THE YEAR BOOK *of* ORTHOPEDICS *and* TRAUMATIC SURGERY

(1960-1961 YEAR BOOK *Series)*

EDITED BY

H. HERMAN YOUNG, M.D.

Chairman, Orthopedic Department, Mayo Clinic; Associate Professor of Orthopedic Surgery, University of Minnesota Graduate School of Medicine, Mayo Foundation: Consultant in Orthopedic Surgery, St. Mary's Hospital, Methodist Hospital and Worrall Hospital, Rochester, Minnesota

With a Section on
PLASTIC SURGERY

EDITED BY

NEAL OWENS, M.D., F.A.C.S., F.I.C.S.

Professor of Clinical Surgery (Plastic Surgery), Tulane University School of Medicine; Senior Associate, Department of Surgery, Consultant in Plastic Surgery, Touro Infirmary, and Head of Plastic Surgery Department, Eye, Ear, Nose and Throat Hospital, New Orleans

YEAR BOOK MEDICAL PUBLISHERS

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200 EAST ILLINOIS STREET

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PLASTIC SURGERY

INTRODUCTION

The preparation of this volume of the YEAR BOOK has been accomplished only after a painstaking review of the world's current orthopedic literature. Hundreds of articles, published in English and in many foreign languages during the past year, have been carefully read and evaluated. Abstracts of the articles selected by your editor and his associates are presented in this volume. A number of excellent articles and monographs, too long for abstracting, have of necessity been omitted; they should be read in detail and in the original in order to appreciate the contributions that they represent.

The arrangement of this year's volume is the same as last year's. The chapter on Rheumatic Diseases is quite large owing to the tremendous increase in investigative work and interest in this field.

We call your attention again this year to the chapter on Experimental Orthopedic Surgery and to the contributions that have been made in this field of investigation—out of which the established treatments of tomorrow may come.

I wish to express my appreciation for guidance in the editorship of this volume to Year Book Medical Publishers and to thank these colleagues who commented on material in special chapters: Dr. John G. Mayne for his comments on the chapter Arthritis and Rheumatism, Dr. Edward D. Henderson for his comments on the chapter Paralytic Diseases, Dr. Paul R. Lipscomb for his review and comments on the chapter The Hand and Wrist and Dr. John C. Ivins for his comments on the chapter Peripheral Vascular Surgery. The comments of these specialists are invaluable to this YEAR BOOK.

To Dr. C. Roger Sullivan I would like to express my special appreciation for his splendid help as assistant to the editor.

H. HERMAN YOUNG

Plantar Dissection: Operation to Release Soft Tissues in Recurrent or Recalcitrant Talipes Equinovarus is described by Frederic C. Bost, Edwin R. Schottstaedt and Loren J. Larsen[1] (Shriners' Hosp. for Crippled Children, San Francisco). This operation is designed to effect the release of the ligamentous tissues associated with the navicular, talar and calcaneal articulations, with due consideration for the effect of the equinovarus deformity on the intrinsic muscles of the sole of the foot and such extrinsic muscles as the gastrocnemius-soleus, tibialis posterior and anterior. The authors studied 51 patients who had this procedure and had achieved sufficient age for results to be evaluated.

TECHNIC.—A medial incision is made, arching in a curvilinear manner from the medial calcaneal tubercle over the medial tubercle of the navicular, and continuing distally along the medial aspect of the internal cuneiform and 1st metatarsal to descend at the middle of the first phalanx of the great toe. It is important to skirt the prominent medial border of the bones to facilitate exposure of the dorsal and plantar surfaces of the tarsal joints (Fig. 1). The abductor hallucis tendon is detached from its insertion and the entire muscle is reflected back to the medial tuberosity of the calcaneus. The master knot of Henry is divided and displaced plantarward. The origin of the flexor hallucis brevis is divided, after which all intrinsic muscles of the plantar surface of the foot can be reflected soleward, carrying the neurovascular bundle with them and protecting it from harm.

