
2.1 Introduction

Growth of bone is determined not only by heredity and nutrition, but also by the work it has to perform. The mechanical factors which influence and are essential for normal longitudinal growth, are movement and weight bearing [2]. Disuse of an extremity from any cause results in atrophy of muscle and other soft tissues in both adults and children. Osteoporosis is a well known feature of inactivity and disuse in adults. Osteoporosis from disuse also occurs in children, but to a lesser extent. If disuse is prolonged in children, growth of bone length is also diminished. Since growth retardation occurs before growth arrest, the process may be reversed if discovered and treated early. If disuse is marked, some physes may cease growing completely. The most commonly reported sites of growth slow down and arrest from disuse are the distal femur and proximal tibia [3]. Unfortunately these physes rank first and second in the amount of longitudinal growth they provide.

The incidence of growth arrest associated with disuse is difficult to measure because the disuse is variable and because recognition of the arrest may require years of observation. Prolonged disuse secondary to bed rest, traction, cast immobilization, braces, crutches, and delayed weight bearing, have all been associated with growth slow down or arrest of one or more physes of the involved extremity. These treatment modalities, previously used in management of children with tuberculosis of the hip, poliomyelitis, lower extremity diaphyseal fracture, Perthes disease, developmental hip dislocation, osteomyelitis, and slipped capital femoral epiphysis, are today used more sparingly for a number of reasons, one of which is the occurrence of growth arrest.

The degree to which growth reduction is associated with neurologic conditions (discussed in Chap. 6) cannot be separated from the accompanying disuse. The statement “that education in the performance of exercises designed to increase the use of the limb to the greatest possible extent may lead to marked reduction of the discrepancy in growth” [1] was written in the context of neurologic conditions, but can be applied to all causes of disuse.

2.2 Tuberculosis

Prior to the mid twentieth century tuberculosis of the hip was common in children and the treatment was “rest, uninterrupted, continuous, and prolonged.” [4] Bed rest, balanced traction, and casts were commonly used for months to years [5, 6, 10]. The duration of treatment was chosen arbitrarily to allow completion of destruction of the femoral head to improve probability of successful hip fusion [10]. One regimen included continuing immobilization and delaying hip fusion until the child had “reached his tenth year of age, providing the acute manifestations of the disease have disappeared.” [6] These children were often more disabled from secondary complications of disuse than from their hip joint tuberculosis [10]. One patient with bilateral tuberculosis of the hip developed growth arrest of both knees [3].

Arrest of physes distal to the hip would often occur even though none of these physes or their accompanying joints were infected [7]. The longer the duration of disuse the more extensive the growth arrest about the knee (Table 2.1) [9]. The limb length discrepancy varied from minimal up to 7 in. [3, 10]. In one series [10],

Table 2.1 Incidence of knee physal arrest with hip tuberculosis in children

Year	Author	No. cases	No. arrests	Percentage ^a
1944	Gill [7]	150	10	6.7
1947	McCarroll and Heath [10]	43	28	65.1
1948	Ross [3]	92	9	9.8
1949	Parke et al. [11]	91	29	31.0
TOTAL		376	76	20.2

^aThe lower percentage of arrests is probably due to earlier hip surgery and shorter time of immobilization [3]. Length of follow-up was not well documented in these series, which also impacts the results

of 43 hips completely immobilized for 2–8 years, 28 (65%) showed limb shortening of from 2 to 7 in. The degree of shortening was in direct proportion to the length of immobilization. The loss of limb length was estimated to be 12% from the diseased and fused hip, and 88% due to premature closure of the distal femur and proximal tibia physes.

The physes most often affected from disuse are the distal femur and proximal tibia, separately or both [3, 7–9, 11]. The fibula and distal tibia physes are never involved [11]. The central portion of the physis is affected first producing cupping (Sect. 1.3), which if left untreated progresses from mild to moderate, to marked (Fig. 1.13) [7, 12]. When the cupping is eccentric, angular deformity occurs [7, 12]. When the proximal tibia is involved, normal fibular growth proceeds to relative overgrowth.

Although observers recognized that growth arrest was associated with prolonged immobilization, the explanation for it was often speculative. It was difficult for authors to separate the accompanying and often more noticeable osteoporosis from the growth arrest [3]. Some authors [7, 8, 13] speculated that in some cases the accompanying osteoporosis predisposed the physis or the accompanying small vessels to microfractures. Others suggested that the arrest was due to a secondary factor, such as a faulty gait [3]. These early attempts to explain arrests by mechanical causes were gradually replaced by vascular insufficiency explanations, confirmed by the work of Trueta and colleagues in the 1950s (Sects. 1.2 and 1.3). The presence of soft tissue atrophy and the absence of pain support the concept of impaired vascular supply [9].

Long periods of immobilization in casts should be avoided unless it is absolutely necessary [8, 13]. Although the distal physal arrests can be found 6–8 months following the hip disease, the clinical features of growth

impairment are often not detected for 2–3 years [9]. Gill [8] advocated the surgical placement of metal markers in the femoral diaphysis, followed by a repeat scanogram every 6 months to observe the amount of growth. The ensuing relative limb discrepancy was usually treated by a shoe lift, contralateral surgical physal arrest or bone shortening, or ipsilateral bone lengthening. On occasion severe leg length discrepancy resulted in amputation and prosthetic fitting [5, 12].

2.3 Poliomyelitis

In the aftermath of poliomyelitis, recovery of useful function in the paralyzed limb is sometimes remarkable, but growth of the limb is usually disturbed [27]. Adults who contract poliomyelitis develop no limb length discrepancy. In children, paresis caused by poliomyelitis often results in growth retardation of a lower limb by 5%; i.e. about 4 cm in the average adult. Growth of the tibia is affected to a greater extent than the femur by a ratio of 3:2, regardless of the distribution of the paralysis (Fig. 2.1) [26]. Central physal closure with cupping (Sect. 1.5) was found in several distal femoral and proximal tibial physes [16]. The age of onset of the disease has little influence on the annual increment of shortening, but influences the final inequality due to the number of years to maturity [26].

