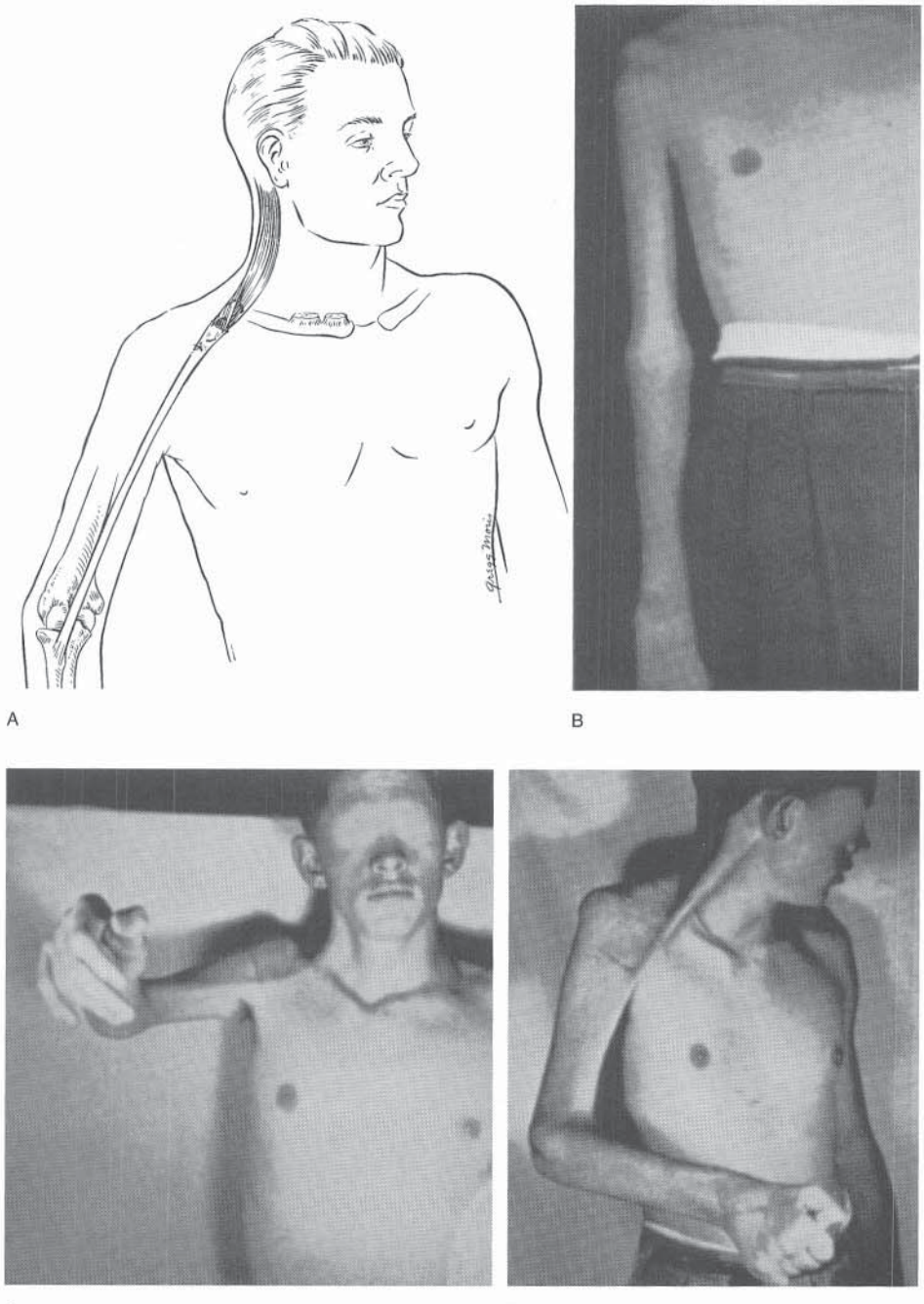

FIGURE 26–34 Bunnell's sternocleidomastoid transfer to restore active flexion of the elbow. **A,** Drawing showing the sternocleidomastoid muscle transfer. **B,** Preoperative photograph of patient with flail shoulder and elbow and partially paralyzed hand. The shoulder was fused, the sternocleidomastoid muscle was transferred, and the wrist was arthrodesed in functional position. **C** and **D,** Postoperative photographs showing the result. Function of the useless right upper limb was greatly improved. (From Bunnell S: Restoring flexion to the paralytic elbow. J Bone Joint Surg 1951; 33-A:569.)

The Forearm

Fixed supination and pronation contractures of the forearm are deformities in flaccid paralysis that require surgical treatment.

Supination contracture of the forearm is rare but disabling. It results from selective paralysis of the four muscles that originate from the medial epicondyle of the humerus (the pronator teres, flexor carpi ulnaris, palmaris longus, and flexor carpi radialis) in the presence of a strong biceps brachii muscle. Contracture of the interosseous membrane soon develops. With growth, and under the influence of unopposed action of the biceps brachii muscle, osseous changes take place, causing the radius to become curved and spiral around the ulna. If deformity remains uncorrected and there is still muscle imbalance, progressive fixed deformity will develop, with permanent shortening of the soft tissues, primarily the interosseous membrane, biceps brachii, and supinator muscles. The radius becomes markedly bowed and the radioulnar joints may subluxate. In children under 12 years of age, closed osteoclasis of the middle third of both bones of the forearm was recommended by Blount.[28] Because of recurrence of deformity, which can occur with further growth, he advised overcorrection. In two of the nine reported cases, osteoclasis was later repeated because of recurrence of supination contracture.

Zaoussis corrected the fixed supination deformity by open osteotomy near the tuberosity of the radius.[349] He

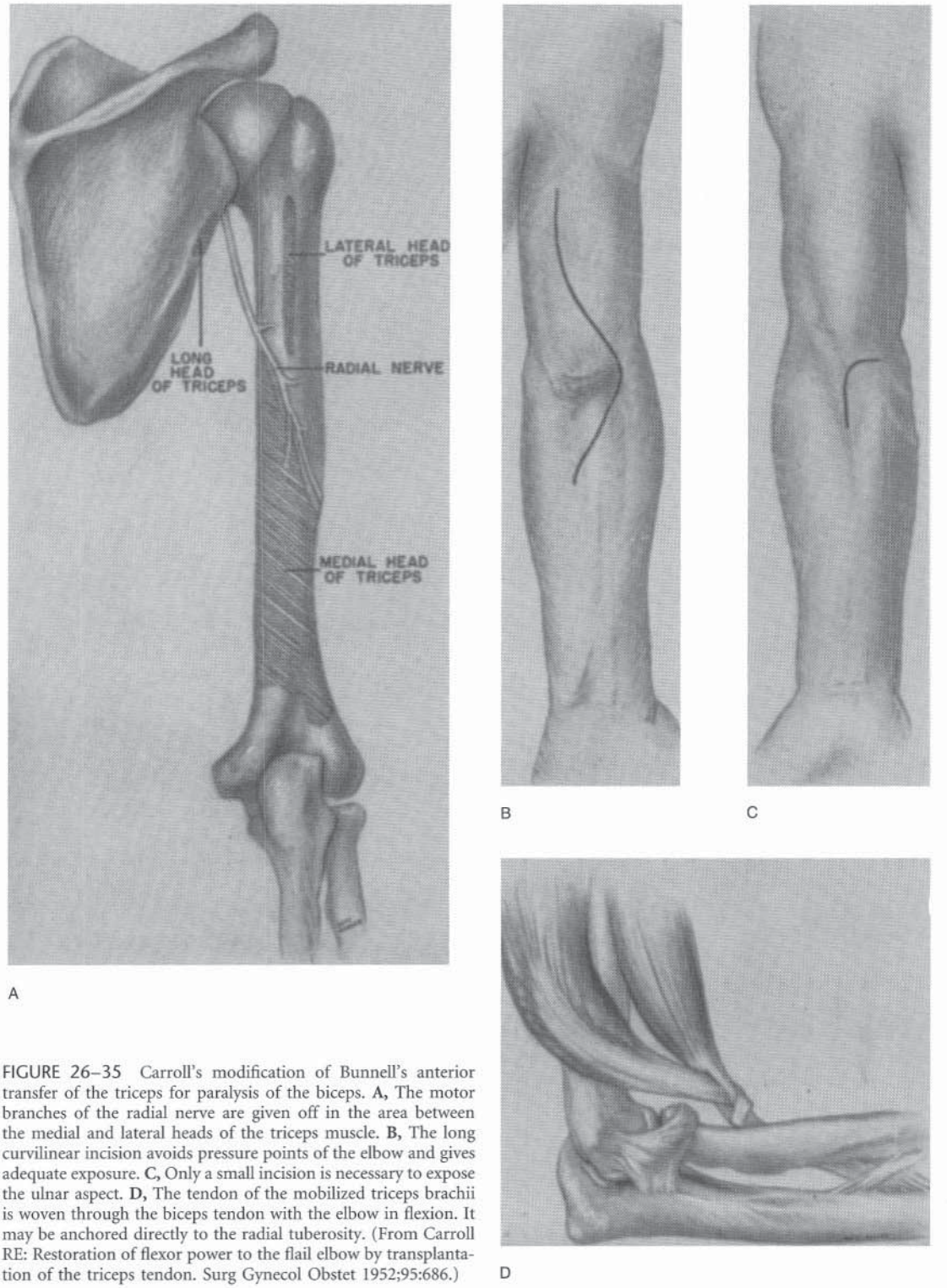

FIGURE 26–35 Carroll's modification of Bunnell's anterior transfer of the triceps for paralysis of the biceps. **A,** The motor branches of the radial nerve are given off in the area between the medial and lateral heads of the triceps muscle. **B,** The long curvilinear incision avoids pressure points of the elbow and gives adequate exposure. **C,** Only a small incision is necessary to expose the ulnar aspect. **D,** The tendon of the mobilized triceps brachii is woven through the biceps tendon with the elbow in flexion. It may be anchored directly to the radial tuberosity. (From Carroll RE: Restoration of flexor power to the flail elbow by transplantation of the triceps tendon. Surg Gynecol Obstet 1952;95:686.)