The insertion of the posterior tibial tendon into the tuberosity of the navicular is dissected from its attachment. The origins of the plantar intrinsic muscles are divided just distal to the lateral and medial tuberosities of the calcaneus. The long and short plantar ligaments are divided at the calcaneo-cuboid joint, together with the plantar capsule of this joint. The ligaments of the talocalcaneonavicular joint are divided circumferentially around the entire proximal margin of the navicular. The deltoid ligament is detached from the navicular, medial margin of the calcaneonavicular, sustentaculum tali and, sometimes, from the inner side of the talus. The posterior talocalcaneal ligament is severed. The interosseous ligament in the tarsal canal is divided, permitting access of the knife blade to the sinus tarsi for division of the ligaments binding the talus to the calcaneus (Fig. 2). The naviculo-cuneiform joint capsule is opened on its medial aspect and on the plantar aspect as far laterally as possible to free the

(1) J. Bone & Joint Surg. 42-A:151-164, January, 1960.

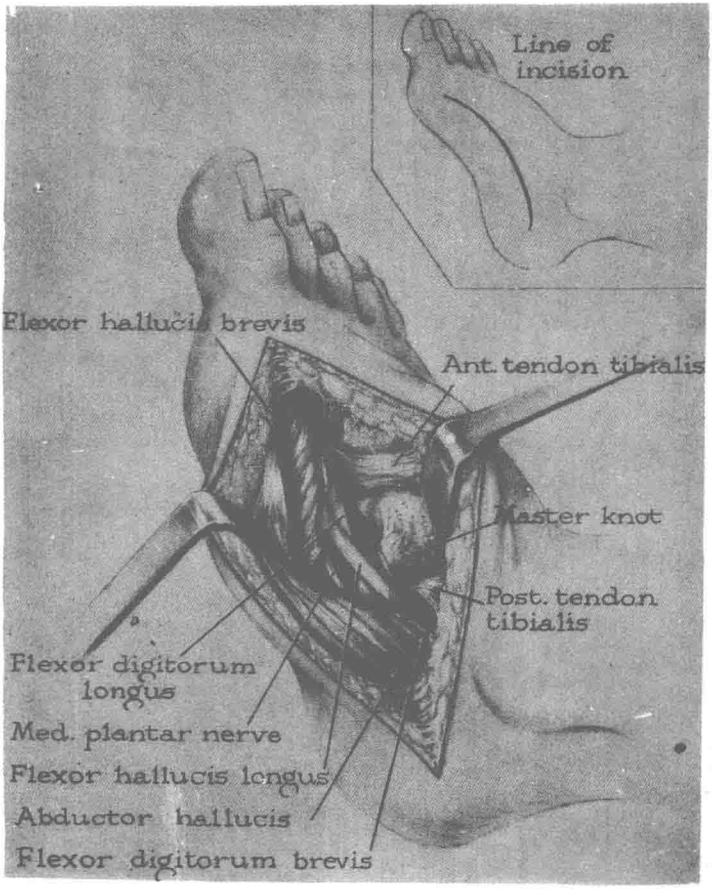

Fig. 1. (Courtesy of Bost, F. C., *et al.*: J. Bone & Joint Surg. 42-A:151-164, January, 1960.)

navicular from all of the cuneiform bones. The insertion of the anterior tibial tendon is lifted, and the cuneiform 1st metatarsal ligament is divided medially along the plantar aspect of the cuneiform metatarsal joints. The peroneus longus tendon is protected.

Of 70 procedures performed, 45 resulted in satisfactory correction of the deformity. Of the 25 unsatisfactory operations, 17 included triple arthrodesis. Overcorrection must be