The etiology of diminished bone growth in the paralytic limb is most likely due to secondary disuse rather than primary nerve dysfunction. The functional use of the involved extremity plays a large role in determining its longitudinal growth [18, 28]. Disuse of the extremity alters the circulation and nutrition of the extremity. Delayed weight bearing is thought to be a greater factor than paralysis. Intermittent weight bearing is a necessary stimulus for optimal growth. The

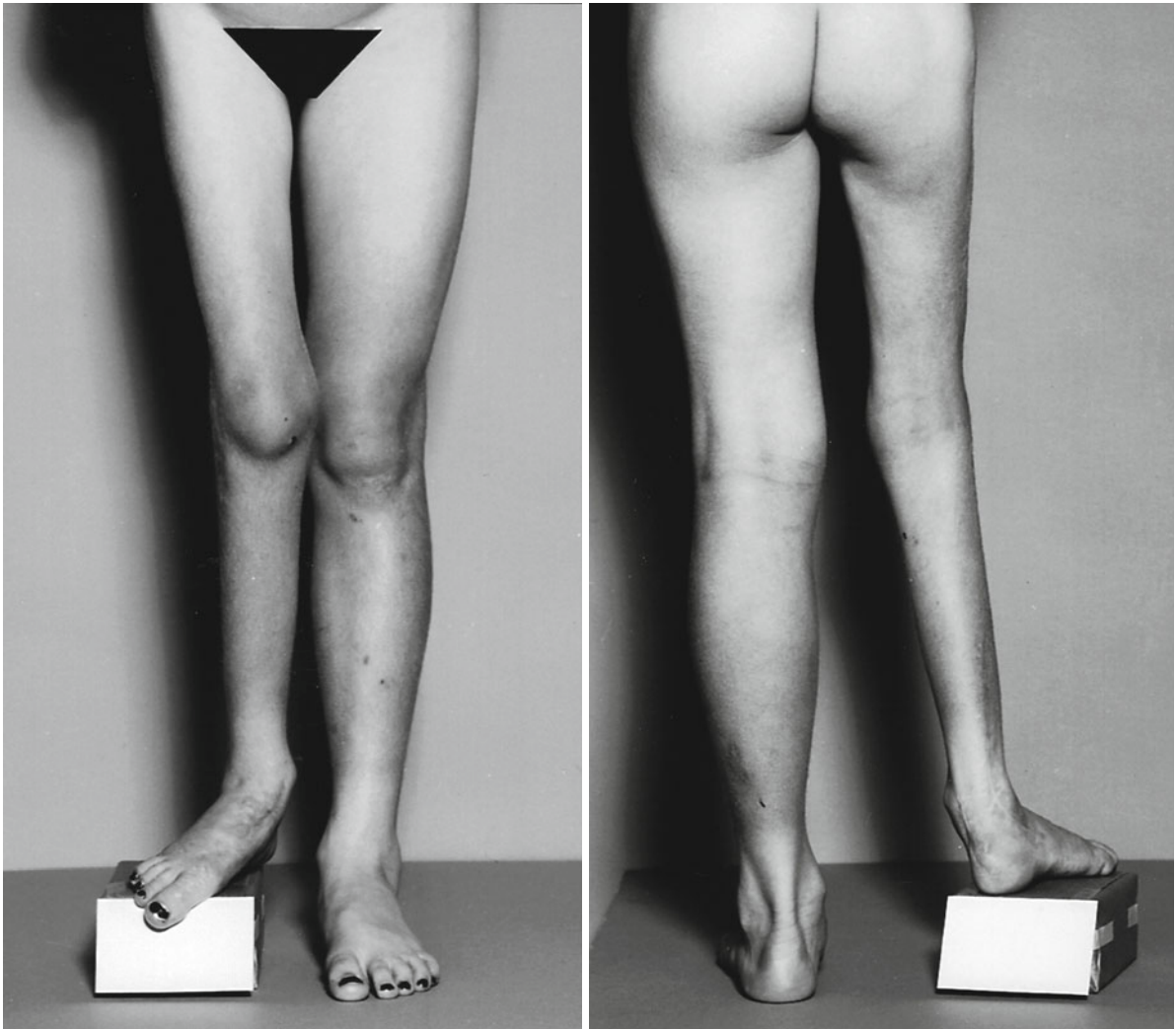


Fig. 2.1 This 8 year 7 month old female had poliomyelitis at age 20 months. The right lower extremity is 5.7 cm shorter than the left (femur 2.0 cm, tibia 3.7 cm)

relationship of motor paralysis and limb vasculature as a cause of diminished physal growth has also been studied. Chronic vascular insufficiency with consequent hypoxia of growth cartilage cells is known to reduce growth in poliomyelitis patients. Harris and McDonald [19] in 1936 noted that the increase in blood supply which follows lumbar sympathectomy in poliomyelitis patients was capable of inducing mild acceleration of the rate of growth. The factors favorable to a good result include paralysis limited to one lower extremity, paralysis of moderate degree, operation before age 6 years, and the use of ganglionectomy rather than ramisection. Subsequent studies, however,

concluded that sympathectomy does not increase the growth potential consistently or appreciably in polio limbs [15, 17, 29]. Sympathectomy performed to increase bone length in poliomyelitis patients is no longer used, most likely because the result is unpredictable, the benefit minor, the side effects permanent, and if the discrepancy is large other treatments would be needed anyway. A related experimental study in rabbits [29] concluded that “motor paralysis (with the sympathetic nerve supply intact) plays a very minor part in the retardation of growth.” Another proposed concept that the circulating polio virus damages blood vessel walls directly [24], was never proven.