found more or less permanent "blocking" of the forearm rotation following surgery, but the synostosis of the proximal radius and ulna did not seem to impair the functional result. As internal fixation was not used by Zaoussis, angulation, displacement, and delayed union of the osteotomy occurred; however, these complications did not affect the cosmetic and functional result.

The transfer of the biceps brachii to the side of the radial tuberosity opposite its normal insertion was mentioned by Schottstaedt and associates in 1958.[283] A year later Grilli described the operative technique of rerouting the biceps tendon insertion to the radial side of the neck of the radius to convert its function from a supinator to that of pronator.[124]

Zancolli included surgical release of the contracted soft tissues in biceps transfer, especially the interosseous mem-

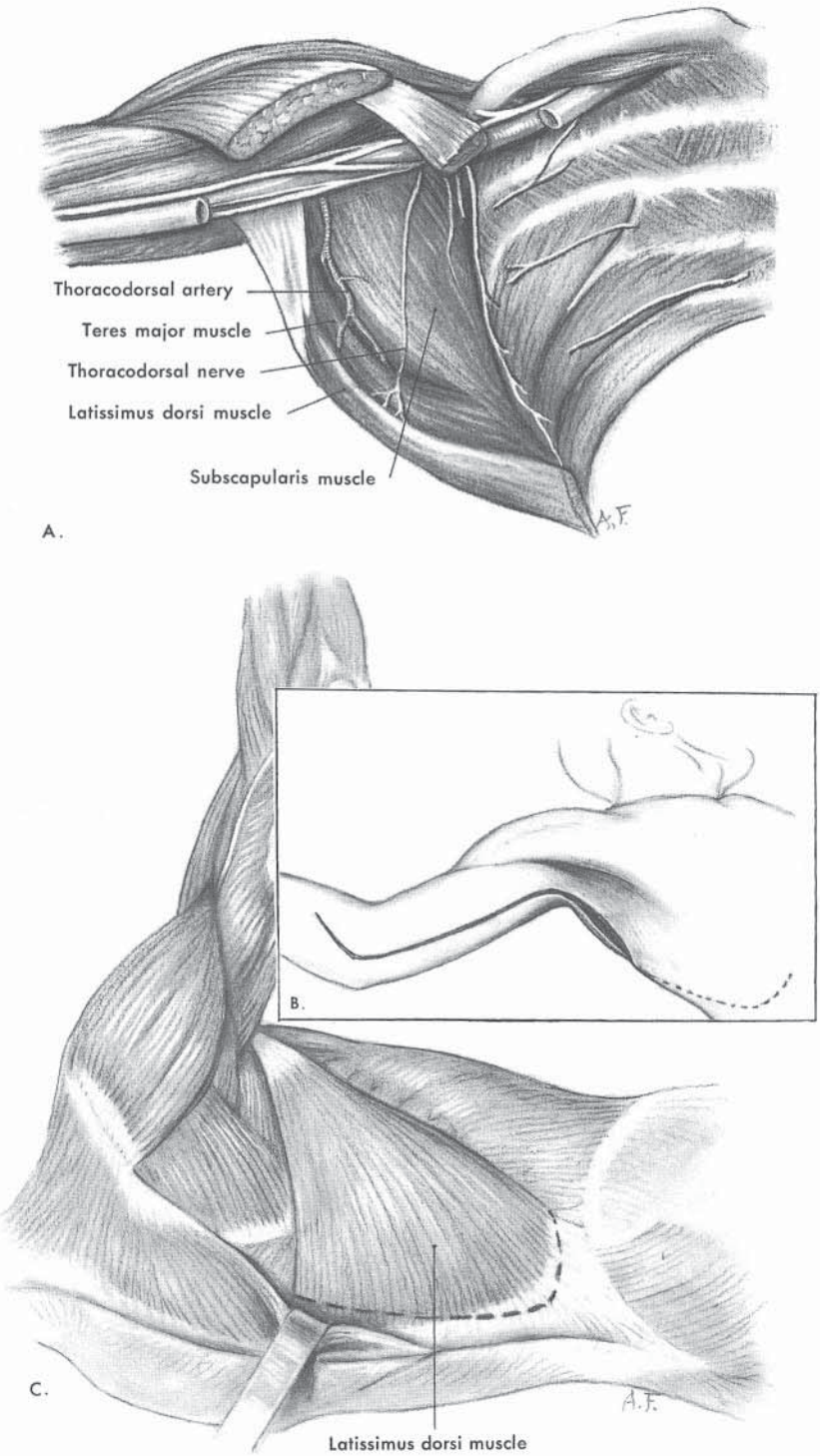

FIGURE 26–36 Latissimus dorsi transfer to restore flexion or extension at the elbow (Hovnanian operation). **A,** The anatomy of the axilla and the latissimus dorsi muscle. **B,** Skin incision used to restore elbow flexion. The lumbar extension of the skin incision is shown by *dotted lines.* **C,** The line of section of the latissimus dorsi muscle across its musculofascial portion inferiorly and its muscle fibers superiorly.

brane.[348] He reported satisfactory results in 14 patients with supination contracture of the forearm (eight patients had obstetric brachial plexus paralysis, four patients had poliomyelitis, and two patients had traumatic quadriplegia). Correction was maintained in all 14 patients. Active pronation (measuring 10 to 60 degrees and using the transferred biceps brachii muscle) was achieved in eight patients. Active supination (measuring 20 to 80 degrees) was retained in eight patients. The procedure permits a more normal anatomic relationship of the radius and ulna to develop, resulting in