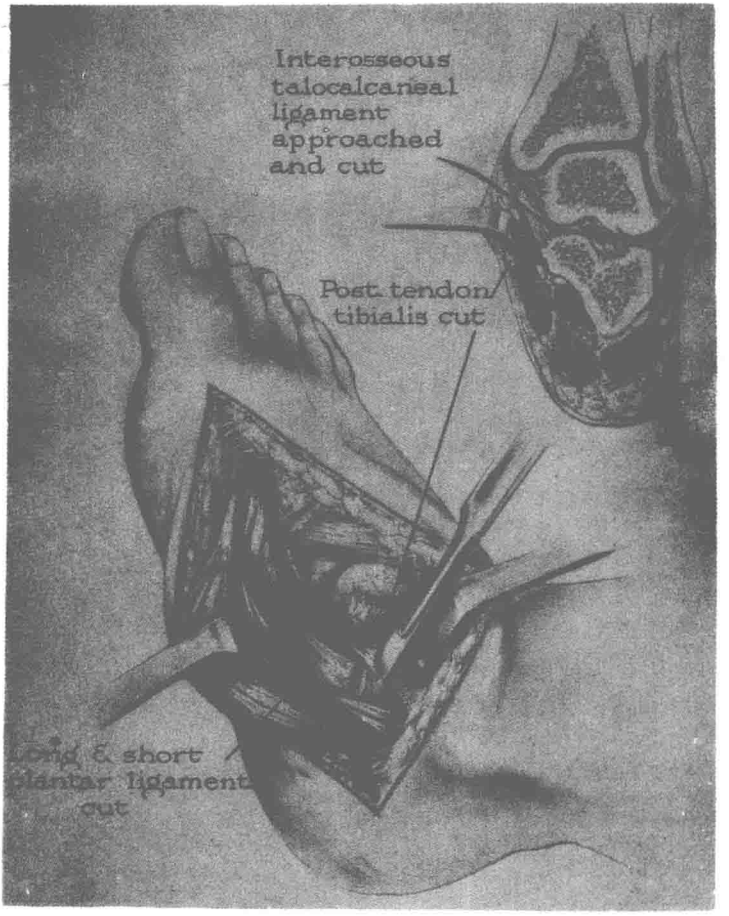

Fig. 2. (Courtesy of Bost, F. C., *et al.*: J. Bone & Joint Surg. 42-A:151-164, January, 1960.)

avoided. Overcorrection has been brought about by too forceful correction of the deformity, unskillful technic in the application of plaster and by overpull of a transplanted anterior tibial tendon.

Surgery should not be used until the benefits of nonsurgical treatment have been exhausted.

Congenital Torticollis: Study of 147 Cases. José R. Pineyro, José Yoel and Marcella Rocca de Pineyro[2] (Buenos Aires) distinguish two types of torticollis, differing in pathogenesis, symptoms, evolution and treatment. One is positional, the other, embryonic. The main sign of the former is the position of the head. Trauma from delivery may produce a hematoma at the level of the sternocleidomastoid, which generally heals without sequelae. This pathologic process is not progressive and is terminated in less than 6 months of treatment in most patients and without treatment in some. Embryonic torticollis develops in the first stages of intrauterine life. Its origin lies in an embryonic alteration of the elements that constitute the muscular and aponeurotic tissues of the neck. The patient appears to have a congenital susceptibility to fibrosis.

The authors treated 115 patients with positional torticollis by traction and massage. Operation was performed on 32 patients with embryonic torticollis.

Results were generally good. Kiesewetter and Brown recommended early removal of the sternocleidomastoid when hematoma is present as effective in preventing the disease. In 4 of 75 patients with hematoma of the sternocleidomastoid, surgical treatment was required later. A hematoma of the sternocleidomastoid always poses the question: will it develop into torticollis, or will it heal with massage? The authors always tried to be prudently conservative.

Early resection would be excellent if every hematoma developed into facial distortion, but this is not the case. If early resection had been performed, 65 patients would have been operated on needlessly, as facial distortion did not develop. Of the 10 patients in whom it did develop, 4 were operated on and 3 others were recommended for operation but did not undergo it. The other 3 recovered. A check of the 4 patients operated on revealed a slight regression of the facial distortion in 3. In 1 it persisted. Increased symptoms were noted in the 3 patients for whom operation was recommended but not performed.

Congenital Muscular Torticollis (Wryneck) was reviewed by Mark B. Coventry, Lloyd E. Harris, Anthony J. Bianco, Jr., and Arthur H. Bulbulian[3] (Mayo Clinic and Found.)

(2) J. Internat. Coll. Surgeons 34:495-505, October, 1960.
(3) Postgrad. Med. 28:383-392, October, 1960.

in 35 infants. Thirty were managed conservatively, with excellent results. Five were treated surgically, and 2 of these had poor results.