Barr (1948) [14] studied 371 cases in which the onset of poliomyelitis was before the age of 16 years. Forty-one per cent had ½ in. or less of shortening, 24% had between ½ and 1½ in. shortening, and 35% had 1½ in. or more of shortening. The age of onset was an important factor. Leg length inequality of moderate or marked severity was greater in boys, due to longer growing period and greater length potential. Premature cessation toward the end of growth was noted in the femur and tibia, together or separately. Although qualitative measurement of the circulatory status was not done, decreased circulation was felt to cause the growth retardation.

Stinchfield et al. (1949) [28] examined 166 adults in whom poliomyelitis developed before age 11 years and found a definite relationship between the muscle strength in the two lower extremities and the discrepancy in limb length, but no relationship between the age of onset and the amount of length discrepancy.

Gullickson et al. (1950) [18] reviewed 88 chronic poliomyelitis patients with unilateral lower extremity involvement and found a definite correlation between atrophy of the thigh or leg and shortening of the femur or tibia, between total atrophy and shortening of the involved extremity, and between total strength of the involved extremity and shortening in that extremity. The age at which acute poliomyelitis occurred affected the percentage of shortening; the younger the onset the greater the percentage of growth retardation (Table 2.2). They concluded that in poliomyelitis there is a decreased blood flow due to increased sympathetic activity or decreased function of the leg, causing an interference with soft tissue and bone metabolism and therefore growth.

Ring (1957) [27] evaluated 55 patients with residual unilateral paralysis and noted that in young children the effect of paralysis on growth increased as age advanced, and that this effect is maximal at age 10 years and then rapidly diminished. Since no mechanical cause could be found, he concluded that since the paralyzed limb is often cold and blue and has a lower resting blood flow than the contralateral leg, bone growth is diminished due to vascular insufficiency.

Ratliff (1959) [25] found leg shortening in 215 of 225 (96%) patients. Most of the shortening was in the tibia rather than in the femur. He noted that although a mild degree of paresis produced a small amount of shortening, severe paresis might produce either a little or more significant shortening.

Table 2.2 Relation between age of onset of acute poliomyelitis and retardation of bone growth expressed as percentage shortening in relation to the normal extremity [18]

Age at onset years	No. cases	Shortening of femur (%)	Shortening of tibia (%)	Shortening of lower extremity (%)
0–5	47	2.52	2.44	2.64
6–10	29	1.96	2.37	2.14
11–14	12	1.40	1.39	1.27

Makin (1965) [22], reviewed 112 patients with unilateral paralysis with leg length discrepancy of more than 2.5 cm, measured by scanography. The onset of poliomyelitis was 6 years or less in all patients. The length deficit was primarily in the tibia in 51 patients, in both the femur and tibia and 48, and in the femur in 13. Shortening of the fibula was greater than that of the tibia in 87 patients (78%). Fibula shortening resulted in valgus and instability of the ankle, and if marked predisposed to genu valgum and lateral tibial torsion.

In addition to relative shortening of the tibia and femur, the fibula and the fourth metatarsal have also been found to be affected. Shortening of the fibula, relative to the tibia, results in a wedge shaped distal tibial epiphysis and ankle valgus [30], just as in other cases of other developmental fibular deficiency (Sect. 19.3). Currarino (1966) [16], found that of 250 unselected patients with poliomyelitis, 22 (9%) had premature closure with shortening of the fourth metatarsal in the paretic limb.

The period of immobilization of poliomyelitis patients should be as short as possible [13]. The “natural history” of the polio disease in an individual case as measured by the recovery of function, is variable and unpredictable early on. Careful follow-up will help identify cases which might benefit from treatment such as physal arrest of the longer extremity. Polio residuals of genu valgum [21] and genu recurvatum [23] may also be related to the disuse and may be treated by staple hemiepiphyseodesis and osteotomy respectively, provided there is no physal bar. A procedure to stimulate growth in the affected leg by the surgical creation of an arteriovenous fistula was successful in many cases of poliomyelitis (Fig. 2.2) [20], but was abandoned because the amount of correction achieved was unpredictable, and subsequent repair of the AV fistula is an operation of considerable magnitude. The procedure and