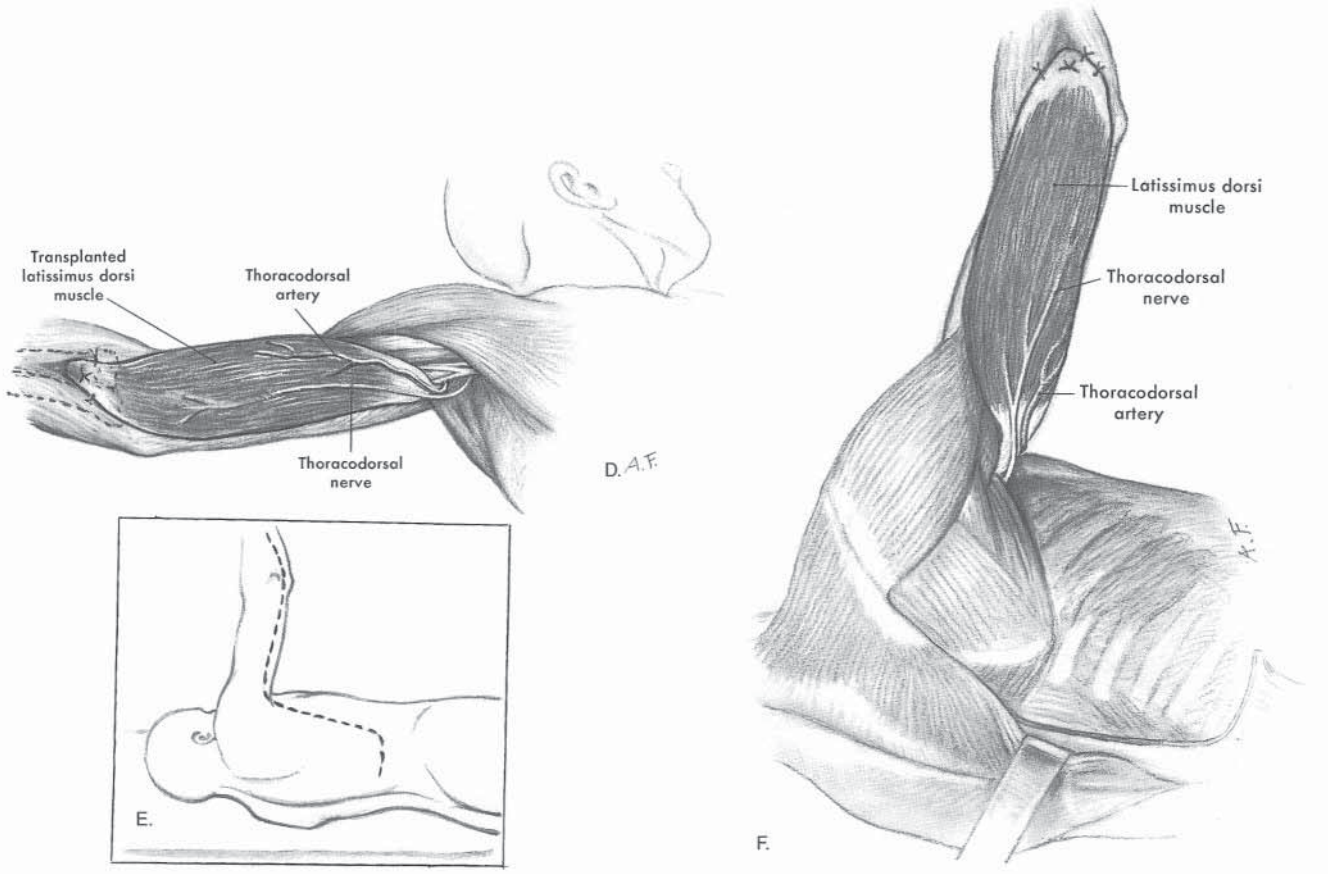

FIGURE 26–36 *Continued.* **D,** To restore elbow flexion, the belly and origin of the latissimus dorsi are transferred into the anteromedial aspect of the arm, and the origin is anchored into the biceps tendon near the radial tuberosity. **E,** Skin incision used to restore elbow extension. **F,** The latissimus dorsi muscle is transferred to the posterior aspect of the arm and anchored to the olecranon and triceps tendon. (From Hovnanian AP: Latissimus dorsi transplantation at the elbow. Ann Surg 1956;143:493.)

a nearly normal shape of the forearm. For details of the operative technique, the reader is referred to the original paper of Zancolli.[348]

REFERENCES

1. Abo W, Chiba S, Yamanaka T, et al: Paralytic poliomyelitis in a child with agammaglobulinemia. Eur J Pediatr 1979;132:11.
2. Anderson JG, Brandsma JW: Bilateral opponens replacement in post polio thenar paralysis, using different techniques: a case report. Hand 1983;15:221.
3. André FE: Control of poliomyelitis by vaccination in Belgium. Rev Infect Dis 1984;6(suppl 2):419.
4. Ansart MB: Pan-arthrodesis for paralytic flail foot. J Bone Joint Surg 1951;33-B:503.
5. d'Aubigné RM: Chirurgia orthopedique des paralysis. Paris, Masson & Cie, 1959.
6. Axer A: Intro-talus transposition of tendons for correction of paralytic valgus foot after poliomyelitis in children. J Bone Joint Surg 1960; 42-A:1119.
7. Axer A: Transposition of gluteus maximus, tensor fasciae latae and ilio-tibial band for paralysis of lateral abdominal muscles in children after poliomyelitis: a preliminary report. J Bone Joint Surg 1958; 40-B:644.
8. Baker AB, Cornwell S: Poliomyelitis: the spinal cord. Arch Pathol 1956;61:185.
9. Baker LD, Dodelin CD: Extra-articular arthrodesis of the subtalar joint (Grice procedure). JAMA 1958;168:1005.
10. Baptista Risi J Jr: The control of poliomyelitis in Brazil. Rev Infect Dis 1984;6(suppl 2):400.
11. Barr JS: Poliomyelitic hip deformity and the erector spinae transplantation. JAMA 1950;144:813.
12. Barr JS: Discussion. J Bone Joint Surg 1964;46-A:1402.
13. Barr JS, Freiberg JA, Colonna PC, et al: A survey of end-results on stabilization of the paralytic shoulder: report of the Research Committee of the American Orthopaedic Association. J Bone Joint Surg 1942;24:699.
14. Barr JS, Record EE: Arthrodesis of the ankle for correction of foot deformity. Surg Clin North Am 1947;27:1281.
15. Barr JS, Stinchfield AJ, Reidy JA: Sympathetic ganglionectomy and limb length in poliomyelitis. J Bone Joint Surg 1950;32-A:793.
16. Bass JW, Halstead SB, Fischer GW, et al: Oral polio vaccine: effect of booster vaccination one to 14 years after primary series. JAMA 1978;239:2252.
17. Basu SN: Paralytic poliomyelitis in children: some facts and figures from a hospital at Calcutta. Part I. Indian J Public Health 1985;29:175.
18. Basu SN: Paralytic poliomyelitis in children. Part II. Age, sex, and seasonal distribution. Indian J Public Health 1985;29:183.
19. Basu SN, Sokhey J: Prevalence of poliomyelitis in India. Indian J Pediatr 1984;51:515.
20. Bateman JE: The Shoulder and Environs. St. Louis, CV Mosby Co, 1954.
21. Bellier G, Carlioz H: [The prediction of growth in long bones in poliomyelitis]. Rev Chir Orthop 1979;65:373.
22. Benyi P: A modified Lambrinudi operation for drop foot. J Bone Joint Surg 1960;42-B:333.
23. Bergeisen GH, Bauman RJ, Gilmore RL: Neonatal paralytic poliomyelitis: a case report. Arch Neurol 1986;43:192.
24. Bernier RH: Improved inactivated poliovirus vaccine: an update. Pediatr Infect Dis 1986;5:289.
25. Bickel WH, Moe JH: Translocation of the peroneus longus tendon for paralytic calcaneus deformity of the foot. Surg Gynecol Obstet 1944;78:627.