Because the tumor of the sternocleidomastoid muscle disappears spontaneously and because contracture of the muscle with torticollis deformity develops only occasionally, treatment of patients under age 1 year should be nonsurgical Stretching, heat or massage have no healing effect. Correction of residual deformity by section of the contracted sternocleidomastoid is indicated for those occasional patients who continue to have torticollis deformity after age 1 year. If operation is delayed until age 12-14 years, complete correction of facial asymmetry may not be possible. Surgical release is accomplished by tenotomy of the sternal and clavicular heads of the contracted sternocleidomastoid muscle under direct vision. Tenotomy of the mastoid attachment occasionally may be necessary. If present, contractures of the platysma and scalene muscles and cervical fascia may have to be corrected at the same time. To prevent recurrence of the deformity after tenotomy, a plaster cast should be used to hold the head in a position of overcorrection for 6 weeks.

Osteochondritis in Congenital Dislocation of Hip: Clinical and Radiographic Study. Three distinct vascular arrangements characterize the femoral head during periods of growth. The earliest is distinguished by absence of penetration into the epiphysis of any vessel from the ligamentum teres, while the lateral epiphysial vessels, with some assistance from the metaphysial vessels, carry the main blood supply to the femoral head. This arrangement lasts until about age 4 years and thus encompasses the ages during which congenital hip dislocation is generally reduced. From ages 4 until about 7 or 8 years, the lateral epiphysial vessels alone supply the femoral head. This is the period when Legg-Perthes disease occurs. At about age 8, the vessels of the ligamentum teres penetrate the femoral head and augment the blood supply.

C. Lima, R. Esteve and J. Trueta[4] (Oxford, England) studied 184 hips treated for congenital dislocation. Of these, 90 showed pathologic disturbances of the femoral head. In 83 instances, it was possible to classify the condition into

(4) Acta orthop. scandinav. 29:218-236, 1960

one of three types of x-ray changes. Type I, comprising 54 patients, showed early osteoporosis of the proximal end of the femur, starting usually in the epiphysis and involving the acetabulum. Thereupon the osteochondritic area may recalcify or flattening of the medial aspect of the epiphysis may take place. In type II (22 patients), x-ray evidence showing the effect of pressure over the femoral head was evident from onset, causing indentations and tangential flattening of the femoral head as the lesion progressed. In type III (7 patients), the x-ray signs and evolution of the process resembled Legg-Perthes disease. In 2 instances, the first sign was increased density of the femoral head, followed by osteoporosis. In the other instances, the first sign was bone reabsorption of the head and neck. Osteoporosis increased and later fragmentation of the epiphysis ensued. Recalcification was eventually associated with some restoration of the shape of the femoral head. The final anatomic result varied with the severity and persistence of the deforming forces.

The incidence of osteochondritis was much greater among the patients treated by manipulation, and less among those patients who were initially treated by reduction on a frame. The severity of the final deformity related similarly to the method of reduction. Increase in the severity of the process with the patient's age at the start of reduction treatment was noted. A much higher occurrence of the more severe forms of osteochondritis was likewise relatable to the anatomic result of reduction, with patients with the more severe conditions showing incomplete reduction of the dislocated hip.

"Inherited Dysplasia" of Hip Joint in Dogs and Rabbits was studied by J. R. M. Innes[5] (Brookhaven Nat'l Lab.). The condition appears to be somewhat analogous to the disease complex in children that has been attributed to delayed ossification of the femoral head and acetabulum with formation of an inadequate ball-and-socket joint, subject to various late sequelae as the result of mechanical stress. The disease in rabbits, known as splayleg, has been more thoroughly studied than that in dogs.

Splayleg becomes clinically detectable about 20 days after birth, at the time the rabbits usually emerge from the nest.

(5) Lab. Invest. 8:1170-1177, Nov.-Dec., 1959.

The condition is characterized by inability to put weight on one or both hind limbs (sometimes also the forelimbs). Later, the limbs are twisted at fantastic angles. With limb deformity, muscle pull becomes disoriented and contractures develop so that the paws are turned completely around. In animals killed at 22, 28, 45, 65 and 92 days, the anatomic basis seemed to be subluxation and later dislocation of one or both hip joints.