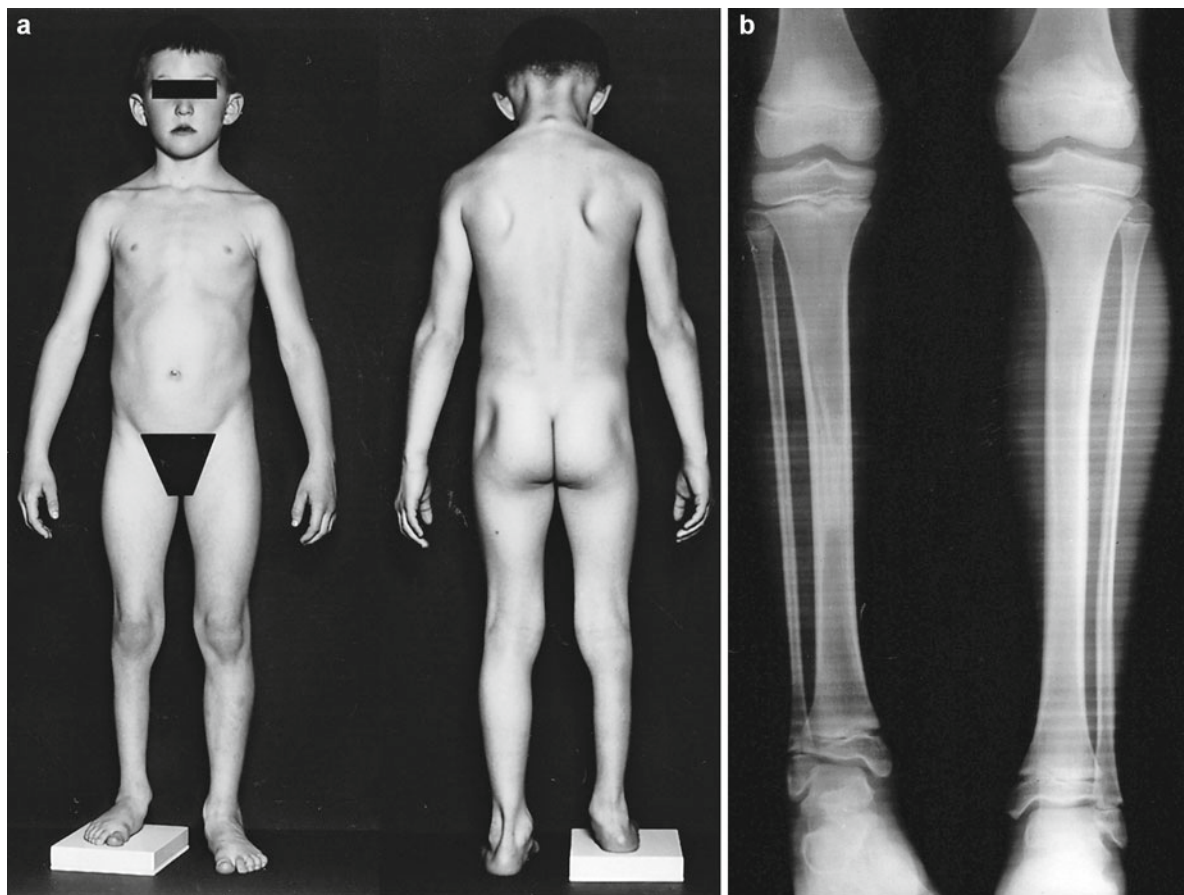


Fig. 2.2 This boy had poliomyelitis at age 6 months. **(a)** At age 7 years 4 months the right lower extremity is 3.5 cm shorter than the left, primarily in the tibia. An arteriovenous fistula was surgically created in the right mid superficial femoral vessels at age

7 years 4 months. **(b)** At age 10 years 9 months the right tibia and fibula have grown faster than the left and the discrepancy is now only 2.4 cm. The bone and physeal structure is comparable to the left

results, however, support the premise that reduction of bone growth in poliomyelitis is a vascular deficiency associated with disuse, and not a primary neurologic deficit of the physis.

2.4 Diaphyseal Fracture

Prolonged immobilization of diaphyseal fractures in children can result in considerable physeal damage, including premature and often eccentric closure, resulting in relative shortening and angular deformity [32]. The physeal arrest does not appear to be related to the severity of injury [31]. Recognition of the arrest is delayed for an average of 1 year 10 months [34].

Femoral and tibial diaphyseal fractures are the bones of concern in the literature.

Fractures of both the proximal and mid femur, when treated by prolonged immobilization, can develop premature arrest of physes distal to the fracture. The arrest may occur in only one, or in any combination of physes distal to the fracture [35, 38], but most often affects the distal femur and proximal tibia (Fig. 2.3) [8, 31, 32]. In one series [39] of 132 femoral fractures, there were 6 cases of arrest of the distal femur (4.5%), all considered to be due to prolonged immobilization in plaster associated with secondary salvage procedures. In most cases the precise cause of the arrest, whether from prolonged disuse or initial vascular insufficiency (Table 1.1), cannot be determined.