26. Biesalski K, Mayer L: Die physiologische Sehnenverpflanzung, vol 14. Berlin, Julius Springer, 1916.
27. Bingold AC: Ankle and subtalar fusion by transarticular graft. J Bone Joint Surg 1956;38-B:862.
28. Blount WP: Osteoclasis for supination deformities in children. J Bone Joint Surg 1940;22:300.
29. Bodian D: Poliomyelitis: neuropathologic observations in relation to motor symptoms. JAMA 1947;134:1148.
30. Bradford EH, Lovett RW: Treatise on Orthopedic Surgery, p 486. London, Bailliere, Tindall & Cox, 1915.
31. Brewster AH: Countersinking the astragalus in paralytic feet. N Engl J Med 1933;209:71.
32. Britian HA: Architectural Principles in Arthrodesis, 2nd ed. Edinburgh, E & S Livingstone, 1952.
33. Broca JS, Chaturvedi SK, Mathur GM: Prevalence of residual polio paralysis in children of 5–15 years age group in Ajmer City. Indian J Public Health 1985;29:193.
34. Brockway A: An operation to improve abduction power of the shoulder in poliomyelitis. J Bone Joint Surg 1939;21:451.
35. Broderick TF, Reidy JA, Barr JS: Tendon transplantation in the lower extremity: a review of end results in poliomyelitis. II. Tendon transplantation at the knee. J Bone Joint Surg 1952;34-A:909.
36. Broms JD: Subtalar extra-articular arthrodesis: follow-up study. Clin Orthop 1965;42:139.
37. Brooks DM: Symposium on reconstructive surgery of paralyzed upper limb: tendon transplantation of the forearm and arthrodesis of the wrist. Proc R Soc Med 1949;42:838.
38. Brooks DM, Seddon HJ: Pectoral transplantation for paralysis of the flexors of the elbow: a new technique. J Bone Joint Surg 1959;41-B:36.
39. Brooks DM, Zaoussis A: Arthrodesis of the shoulder in reconstructive surgery of paralysis of the upper limb. J Bone Joint Surg 1959;41-B:207.
40. Buck-Gramcko H: Zur Technik der intraartikularen Schultergelenksarthrosese. Z Orthop 1959;91:198.
41. Bunnell S: Restoring flexion to the paralytic elbow. J Bone Joint Surg 1951;33-A:566.
42. Burman M: Paralytic supination of the forearm. J Bone Joint Surg 1956;38-A:303.
43. Caldwell GD: Correction of paralytic footdrop by hemigastrocsoleus transplant. Clin Orthop 1958;11:81.
44. Caldwell GD: Transplantation of the biceps femoris to the patella by the medial route in poliomyelitic quadriceps paralysis. J Bone Joint Surg 1955;37-A:347.
45. Calmes SH: Memories of polio. Arch Intern Med 1984;144:1273.
46. Campbell WC: End results of operation for correction of drop-foot. JAMA 1925;85:1927.
47. Campbell WC: An operation for the correction of "drop-foot." J Bone Joint Surg 1923;5:815.
48. Carayon A, Bourrel P, Bourges M, et al: Dual transfer of the posterior tibial and flexor digitorum longus tendons for drop foot: report of thirty-one cases. J Bone Joint Surg 1967;49-A:144.
49. Carmack JC, Hallock H: Tibiotarsal arthrodesis after astragalectomy: a report of eight cases. J Bone Joint Surg 1947;29:476.
50. Carroll RE: Restoration of elbow flexion by transplantation of the sternocleidomastoid muscle. In Proceedings of the American Society of Surgery of the Hand. J Bone Joint Surg 1962;44-A:1039.
51. Carroll RE: Restoration of flexor power to the flail elbow by transplantation of the triceps tendon. Surg Gynecol Obstet 1952;95:685.
52. Carroll RE, Gartland JJ: Flexorplasty of the elbow: an evaluation of a method. J Bone Joint Surg 1953;35-A:706.
53. Carroll RE, Hill NA: Triceps transfer to restore elbow flexion. J Bone Joint Surg 1970;52-A:239.
54. CDSC report: Outbreak of poliomyelitis in Finland. Br Med J 1985;291:41.
55. Chambers EFS: Operation for correction of flexible flat feet in adolescents. West J Surg 1946;54:77.
56. Charnley J: Compression Arthrodesis. London, E & S Livingstone, 1953.
57. Charnley J: Compression arthrodesis of the ankle and shoulder. J Bone Joint Surg 1951;33-B:180.
58. Charnley J, Houston JK: Compression arthrodesis of the shoulder. J Bone Joint Surg 1964;46-B:614.
59. Chaves JP: Pectoralis minor transplant for paralysis of the serratus anterior. J Bone Joint Surg 1951;33-B:228.
60. Chen CJ, Lin TM, You SL: Epidemiological aspects of a poliomyelitis outbreak in Taiwan, 1982. Ann Acad Med Singapore 1984;13:149.
61. Chigot PL, Sananes P: Arthrodese de Grice: variente technique. Rev Chir Orthop 1965;51:53.
62. Cholmeley JA: Elmslie's operation for the calcaneus foot. J Bone Joint Surg 1951;33-B:228.
63. Chuinard EG, Peterson RE: Distraction-compression bone-graft arthrodesis of the ankle: a method especially applicable for children. J Bone Joint Surg 1963;45-A:481.
64. Clark JMP: Reconstruction of biceps brachii by pectoral muscle transplantation. Br J Surg 1946;34:180.
65. Clark JMP, Axer A: A muscle-tendon transportation for paralysis of the lateral abdominal muscles in poliomyelitis. J Bone Joint Surg 1956;38-B:475.
66. Cleveland M: Operative fusion of the unstable or flail knee due to anterior poliomyelitis: a study of late results. J Bone Joint Surg 1932;14:525.
67. Clippinger FW Jr, Irwin CE: The opponens transfer: analysis of end results. South Med J 1962;55:33.
68. Close JR, Todd FN: The phasic activity of the muscles of the lower extremity and the effects of tendon transfer. J Bone Joint Surg 1959;41-A:189.
69. Codivilla A: Meine Erfahrungen über Schnenverpflanzungen. Z Orthop Chir 1904;12:221.
70. Conner AN: The treatment of flexion contractures of the knee in poliomyelitis. J Bone Joint Surg 1970;52-B:138.
71. Coonrad RW, Irwin CE, Gucker T III, et al: The importance of plantar muscles in paralytic varus feet: the results of treatment by neurectomy and myotenotomy. J Bone Joint Surg 1956;38-A:563.
72. Cravener EK: Device for overcoming non-bony flexion contractures of the knee. J Bone Joint Surg 1930;12:437.
73. Crego CH Jr, Fischer FJ: Transplantation of the biceps femoris for the relief of quadriceps femoris paralysis in residual poliomyelitis. J Bone Joint Surg 1931;13:515.
74. Crego CH Jr, McCarroll HR: Recurrent deformities in stabilized paralytic feet: a report of 1100 consecutive stabilizations in poliomyelitis. J Bone Joint Surg 1938;20:609.
75. Cross AB: Crawling patterns in neglected poliomyelitis in the Solomon Islands. J Bone Joint Surg 1977;59-B:428.
76. Dalakas MC, Elder G, Hallett M, et al: A long-term follow-up study of patients with post-poliomyelitis neuromuscular symptoms. N Engl J Med 1986;314:959.
77. Davidson WD: Traumatic deltoid paralysis treated by muscle transplantation. JAMA 1936;106:2237.
78. Davis GG: The treatment of hollow foot (pes cavus). Am J Orthop Surg 1913;11:231.
79. Davis GG: Wedge-shaped resection of the foot for the relief of old cases of varus. NY J Med 1892;56:379.
80. Davis JB, Cotrell GW: A technique for shoulder arthrodesis. J Bone Joint Surg 1962;44-A:657.
81. Dehne E, Hall RM: Active shoulder motion in complete deltoid paralysis. J Bone Joint Surg 1959;41-A:745.
82. Dekking F: Poliomyelitis in the Netherlands. Ned Tijdschr Geneeskd 1978;122:1142.
83. Den Hartog JG: Hip and knee flexion contracture after poliomyelitis. South Med J 1980;73:694.
84. Dickson FD: Fascial transplants in paralytic and other conditions. J Bone Joint Surg 1937;19:405.
85. Dickson FD: An operation for stabilizing paralytic hips: a preliminary report. J Bone Joint Surg 1927;9:1.
86. Dickson FD, Diveley RL: Operation for correction of mild claw foot, the results of infantile paralysis. JAMA 1926;87:1275.
87. Drew AJ: The late results of arthrodesis of the foot. J Bone Joint Surg 1951;33-B:496.
88. Dunn N: Stabilizing operations in the treatment of paralytic deformities of the foot. Proc R Soc Med 1922;15:15.
89. Dunn N: Suggestion based on ten years' experience of arthrodesis of the tarsus in the treatment of deformities of the foot. In Robert Jones Birthday Volume, p 395. London, Oxford University Press, 1928.
90. Dunne JW, Harper CG, Hilton JM: Sudden infant death syndrome caused by poliomyelitis. Arch Neurol 1984;41:775.
91. Durman DC: An operation for paralysis of the serratus anterior. J Bone Joint Surg 1945;27:380.
92. duToit GT, Levy SJ: Transposition of latissimus dorsi for paralysis of the triceps brachii: report of a case. J Bone Joint Surg 1967;49-B:135.
93. Eaton GO: Results of abdominal stabilization. South Med J 1941; 34:443.