In animals killed at an early stage, no true histologic changes in the hip were noted, other than a poorly shaped and flattened femoral head, incorrect angulation of the neck and a shallow acetabulum. The joint capsule was normal, the articular cartilages were not eroded and there was no necrosis, mucoid degeneration or fibrillary changes. Later, after onset of daily trauma caused by abnormal movements of a dislocated joint, a whole series of chronic changes developed: avulsion of the round ligament, destruction of the articular cartilages, fibrosis around the cotyloid ligament and capsule, erosion of the femoral head and acetabulum, twisting and bowing of the shaft, i.e., a picture of severe traumatic osteochondritis with exostosis and false joint. In the various developmental phases of the condition in rabbits, traced through different cases, many, if not all, of the hip joint lesions recognized in dogs, and depicted as entities radiologically, might appear to have been represented.

Subluxation dislocation (or dysplasia) as a clinical entity in dogs has been known for decades. It occurs in many different breeds, although some breeds are said to be more prone than others, e.g., Alsatian, boxer, fox terrier, Irish setter. There has been a somewhat fruitless controversy on the nature of dysplasia based largely on clinical and x-ray data. No work has been done on the genetic or pathologic aspects, particularly of the early stages before trauma of subluxation and dislocation begin to exert effects. If in such conditions examination was done very early, perhaps the analogy to the rabbit disease would be close, i.e., apart from a malformed joint, no pathologic changes would be found until the trauma of weight-bearing started. A study of late stages is of no use in an attempt to establish the basic nature of the disease. This could have immense value in comparative pathology because in children, all disorders that might be termed "dysplasia," because they are not lethal and are improved by

orthopedic methods and time, do not provide opportunities for good pathologic study.

Congenital Dislocation of Knee, a relatively rare condition, is often associated with congenital dislocation of the hip. The characteristic deformity is anterior displacement

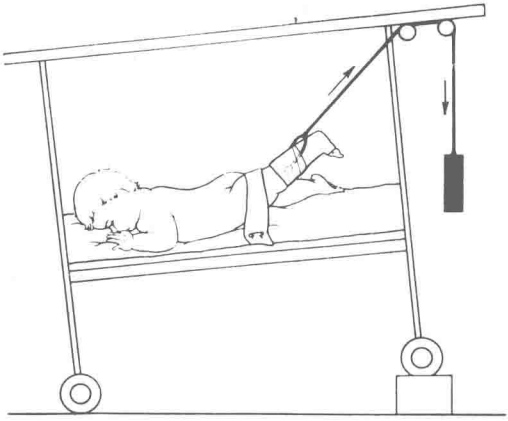

Fig. 3.—Traction method used successfully in treatment of 6 knees. (Courtesy of Niebauer, J. J., and King, D. E.: J. Bone & Joint Surg. 42-A:207-225, March, 1960.)

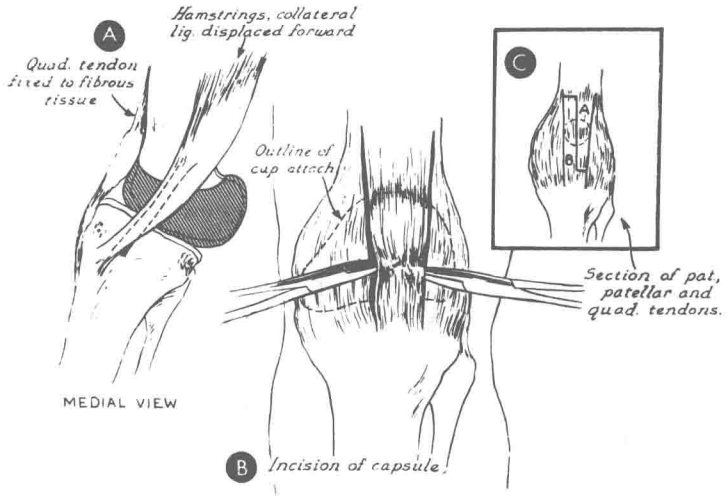

Fig. 4.—Open reduction. (Courtesy of Niebauer, J. J., and King, D. E.: J. Bone & Joint Surg. 42-A:207-225, March, 1960.)