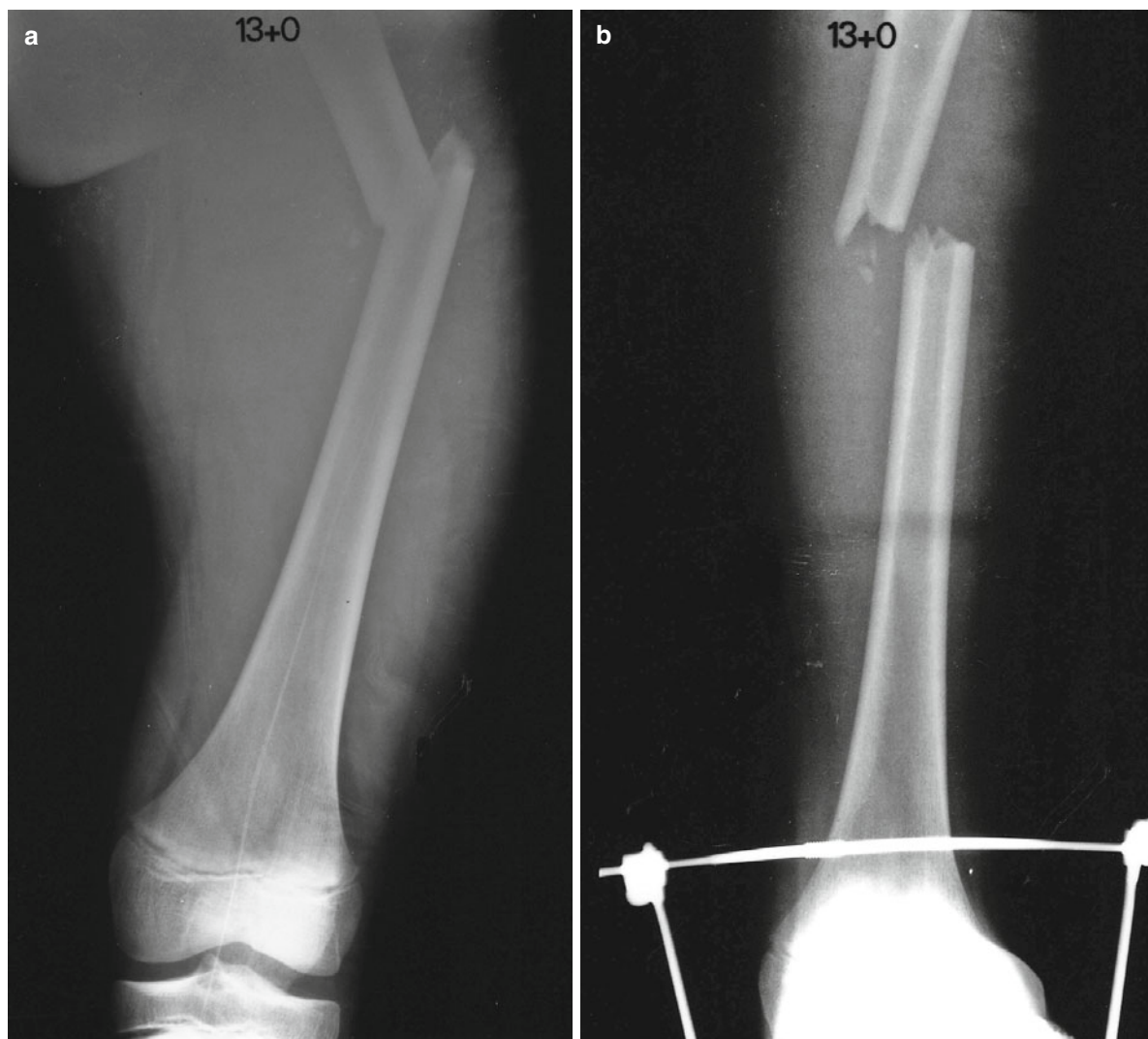
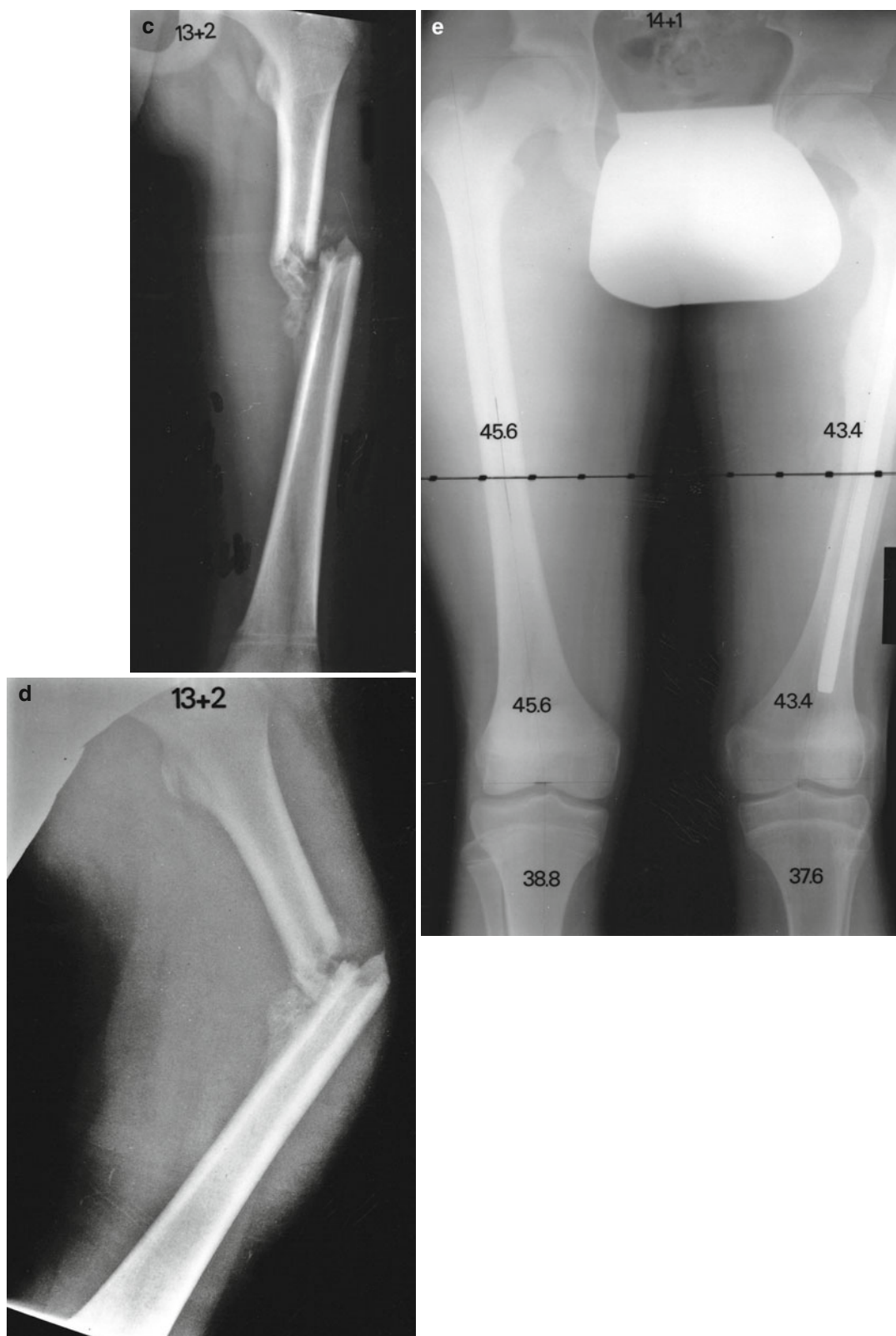