94. Eberle CF: Pelvic obliquity and the unstable hip after poliomyelitis. J Bone Joint Surg 1982;64-B:300.
95. Elkins EC, Janes JM, Henderson ED, et al: Peroneal translocation for paralysis of plantar flexor muscles. Surg Gynecol Obstet 1956;102:469.
96. Elmslie RL: In Turner GG (ed): Modern Operative Surgery, 2nd ed. London, Cassell & Co, 1934.
97. Emmel HE, LeCoco JR: Hamstring transplant for the prevention of calcaneocavus foot in poliomyelitis. J Bone Joint Surg 1958;40-A:911.
98. Fernandez de Castro J: Mass vaccination against poliomyelitis in Mexico. Rev Infect Dis 1984;6:397.
99. Fitchet SM: "Flexion deformity" of the hip and the lateral intermuscular septum. N Engl J Med 1933;209:74.
100. Fitzgerald FP, Seddon HJ: Lambrinudi's operation for drop-foot. Br J Surg 1937;25:283.
101. Flexner S, Lewis PA: Transmission of acute poliomyelitis to monkeys. JAMA 1909;53:1639.
102. Flint MH, MacKenzie IC: Anterior laxity of the ankle: a cause of recurrent paralytic drop foot deformity. J Bone Joint Surg 1962;44-B: 377.
103. Forbes AM: The tensor fasciae femoris as a cause of deformity. J Bone Joint Surg 1928;10:579.
104. Fried A, Hendel C: Paralytic valgus deformity of the ankle: replacement of the paralyzed tibialis posterior by the peroneus longus. J Bone Joint Surg 1957;39-A:921.
105. Friedenberg ZB: Arthrodesis of the tarsal bones: a study of failure of fusion. Arch Surg 1948;57:162.
106. Friedenberg ZB: Transposition of the biceps brachii for triceps weakness. J Bone Joint Surg 1954;36-A:656.
107. Gaebler JW, Kleiman MB, French ML, et al: Neurologic complications in oral polio vaccine recipients. J Pediatr 1986;108:878.
108. Gill AB: A new operation for arthrodesis of the shoulder. J Bone Joint Surg 1931;13:287.
109. Gill AB: An operation to make a posterior bone block at the ankle to limit foot-drop. J Bone Joint Surg 1933;15:166.
110. Girard PM: Ankle joint stabilization with motion. J Bone Joint Surg 1935;17:802.
111. Goldner JL: Paralytic equinovarus deformities of the foot. South Med J 1949;42:83.
112. Goldner JL, Irwin CE: Paralytic deformities of the foot. Instruct Course Lect 1948;5.
113. Goldthwait JE: The direct transplantation of muscles in the treatment of paralytic deformities: five cases of transplantation of the sartorius muscle. Boston Med Surg J 1897;137:489.
114. Goldthwait JE: An operation for the stiffening of the ankle joint in infantile paralysis. Am J Orthop Surg 1908;5:271.
115. Goldthwait JE: Tendon transplantation in the treatment of deformities resulting from infantile paralysis. Boston Med Surg J 1895;133:447.
116. Green WT: Tendon transplantation in rehabilitation. JAMA 1957; 163:1235.
117. Green WT, Grice DS: The management of calcaneus deformity. Instruct Course Lect 1956;13:135.
118. Green WT, Grice DS: The management of chronic poliomyelitis. Instruct Course Lect 1952;9.
119. Green WT, Grice DS: The surgical correction of the paralytic foot. Instruct Course Lect 1953;10:343.
120. Green WT, Grice DS: The treatment of poliomyelitis: acute and convalescent stages. Instruct Course Lect 1951;13:261.
121. Grice DS: An extra-articular arthrodesis of the subastragalar joint for correction of paralytic flat feet in children. J Bone Joint Surg 1952; 34-A:927.
122. Grice DS: Further experience with extra-articular arthrodesis of the subtalar joint. J Bone Joint Surg 1955;36-A:246.
123. Grice DS: The role of subtalar fusion in the treatment of valgus deformities of the feet. Instruct Course Lect 1959;16:127.
124. Grilli FP: I1 trapianto del bicipite brachiale in funzione pronatoria. Arch Putti 1959;12:359.
125. Grist NR: Poliomyelitis vaccine precautions [editorial]. Br Med J 1983;287:1823.
126. Grist NR, Bell EJ: Paralytic poliomyelitis and nonpolio enteroviruses: studies in Scotland. Rev Infect Dis Suppl 1984;2:385.
127. Groves EWH: Some contributions to the reconstructive surgery of the hip. Br J Surg 1927;14:486.
128. Guidal P, Sodeman T: Results of 256 tri-articular arthrodeses of the foot in sequelae of infantile paralysis. Acta Orthop Scand 1930–1931;1:199.
129. Guillozet N: Perthes disease after poliomyelitis: recognition and management of aseptic capital femoral necrosis. Clin Pediatr 1981;20:19.
130. Gunn DR, Molesworth BD: The use of tibialis posterior as a dorsiflexor. J Bone Joint Surg 1957;39-B:674.
131. Haas SL: Correction of extreme flexion contracture of the knee joint. J Bone Joint Surg 1938;20:839.
132. Haas SL: The treatment of permanent paralysis of the deltoid muscle. JAMA 1935;104:99.
133. Hajar MM, Zeid AS, Saif MA, et al: Prevalence, incidence, and epidemiological features of poliomyelitis in the Yemen Arab Republic. Bull WHO 1983;61:353.
134. Hallgrimsson S: Studies on reconstructive and stabilizing operations on the skeleton of the foot, with special reference to subastragalar arthrodesis in treatment of foot deformities following infantile paralysis. Acta Chir Scand 1944;88(suppl 78):1.
135. Hallock H: Arthrodesis of the hip for instability and pain in poliomyelitis. J Bone Joint Surg 1950;2-A:904.
136. Hallock H: Hip arthrodesis in poliomyelitis. Bull NY Hosp 1958;2:18.
137. Hallock H: Surgical stabilization of dislocated paralytic hips: end-results study. Surg Gynecol Obstet 1942;75:742.
138. Hamsa WR: Panastragaloid arthrodesis: a study of end-results in eighty-five cases. J Bone Joint Surg 1936;18:732.
139. Harmon PH: Anterior transplantation of the posterior deltoid for shoulder palsy and dislocation in poliomyelitis. Surg Gynecol Obstet 1947;84:117.
140. Harmon PH: Surgical reconstruction of the paralytic shoulder by multiple muscle transplantation. J Bone Joint Surg 1950;32-A:583.
141. Harmon PH: Technic of utilizing latissimus dorsi muscle in transplantation for triceps palsy. J Bone Joint Surg 1949;31-A:409.
142. Hart VL: Arthrodesis of the foot in infantile paralysis. Surg Gynecol Obstet 1937;64:794.
143. Hart VL: Corrective cast for flexion-contracture deformity of the knee. J Bone Joint Surg 1934;16:970.
144. Hart VL: Lambrinudi operation for drop-foot. J Bone Joint Surg 1940;22:937.
145. Henderson MS: Reconstructive surgery in paralytic deformities of the lower leg. J Bone Joint Surg 1929;11:810.
146. Henry AH: Extensile Exposure Applied to Limb Surgery, 2nd ed. Baltimore, Williams & Wilkins, 1957.
147. Henry AH: An operation for slinging a dropped shoulder. Br J Surg 1927;15:95.
148. Herndon CH: Tendon transplantation at the knee and foot. Instruct Course Lect 1961;18.
149. Herndon CH, Strong JM, Heyman CH: Transposition of the tibialis anterior in the treatment of paralytic talipes calcaneus. J Bone Joint Surg 1956;38-A:751.
150. Herzmark MH: Traumatic paralysis of the serratus anterior relieved by transplantation of the rhomboidei. J Bone Joint Surg 1951;33-A:235.
151. Heydarian K, Akbarnia BA, Jabalameli M, et al: Posterior capsulotomy for the treatment of severe flexion contracture of the knee. J Pediatr Orthop 1984;4:700.
152. Heyman CH: A method for the correction of paralytic genu recurvatum: report of a bilateral case. J Bone Joint Surg 1924;6:689.
153. Heyman CH: Operative treatment of paralytic genu recurvatum. J Bone Joint Surg 1947;29:644.
154. Heyman CH: Operative treatment of paralytic genu recurvatum. J Bone Joint Surg 1962;44-A:1246.
155. Hildebrandt A: Uber eine neue Methode der Muskel-transplantation. Arch Klin Chir 1906;78:75.
156. Hipps HE: Clinical significance of certain microscopic changes in muscles of anterior poliomyelitis. J Bone Joint Surg 1942;24:68.
157. Hogshead HP, Ponseti IV: Fascia lata transfer to the erector spinae for the treatment of flexion-abduction contractures of the hip in patients with poliomyelitis and meningomyelocele: evaluation of results. J Bone Joint Surg 1964;46-A:1389.
158. Hohmann G: Eratz des gelahmten Biceps brachii durch den Pectoralis major. Munchen Med Wochenschr 1918;65:1240.
159. Hohmann G: Operative Verwertung erhaltener Muskeln bei Kinderlahmung. Munchen Med Wochenschr 1950;92:249.
160. Hoke M: An operation for stabilizing paralytic feet. Am J Orthop Surg 1921;3:494.
161. Holmdahl HC: Astragalectomy as a stabilizing operation for foot paralysis following poliomyelitis: results of a follow-up investigation of 153 cases. Acta Orthop Scand 1956;25:207.

162. Hovnanian AP: Latissimus dorsi transplantation for loss of flexion or extension at the elbow. Ann Surg 1956;143:493.
163. Hsu LC, O'Brien JP, Yau ACMC, et al: Batchelor's extra-articular subtalar arthrodesis: a report on sixty-four procedures in patients with poliomyelitis deformities. J Bone Joint Surg 1977;58-A:243.
164. Hunt JC, Brooks AL: Subtalar extra-articular arthrodesis for correction of paralytic valgus deformity of the foot: evaluation of forty-four procedures with particular reference to associated tendon transference. J Bone Joint Surg 1965;47-A:1310.
165. Hunt WS Jr, Thompson HA: Pantalar arthrodesis: a one-stage operation. J Bone Joint Surg 1954;36-A:349.
166. Inclan A: End results in physiological blocking of flail joints. J Bone Joint Surg 1949;31-A:748.
167. Ingersoll RE: Transplantation of peroneus longus to anterior tibial insertion in poliomyelitis. Surg Gynecol Obstet 1948;86:717.
168. Ingram AJ, Hundley JM: Posterior bone block of the ankle for paralytic equinus: an end-result study. J Bone Joint Surg 1951;33-A:679.
169. Irwin CE: Genu recurvatum following poliomyelitis: controlled method of operative correction. JAMA 1942;120:277.
170. Irwin CE: The iliotibial band: its role in producing deformity in poliomyelitis. J Bone Joint Surg 1949;31-A:141.
171. Irwin CE: Subtrochanteric osteotomy in poliomyelitis. JAMA 1947; 133:231.
172. Irwin CE: Transplants to the thumb to restore function of opposition: end results. South Med J 1942;35:257.
173. Irwin CE, Eyler DL: Surgical rehabilitation of the hand and forearm disabled by poliomyelitis. J Bone Joint Surg 1951;33-A:679.
174. Johnson EW Jr: Contractures of the iliotibial band. Surg Gynecol Obstet 1953;96:509.
175. Johnson EW Jr: Results of modern methods of treatment of poliomyelitis. J Bone Joint Surg 1945;27:223.
176. Jones GB: Paralytic dislocation of the hip. J Bone Joint Surg 1954; 36-B:375.
177. Jones GB: Paralytic dislocation of the hip. J Bone Joint Surg 1962; 44-B:573.
178. Kettelkamp DB, Larson CB: Evaluation of the Steindler flexorplasty. J Bone Joint Surg 1963;45-A:513.
179. Key JA: Arthrodesis of the shoulder by means of osteoperiosteal grafts. Surg Gynecol Obstet 1930;50:468.
180. Khuri Bulos N, Melnick JL, Hatch MH, et al: The paralytic poliomyelitis epidemic of 1978 in Jordan: epidemiological implications. Bull WHO 1984;62:83.
181. King BB: Ankle fusion for the correction of paralytic drop foot and calcaneus deformity. Arch Surg 1940;40:90.
182. Kleinberg S: The transplantation of the adductor longus in its entirety to supplement the quadriceps femoris. Bull Hosp Jt Dis 1957;18:117.
183. Kreuscher PH: The substitution of the erector spinae for paralyzed gluteal muscles. Surg Gynecol Obstet 1925;40:593.
184. Kuhlmann RF, Bell JF: A clinical evaluation of tendon transplantation for poliomyelitis affecting the lower extremities. J Bone Joint Surg 1952;34-A:915.
185. Kumar K, Kapahtia NK: The pattern of muscle involvement in poliomyelitis of the upper limb. Int Orthop 1986;10:11.
186. Lambrinudi C: A method of correcting equinus and calcaneus deformities at the sub-astragaloid joint. Proc R Soc Med 1933;26:788.
187. Lambrinudi C: New operation on drop-foot. Br J Surg 1927;15:193.
188. Landsteiner K, Popper E: Uberstragung der Poliomyelitis acuta auf Affen. Z Immunitatsforsch Exp Ther Orig 1909;2:377.
189. Lange F: Die epidemische Kinderlahmung. Munich, JF Lehmann, 1930.
190. Leavitt DG: Subastragaloid arthrodesis for the os calcis type of flat foot. Am J Surg 1943;59:501.
191. LeCoeur P: Procedes de restauration de la flexion du coude paralytique. Rev Chir Orthop 1953;39:655.
192. Legg AT: Tensor fasciae femoris transplantation in cases of weakened gluteus medius. N Engl J Med 1933;209:61.
193. Legg AT: Transplantation of tensor fasciae femoris in cases of weakened gluteus medius. JAMA 1923;80:242.
194. Legg AT, Merrill JT: Physical Therapy in Infantile Paralysis. Hagerstown, MD, WF Prior Co, 1932.
195. Leong JC, Alade CO, Fang D: Supracondylar femoral osteotomy for knee flexion contracture resulting from poliomyelitis. J Bone Joint Surg 1982;64-B:198.
196. Leong JC, Wilding K, Mok CK, et al: Surgical treatment of scoliosis following poliomyelitis: a review of one hundred and ten cases. J Bone Joint Surg 1981;63-A:726.
197. Lewis DD: Trapezius transplantation in the treatment of deltoid paralysis. JAMA 1910;55:2211.
198. Liebolt FL: Pantalar arthrodesis in poliomyelitis. Surgery 1939;6:31.
199. Lipscomb PR, Sanchez JJ: Anterior transplantation of the posterior tibial tendon for persistent palsy of the common peroneal nerve. J Bone Joint Surg 1961;43-A:60.
200. Lorthioir J: Huit cas d'arthrodese du pied avec extirpation temporaire de l'astragale. J Chir Ann Soc Belge Chir 1911;11:184.
201. Lovett RW: The Treatment of Infantile Paralysis. Philadelphia, P Blakiston's Son & Co, 1916.
202. Lowman CL: Abdominal Fascial Transplants. Los Angeles, n. p., 1954.
203. Lowman CL: Fascial transplants in paralysis of abdominal and shoulder girdle muscles. Instruct Course Lect 1957;14:300.
204. Lowman CL: Fascial transplants in relation to muscle function. J Bone Joint Surg 1963;45-A:199.
205. Lundbeck H: International symposium of poliomyelitis control: strategies for control. A discussion. Rev Infect Dis Suppl 1984;2:483.
206. MacAusland WR, MacAusland AR: Astragalectomy (the Whitman operation) in paralytic deformities of the foot. Ann Surg 1924;80:861.
207. MacKenzie IG: Lambrinudi's arthrodesis. J Bone Joint Surg 1959; 41-B:738.
208. Macnicol MF, Catto AM: Twenty-year review of tibial lengthening for poliomyelitis. J Bone Joint Surg 1982;64-B:607.
209. Magoflin RL, Lennette EH, Hollister AC Jr, et al: An etiology study of clinical paralytic poliomyelitis. JAMA 1961;175:269.
210. Makin M: Tibiofibular relationship in paralyzed limbs. J Bone Joint Surg 1965;47-B:500.
211. Makin M, Yossipovich Z: Translocation of the peroneus longus in the treatment of paralytic pes calcaneus: a follow-up study of thirty-three cases. J Bone Joint Surg 1966;48-A:1541.
212. Malvarez O: Arthrodesis subastragalina extraarticular en el pie valgo pronado pavalitico: arthrodesis minima. Estudio de 87 casos. Rev Ortop Traum Lat Am 1957;2:251.
213. Marek FM, Schein AJ: Aseptic necrosis of the astragalus following arthrodesing procedures of the tarsus. J Bone Joint Surg 1945;27:587.
214. Mayer L: Fixed paralytic obliquity of the pelvis. J Bone Joint Surg 1931;13:1.
215. Mayer L: Operative reconstruction of the paralyzed upper extremity. J Bone Joint Surg 1939;21:377.
216. Mayer L: The physiologic method of tendon transplants: reviewed after forty years. Instruct Course Lect 1956;13:116.
217. Mayer L: The physiological method of tendon transplantation. I. Historical. Anatomy and physiology of tendons. Surg Gynecol Obstet 1916;22:182.
218. Mayer L: The physiological method of tendon transplantation. II. Operative technique. Surg Gynecol Obstet 1916;22:298.
219. Mayer L: The physiological method of tendon transplantation. III. Experimental and clinical experiences. Surg Gynecol Obstet 1916; 221:472.
220. Mayer L: The significance of the iliocostal fascial graft in the treatment of paralytic deformities of the trunk. J Bone Joint Surg 1944;26:257.
221. Mayer L: Transplantation of the trapezius for paralysis of the abductors of the arm. J Bone Joint Surg 1927;9:412.
222. Mayer L, Green W: Experience with the Steindler flexorplasty at the elbow. J Bone Joint Surg 1954;36-A:775.
223. Mayer TR: Duration of vaccine-induced poliomyelitis immunity. J Fam Pract 1984;19:385.
224. Maynard FM: Post-polio sequelae: differential diagnosis and management. Orthopedics 1985;8:857.
225. McFarland B: Paralytic instability of the foot [editorial]. J Bone Joint Surg 1951;33-B:493.
226. Medin O: L'état aigu de la paralysie infantile. Arch Med Enf 1898;1:257.
227. Mertens T, Schurmann W, Kruppenbacher JP, et al: Two cases of vaccine-induced poliomyelitis. Acta Paediatr Scand 1984;73:133.
228. Miller OL: Paralytic knee fusions. South Med J 1927;20:782.
229. Miller OL: Surgical management of pes calcaneus. J Bone Joint Surg 1936;18:169.
230. Mitchell GP: Posterior displacement of poliomyelitis scoliosis. J Bone Joint Surg 1977;59-B:233.
231. de Morais JC, Eduardo MB, Camargo MC, et al: Epidemiological course of poliomyelitis 1970–1981, in Sao Paulo Rev Paul Med 1982;99:34.
232. Mortens J, Gregersey P, Zachariae L: Tendon transplantation in the