Fig. 2.3 Premature arrest of the distal femur and proximal tibial physes associated with a femoral diaphyseal fracture and prolonged disuse. This 13 year 0 month old male fell from a dirt bike injuring his left thigh. **(a)** There is a displaced femoral diaphyseal fracture. The distal femoral and proximal tibial physes are normal. **(b)** Reduction was incomplete after 7 days in 90/90, 20 lbs. skeletal traction. Traction continued 22 days at which time the traction pin was removed and a spica cast applied. The cast was worn 28 days. **(c)** Status of healing 52 days post fracture 2 days after cast removal. Note maintenance of length despite poor fracture healing. No new bone formation on the lateral side suggests that the distal fragment may have a button-hole through the periosteum **(a)**. **(d)** Refracture 5 days post cast removal. The fracture was manipulated and a spica cast reapplied. The second cast was worn 44 days, followed by crutches. Delayed malunion resulted in open intramedullary nailing and bone grafting 6 weeks later. Crutches were again used and discontinued 8 months post fracture. **(e)** Scanogram 13 months post fracture, age 14 years 1 month. The left femur is 22 mm shorter and left tibia 12 mm shorter than the right. The femoral fracture was healed. **(f)** Four coronal tomograms of the uninjured right knee show open femoral and tibial physes. **(g)** Four coronal

tomograms of the left knee show significant narrowing of the distal femoral and proximal tibial physes. Two months later the patient underwent rod removal and right distal femoral and proximal tibial surgical epiphysiodeses. **(h)** Age 16 years 4 months. The patient was normally active and asymptomatic and played high school hockey goalie. Scanogram showed a leg length discrepancy of 27 mm (femoral 16, tibial 11). All physes are closed. The patient wore a permanent shoe lift except for athletic shoes. The left foot is two shoe sizes smaller than the right (implying premature arrest of metatarsals and/or phalanges). The patient's height was equal to his father: 177.7 cm. Note: This fractured right femur was treated by 22 days traction, 72 days spica cast, intramedullary nailing, and crutches for 8 months. There was significant slow-down of growth of knee physes distal to the fracture as documented by relative shortening compared with the opposite extremity. The surgical physal arrests on the normal knee prevented the discrepancy from increasing. The patient was left with a 27 mm leg length discrepancy. In this case the premature physal closures could have been due to prolonged disuse, to initial vascular insufficiency (Sect. 1.3), or to both

**Fig. 2.3** (continued)

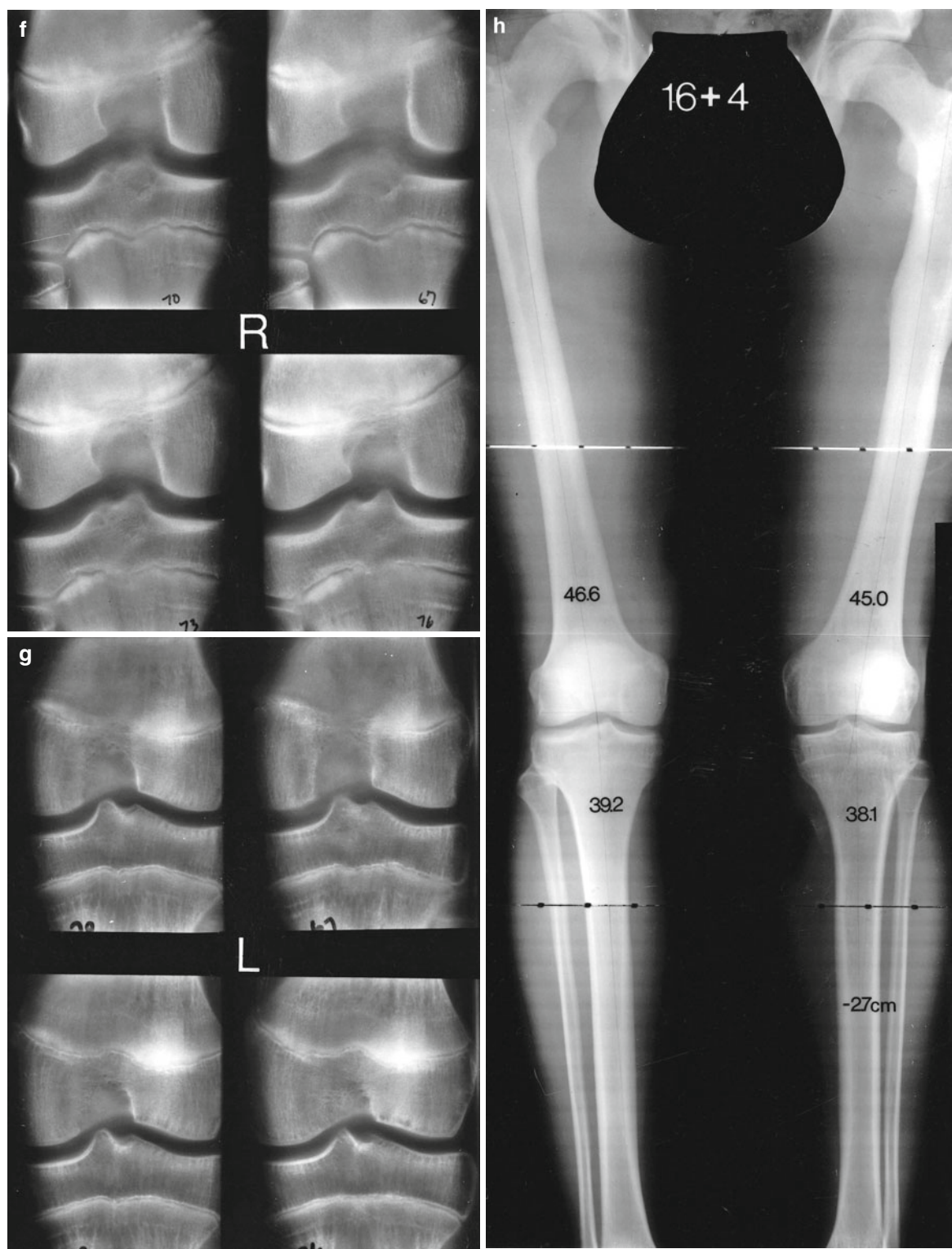


Fig. 2.3 (continued)