foot after poliomyelitis in children. Acta Orthop Scand 1957–1958;27:153.
233. Mortens J, Pilcher MF: Tendon transplantation in the prevention of foot deformities after poliomyelitis in children. J Bone Joint Surg 1956;38-B:633.
234. Moses PD, Pereira SM, John TJ, et al: Poliovirus infection and Bell's palsy in children. Ann Trop Paediatr 1985;5:195.
235. Mustard WT: A follow-up study of iliopsoas transfer for hip instability. J Bone Joint Surg 1959;41-B:289.
236. Mustard WT: Iliopsoas transfer for weakness of the hip abductors: preliminary report. J Bone Joint Surg 1952;34-A:647.
237. Nicoladoni C: Nachtrag zum Pes calcaneus und zur Transplantation der Peronealschnen. Arch Klin Chir 1881;27:660.
238. Nieny K: Behandlung der Fussdeformation bei ausgedehnten Lahmungen. Arch Orthop Unfallchir 1905;3:60.
239. Nyholm K: Elbow flexorplasty in tendon transposition (an analysis of the functional results in 26 patients). Acta Orthop Scand 1963;33:30.
240. Ober FR: An operation for relief of paralysis of the gluteal maximus muscle. JAMA 1927;88:1063.
241. Ober FR: An operation to relieve paralysis of the deltoid muscle. JAMA 1932;99:2182.
242. Ober FR: Operative and postoperative treatment of infantile paralysis. N Engl J Med 1931;205:300.
243. Ober FR: The role of the iliotibial band and fascia lata as a factor in the causation of low back disabilities and sciatica. J Bone Joint Surg 1936;18:105.
244. Ober FR: Tendon transplantation in the lower extremity. N Engl J Med 1933;209:52.
245. Ober FR: Transplantation to improve the function of the shoulder joint and extensor function of the elbow joint. Lect Reconstr Surg 1944;2:274.
246. Ober FR, Barr JS: Brachioradialis muscle transposition for triceps weakness. Surg Gynecol Obstet 1938;67:105.
247. Paluska DJ, Blount WP: Ankle valgus after the Grice subtalar stabilization: the late evaluation of a personal series with a modified technic. Clin Orthop 1968;59:137.
248. Parekh PK: Flexion contractures of the knee following poliomyelitis. Int Orthop 1983;7:165.
249. Parsons DW, Seddon HJ: The results of operations for disorders of the hip caused by poliomyelitis. J Bone Joint Surg 1968;50-B:266.
250. Patterson RL, Parrish FF, Hathaway EN: Stabilizing operation on the foot: a study of the indications, techniques used and end results. J Bone Joint Surg 1950;32-A:1.
251. Pauker E: Correction of the outwardly rotated leg from poliomyelitis. J Bone Joint Surg 1959;41-B:70.
252. Peabody CW: Tendon transposition: an end-result study. J Bone Joint Surg 1938;20:193.
253. Peabody CW: Tendon transposition in the paralytic foot. Instruct Course Lect 1949;6:178.
254. Peabody CW, Draper G, Dochez AR: A Clinical Study of Acute Poliomyelitis. Monograph No. 4. New York, Rockefeller Institute of Medical Research, 1912.
255. Perry J, Fleming C: Polio: Long-term problems. Orthopedics 1985; 8:877.
256. Pollock JH, Carrell B: Subtalar extra-articular arthrodesis in the treatment of paralytic valgus deformities: a review of 112 procedures in 100 patients. J Bone Joint Surg 1964;46-A:533.
257. Pollock LJ: Accessory muscle movement in deltoid paralysis. JAMA 1922;79:526.
258. Putti V: Due sindromi paralitiche del'arto superiore: note di fisiopatologia della rotazione antibrachiale. Chir Organi Mov 1940;26:215.
259. Putti V: Rapporti statici fra piede e ginocchio nell'arto paralitico. Chir Organi Mov 1922;6:125.
260. Pyka RA, Coventry MB, Moe JH: Anterior subluxation of the talus following triple arthrodesis. J Bone Joint Surg 1964;46-A:16.
261. Rapp IH: Serratus anterior paralysis by transplantation of pectoralis minor. J Bone Joint Surg 1954;36-A:852.
262. Reidy JA, Broderick TF Jr, Barr JS: Tendon transplantation in the lower extremity: a review of end results in poliomyelitis. I. Tendon transplantation about the foot and ankle. J Bone Joint Surg 1952; 34-A:900.
263. Riedel G: Zur Frage der Muskeltransplantation bei Deltoides Lahmung. Ergeb Inn Chir Orthop 1928;21:489.
264. Riska EB: Transposition of the tractus iliotibialis to the patella as a treatment of quadriceps paralysis and certain deformities of the lower extremity after poliomyelitis. Acta Orthop Scand 1962;32:140.
265. Rissler J: Kenntnis der Veranderungen des Nervensystems bei Poliomyelitis anterior acuta. Nord Med Ark 1888;11:22(1).
266. Roundtree CR, Rockwood CA Jr: Arthrodesis of the shoulder in children following infantile paralysis. South Med J 1959;52:861.
267. Rugtveit A: Extra-articular arthrodesis according to Green-Grice, in flat feet. Acta Orthop Scand 1964;34:367.
268. Ryerson EW: Arthrodesing operations on the feet. J Bone Joint Surg 1923;5:453.
269. Sabin AB: Oral poliovirus vaccine: history of its development and prospects. Eradication of poliomyelitis. JAMA 1965;194:872.
270. Sabin AB: Oral poliovirus vaccine: history of its development and use and current challenge to eliminate poliomyelitis from the world. J Infect Dis 1985;151:420.
271. Sabin AB: Pathology and pathogenesis of human poliomyelitis. JAMA 1942;120:506.
272. Sabin AB: Strategy for rapid elimination and continuing control of poliomyelitis and other vaccine preventable diseases of children in developing countries. Br Med J 1986;292:531.
273. Sabin AB, Michaels RH, Spigland I, et al: Community-wide use of oral poliovirus vaccine. Am J Dis Child 1961;101:546.
274. Saha AK: Surgery of the paralyzed and flail shoulder. Acta Orthop Scand Suppl 1967;97.
275. Saha AK: Surgical rehabilitation of paralyzed shoulder following poliomyelitis in adults and children. J Int Coll Surg 1964;42:198.
276. Saha AK: Theory of Shoulder Mechanism: Descriptive and Applied. Springfield, IL, Charles C Thomas, 1961.
277. Salk JE: Studies in human subjects on active immunization against poliomyelitis. JAMA 1953;151:1081.
278. Sammons JH: Polio: out of sight, out of mind [editorial]. JAMA 1977;238:2403.
279. Scheer GE, Crego CH Jr: A two-stage stabilization procedure for correction of calcaneocavus. J Bone Joint Surg 1956;38-A:1247.
280. Schnute WJ, Tachdjian MO: Intermetacarpal bone block for thenar paralysis following poliomyelitis. J Bone Joint Surg 1963;45-A:1663.
281. Schonberger LB, Kaplan J, Kim Farley R, et al: Control of paralytic poliomyelitis in the United States. Rev Infect Dis Suppl 1984;2:424.
282. Schottstaedt ER, Larsen LJ, Bost FC: Complete muscle transposition. J Bone Joint Surg 1955;37-A:897.
283. Schottstaedt ER, Larsen LJ, Bost FC: The surgical reconstruction of the upper extremity paralyzed by poliomyelitis. J Bone Joint Surg 1958;40-A:633.
284. Schwartz RP: Arthrodesis of subtalus and midtarsal joint of the foot: historical review, preoperative determinations, and operative procedures. Surgery 1946;20:619.
285. Schwartzmann JR, Crego CH Jr: Hamstring-tendon transplantation for the relief of quadriceps femoris paralysis in residual poliomyelitis: a follow-up study of 134 cases. J Bone Joint Surg 1948;30-A:541.
286. Seddon HJ: Transplantation of pectoralis major for paralysis of the flexors of the elbow. Proc R Soc Med 1949;42:837.
287. Segal A, Seddon HJ, Brooks DM: Treatment of paralysis of the flexors of the elbow. J Bone Joint Surg 1959;41-B:44.
288. Seymour N, Evans DK: A modification of the Grice subtalar arthrodesis. J Bone Joint Surg 1968;50-B:374.
289. Sharp NN, Guhl JF, Sorensen RI, et al: Hip fusion in poliomyelitis in children: a preliminary report. J Bone Joint Surg 1964;46-A:121.
290. Sharrard WJW: Muscle recovery in poliomyelitis. J Bone Joint Surg 1955;37-B:63.
291. Siffert RS, Forster RI, Nachamie B: "Beak" triple arthrodesis for correction of severe cavus deformity. Clin Orthop Rel Res 1966;45.
292. Smith AD, Lackum HL: Subastragaloid arthrodesis. Surg Gynecol Obstet 1925;40:836.
293. Smith ET, Pevey JK, Shindler TO: The erector spinae transplant: a misnomer. Clin Orthop 1963;30:144.
294. Smith JB, Westin G: Subtalar extra-articular arthrodesis. J Bone Joint Surg 1968;50-A:1027.
295. Somerville EW: Paralytic dislocation of the hip. J Bone Joint Surg 1959;41-B:279.
296. Soule RE: Further considerations of arthrodesis in the treatment of lateral deformity of the foot. Am J Orthop Surg 1915;12:422.
297. Soutter R: A new operation for hip contractures in poliomyelitis. Boston Med Surg J 1914;170:380.
298. Spira E: Replacement of biceps brachii by pectoralis minor transplant: report of a case. J Bone Joint Surg 1957;39-B:126.