When the tibial diaphysis is fractured, however, the arrest may occur proximal to the tibial fracture (Fig. 1.10) [36, 37]. When this occurs vascular insufficiency due to disuse may be more plausible than vascular insufficiency due to the fracture. In one series of 354 adolescents with tibial fractures there were 7 cases (2%) of premature physeal closure [33]. All fractures resulting in arrest occurred in children between 12 and 15 years of age [33, 36, 37]. Consequently, when fracture-associated arrest occurs in this age range, the length discrepancy is often discovered too late for surgical arrest on the contralateral side. Thus, either surgical lengthening of the injured tibia or shortening of the normal femur is often required. Adolescents with tibial diaphyseal fractures should be monitored until growth is complete because of the risk of developing leg length discrepancy as a consequence of premature closure of one or more of the leg physes [37].

The physes exhibiting premature closure are always large, i.e., the distal femur, proximal and distal tibia (Table 1.1). Except for the closure of one proximal fibula [35], no other small physes, such as the metatarsals or phalanges have been recorded. This is similar to physeal arrest with vascular deficiency due to decreased quantity discussed in Sect. 1.3, but different from poliomyelitis where the fourth metatarsal is sometimes affected [16].

It is essential to treat fractures in children effectively and in the shortest possible time [32]. Early recognition of limb length discrepancy can be treated with surgical arrest of one or more physes. If the discrepancy is discovered later, physeal arrest will only prevent the discrepancy from increasing, and bone shortening or lengthening operations might be necessary.

2.5 Developmental Dislocation of the Hip

Premature closure of the distal femur and proximal tibia physes occurred on the side of the developmental hip dislocation (DDH) in three children immobilized in frog position casts for 12, 16, and 19 months [40]. The authors concluded that “relative ischemia of the involved physes is the most likely cause.” Kestler [9] illustrated one case but did not specify the type or duration of treatment. Although ischemia explains the

physeal arrest in the absence of trauma or toxic effect, the reason for involvement of only the ipsilateral knee (not the ipsilateral fibula or distal tibia, or the contralateral extremity) is not known. Complete immobilization should be as brief as possible [13].

When DDH is associated with avascular necrosis of the femoral head, the proximal femoral physis is at an even higher risk of arrest. If immobilization in a hip spica cast is prolonged, disuse compounds the underlying vascular deficiency of the epiphysis (Chapter 1B).

2.6 Perthes Disease

Premature closure of the distal physis has been noted to occur in the same femur as the Perthes disease [41]. Disuse, the cast, and position in the cast (similar to the discussion in Chap. 1, DDH), could all be factors. Of 147 patients with unilateral Perthes disease followed 5 years or more, the average femoral shortening was 1.38 cm, and the average tibial shortening was 0.93 cm (total 2.14 cm) [42]. The extent of the tibial discrepancy correlated well with the time of immobilization in the unilateral abduction ischial weight-bearing brace.

2.7 Osteomyelitis

Physeal arrest associated with osteomyelitis is usually thought to be due to infection in the metaphysis or epiphysis directly affecting the physis (Chap. 3). On occasion, however, the osteomyelitis is in the diaphysis, well away from the physis. If immobilization is prolonged, the arrest may be due to vascular insufficiency associated with disuse. The fact that osteomyelitis of the femoral diaphysis is associated with arrest in the proximal tibia [3, 43], as well as in the distal femur, supports this premise.

2.8 Slipped Capital Femoral Epiphysis

In the past, slipped capital femoral epiphysis (SCFE) was sometimes treated by prolonged traction or cast. One 11-year-old boy immobilized in plaster for 7½ months developed arrest of the proximal tibial physis [3].

Physeal arrest of the involved hip is more likely to occur, partly due to disuse. In one series 37 hips were treated with bed rest and traction for relief of symptoms, followed by spica cast immobilization, 8–16 weeks, until the physis reverted from increased width to normal width [44]. Fourteen of the 37 hips (38%) developed premature closure of the physis of the involved hip. Because the proximal femur has little growth remaining in these older children the resulting limb length discrepancy was no more than 2 cm (average 1.1 cm). In a second series, 10 of 17 hips (59%) treated by initial traction to allow pain and spasm to resolve, followed by a one and one-half or a bilateral spica cast (requiring bed rest) for 3–4 months, developed partial or complete physeal arrest of the proximal femur [45]. The high rate of complications, including physeal arrest, resulted in abandonment of the use of the spica cast [45].

2.9 Chemically Induced Immobilization

Induction of rigid immobilization with decamethonium bromide and flaccid immobilization with panurionium bromide in chick embryos *in ovo* for 3 days resulted in greatly reduced limb lengths and decreased width of epiphyses, most marked and more pronounced distally [46]. Could induced prolonged coma in a very young child have a similar effect?