299. Spira E: The treatment of dropped shoulder: a new operative technique. J Bone Joint Surg 1948;30-A:220.
300. Staples OS: Posterior arthrodesis of the ankle and subtalar joints. J Bone Joint Surg 1956;38-A:50.
301. Staples OS, Watkins AL: Full active abduction in traumatic paralysis of deltoid. J Bone Joint Surg 1943;25:85.
302. Steigman AJ: The control of poliomyelitis. J Pediatr 1961;59:163.
303. Steindler A: A muscle plasty for the relief of flail elbow in infantile paralysis. Interstate Med J 1918;25:235.
304. Steindler A: Muscle and tendon transplantation at the elbow. Instruct Course Lect 1944;00:276.
305. Steindler A: Newer pathological and physiological concepts of anterior poliomyelitis and their clinical interpretation. J Bone Joint Surg 1947;29:59.
306. Steindler A: Operative treatment of paralytic conditions of the upper extremity. J Orthop Surg 1919;1:608.
307. Steindler A: Orthopedic Operations: Indications, Technique, and End Results, p 129. Springfield, IL, Charles C Thomas, 1940.
308. Steindler A: Orthopedic reconstruction work on hand and forearm. NY Med J 1918;108:1117.
309. Steindler A: Reconstruction of poliomyelitis upper extremity. Bull Hosp Jt Dis 1954;15:21.
310. Steindler A: Reconstructive Surgery of the Upper Extremity, p 56. New York, Appleton & Co, 1923.
311. Steindler A: The treatment of the flail ankle: panastragaloid arthrodesis. J Bone Joint Surg 1923;5:284.
312. Storen G: Genu recurvatum: treatment by wedge osteotomy of tibia with use of compression. Acta Chir Scand 1957;114:40.
313. Straub LR, Harvey JP Jr, Fuerst CE: A clinical evaluation of tendon transplantation in the paralytic foot. J Bone Joint Surg 1957;39-A:1.
314. Sutherland DH, Bost FC, Schottstaedt ER: Electromyographic study of transplanted muscles about the knee in poliomyelitic patients. J Bone Joint Surg 1960;42-A:919.
315. Thomas CI, Thompson TC, Straub CR: Transplantation of the external oblique muscle for abductor paralysis. J Bone Joint Surg 1950; 32-A:207.
316. Thompson CE: Fusion of the metacarpals of the thumb and index finger to maintain functional position of the thumb. J Bone Joint Surg 1942;24:907.
317. Thompson TC: Astragalectomy and the treatment of calcaneovalgus. J Bone Joint Surg 1939;21:627.
318. Thompson TC: A modified operation for opponens paralysis. J Bone Joint Surg 1942;24:632.
319. Toupet R: Technique d'enchevillement du tarse, realisant l'arthrodese de torsion et la limitation des movements d'extension du pied. J Chir (Paris) 1920;16:268.
320. Towsend WR: Treatment of the paralytic clubfoot by arthrodesis. Am J Orthop Surg 1905;3:378.
321. Tubby AH: A case illustrating the operative treatment of paralysis of serratus magnus by muscle grafting. Br Med J 1904;2:1159.
322. Underwood M: Treatise on Diseases of Children with General Directions of Infants from Birth. London, Churchill, 1789.
323. Von Baeyer H: Translokation von Schuen. Z Orthop Chir 1932;56:552.
324. Von Heine J: Beobachtungen über Lahmungszustande der unteren Extremitaten und deren Behandlung. Stuttgart, Kohler, 1810.
325. Von Lesser L: Ueber operative Behandlung des Pes varus paralyticus. Z Chir 1879;6:497.
326. Wagner LC: Modified bone block (Campbell) of ankle for paralytic foot drop with report of twenty-seven cases. J Bone Joint Surg 1931;13:142.
327. Wagner LC, Rizzo PC: Stabilization of the hip by transplantation of the anterior thigh muscles. J Bone Joint Surg 1936;18:180.
328. Waterman JH: Tendon transplantation: its history, indications and technic. Med News 1902;81:54.
329. Watkins MB, Jones JB, Ryder CT Jr, et al: Transplantation of the posterior tibial tendon. J Bone Joint Surg 1954;36-A:1181.
330. Watson-Jones R: Extra-articular arthrodesis of the shoulder. J Bone Joint Surg 1933;15:862.
331. Waugh TR, Wagner J, Stinchfield FE: An evaluation of pantalar arthrodesis: a follow-up study of one hundred and sixteen operations. J Bone Joint Surg 1965;47-A:1315.
332. Weissman SL: Capsular arthroplasty in paralytic dislocation of the hip: a preliminary report. J Bone Joint Surg 1959;41-A:429.
333. Weissman SL, Torok G, Kharmosh D: L'arthrodese extraarticulaire avec transplantation tendineuse concomitante dans le traitement du pied plat valgue paralytique de jeune enfant. Rev Chir Orthop 1957;43:79.
334. Westin GW: Tendon transfer about the foot, ankle, and hip in the paralyzed lower extremity [Instructional Course Lecture]. J Bone Joint Surg 1965;42-A:1430.
335. Whitman A: Astragalectomy and backward displacement of the foot: an investigation of its practical results. J Bone Joint Surg 1922; 4:266.
336. Whitman R: The operative treatment of paralytic talipes of the calcaneus type. Am J Med Sci 1901;122:593.
337. Wickman OL: Studien über Poliomyelitis acuta. Arb Pathol Inst Univ Helsingfors Berl 1905;1:109.
338. Willard DP: Subastragalar arthrodesis in lateral deformities of paralytic feet. Am J Orthop Surg 1916;14:323.
339. Williamson GA, Moe JH, Basom WC: Results of the Lowman operation for paralysis of the abdominal muscles. Minn Med 1942; 25:117.
340. Wilson FC Jr, Fay GF, Lamotte P, et al: Triple arthrodesis: a study of the factors affecting fusion after three hundred and one procedures. J Bone Joint Surg 1929;11:40.
341. Wilson PD: Posterior capsulotomy in certain flexion contractures of the knee. J Bone Joint Surg 1929;11:40.
342. Wright PF, Hatch MH, Kasselberg AG, et al: Vaccine-associated poliomyelitis in a child with sex-linked agammaglobulinemia. J Pediatr 1977;91:408.
343. Yadav SS: Complete rotation of the leg with associated deformities in poliomyelitis. Clin Orthop 1976;76:287.
344. Yadav SS: Muscle transfer for abduction paralysis of the shoulder in poliomyelitis. Clin Orthop 1978;135:121.
345. Yount CC: An operation to improve function in quadriceps paralysis. J Bone Joint Surg 1938;20:314.
346. Yount CC: The role of the tensor fasciae femoris in certain deformities of the lower extremities. J Bone Joint Surg 1926;8:171.
347. Zachariae L: The Grice operation for paralysis flat feet in children. Acta Orthop Scand 1963;33:80.
348. Zancolli EA: Paralytic supination contracture of the forearm. J Bone Joint Surg 1967;40-A:1275.
349. Zaoussis AL: Osteotomy of the proximal end of the radius for paralytic supination deformity in children. J Bone Joint Surg 1963; 45-B:523.

ADDED REFERENCES

1a. Asirvatham R, Rooney RJ, Watts HG: Proximal tibial extension medial rotation osteotomy to correct knee flexion contracture and lateral rotation deformity of tibia after polio. J Pediatr Orthop 1991;11: 646.
2a. el-Batouty MM, Aly ES, el-Lakkany MR, et al: Triple arthrodesis for paralytic valgus: a modified technique. Brief report. J Bone Joint Surg 1988;70-B:493.
3a. Cochi SL, Hull HF, Sutter RW, et al: [Commentary]. The unfolding of global poliomyelitis eradication. J Infect Dis 1997;175(suppl 1):S1.
4a. Cruz Martinez A, Perez Conde MC, Ferrer MT: Chronic partial denervation is more widespread than is suspected clinically in paralytic poliomyelitis: electrophysiological study. Eur Neurol 1983;22:314.
5a. Eberle CF: Failure of fixation after segmental spinal instrumentation without arthrodesis in the management of paralytic scoliosis. J Bone Joint Surg 1988;70-A:696.
6a. Fetwell MR, Smallberg G, Lewis LD, et al: A benign motor neuron disorder: delayed cramps and fasciculation after poliomyelitis or myelitis. Ann Neurol 1982;11:423.
7a. Lau JH, Parker JC, Hsu LC, et al: Paralytic hip instability in poliomyelitis. J Bone Joint Surg 1986;68-B:528.
8a. Lee DY, Choi IH, Chung CY, et al: Fixed pelvic obliquity after poliomyelitis: classification and management. J Bone Joint Surg 1997; 79-B:190.
9a. Leong JC, Wilding K, Mok CK, et al: Surgical treatment of scoliosis following poliomyelitis: a review of one hundred and ten cases. J Bone Joint Surg 1981;63-A:726.
10a. Mayer PJ, Dove J, Ditmanson M, et al: Post-poliomyelitis paralytic scoliosis: a review of curve patterns and results of surgical treatments in 118 consecutive patients. Spine 1981;6:573.
11a. Mehta SN, Mukherjee AK: Flexion osteotomy of the femur for genu recurvatum after poliomyelitis. J Bone Joint Surg 1991;73-B:200.

12a. Men HX, Bian CH, Yang CD, et al: Surgical treatment of the flail knee after poliomyelitis. J Bone Joint Surg 1991;73-B:195.
13a. Perry J, Barnes G, Gronley JK: The postpolio syndrome: an overuse phenomenon. Clin Orthop 1988;233:145.
14a. Strebel PM, Sutter RW, Cochi SL, et al: Epidemiology of poliomyelitis in the United States one decade after the last reported case of indigenous wild virus–associated disease. Clin Infect Dis 1992;14:568.
15a. Westin GW, Dingerman RD, Gausewitz SH: The results of tenodesis of the tendo achilles to the fibula for paralytic pes calcaneus. J Bone Joint Surg 1988;70-A:320.