2.10 Author's Perspective

After reading Chaps. 1 and 2 it is easy to conclude that the basic cause of most of the conditions discussed in Chap. 2 are the result of vascular deficiency due to reduced quantity discussed in Chap. 1. The conditions discussed in Chap. 2 are presented separately, because disuse is the primary event that initiates the vascular deprivation. Disuse is mentioned as an adjunct cause of physeal arrest in several other chapters, most notably Chap. 5, Metabolic Injuries, and Chap. 6, Neural Injuries. In these and other injuries it is more difficult to determine the precise role of disuse from the specific injury. Disuse of any part of the body increases risk of complications including growth arrest, and should be as short as possible.

References

Introduction

1. Gillespie JA (1954) The nature of the bone changes associated with nerve injuries and disuse. *J Bone Joint Surg* 36B(3):464–473
2. Golding JSR (1994) The mechanical factors which influence bone growth. *Eur J Clin Nutr* 48(Suppl 1):S178–S185
3. Ross D (1948) Disturbance of longitudinal growth associated with prolonged disability of the lower extremity. *J Bone Joint Surg Am* 30A:103–115

Tuberculosis

4. Barr JS (1947) Tuberculosis of the hip in children (discussion). *J Bone Joint Surg* 29:905
5. Chandler FA, Fox TA (1949) Amputation for discrepancy of limb length in tuberculosis of the hip. *J Bone Joint Surg Am* 31A:420–425
6. Gill AB (1947) Tuberculosis of the hip in children (discussion). *J Bone Joint Surg* 29:906
7. Gill GG (1944) The cause of discrepancy in the length of the limbs following tuberculosis of the hip in children. *J Bone Joint Surg* 26:272–281
8. Gill GG (1947) Tuberculosis of the hip in children (discussion). *J Bone Joint Surg* 29:905
9. Kestler OC (1947) Unclassified premature cessation of epiphyseal growth about the knee joint. *J Bone Joint Surg Am* 29:788–797
10. McCarroll HR, Heath RD (1947) Tuberculosis of the hip in children: certain roentgenographic manifestations, secondary changes in the extremity, and some suggestions for a program of therapy. *J Bone Joint Surg Am* 29:889–906
11. Parke W, Colvin OS, Almond AH (1949) Premature epiphyseal fusion at the knee in tuberculous disease of the hip. *J Bone Joint Surg Br* 31B:63–73
12. Sissons HA (1952) Osteoporosis and epiphyseal arrest in joint tuberculosis: an account of the histological changes in involved tissues. *J Bone Joint Surg Br* 34-B:275–290
13. Slee GC (1966) Premature fusion of the knee epiphyses (abstr). *J Bone Joint Surg* 48B:589

Poliomyelitis

14. Barr JS (1948) Growth and inequality of leg length in poliomyelitis. *N Engl J Med* 238:737–743
15. Barr JS, Stinchfield AJ, Reidy JA (1950) Sympathetic ganglionectomy and limb length in poliomyelitis. *J Bone Joint Surg Am* 32A:793–802
16. Currarino G (1966) Premature closure of epiphyses in the metatarsals and knees: a sequel of poliomyelitis. *Radiology* 87:424–428
17. Goetz RH, Du Toit JG, Swart BH (1955) Vascular changes in poliomyelitis and the effect of sympathectomy on bone growth. *Acta Med Scand Suppl* 306:56–83

18. Gullickson G, Olson M, Kottke FJ (1950) The effect of paralysis of one lower-extremity on bone growth. *Arch Phys Med Rehabil* 31:392–400
19. Harris RI, McDonald JL (1936) The effect of lumbar sympathectomy upon the growth of legs paralyzed by anterior poliomyelitis. *J Bone Joint Surg* 18:35–45
20. Janes JM, Jennings WK Jr (1961) Effect of induced arteriovenous fistula on leg length: 10-year observations. *Proc Staff Meet Mayo Clin* 36:1–11
21. Kai Lai S, Hahn Sheng P, Kao Wha C, Po Ling L (1994) Partial medial epiphysiodesis for genu valgum in poliomyelitis. *J Surg Assoc Republic China* 27(2):2261–2267
22. Makin M (1965) Tibio-fibular relationship in paralyzed limbs. *J Bone Joint Surg Br* 47:500–506
23. Mehta SN, Mukherjee AK (1991) Flexion osteotomy of the femur for genu recurvatum after poliomyelitis. *J Bone Joint Surg Br* 73(2):200–202
24. Prick JJG (1958) On morbidly changed bloodvessels as the central aspect of the pathological anatomy of poliomyelitis, also in connection with clinical observations. *Folia Psychiatr Neurol Neurochir Neerl* 61:593–604
25. Ratliff AHC (1959) The short leg in poliomyelitis. *J Bone Joint Surg Br* 41-B:56–69
26. Ring PA (1958) Prognosis of limb inequality following paralytic poliomyelitis. *Lancet* 2:1306–1308
27. Ring PA (1957) Shortening and paralysis in poliomyelitis. *Lancet* 273:980–983
28. Stinchfield AJ, Reidy JA, Barr JS (1949) Prediction of unequal growth of the lower extremities in anterior poliomyelitis. *J Bone Joint Surg Am* 31A:478–486, 500
29. Troup H (1961) Nervous and vascular influence on longitudinal growth of bone. An experimental study on rabbits. *Acta Orthop Scand Suppl* 51:1–78
30. Westin GW, Dingeman RD, Gausewitz SH (1988) The results of tenodesis of the tendo achillis to the fibula for paralytic pes calcaneus. *J Bone Joint Surg Am* 70(3):320–328
37. Navascués JA, Gonzalez-Lopez JL, Lopez-Valverde S, Soletto J, Rodriguez-Durantez JA, Garcia-Trevijano JL (2000) Premature physal closure after tibial diaphyseal fractures in adolescents. *J Pediatr Orthop* 20:193–196
38. Poznanski AK (1978) Diagnostic clues in the growing ends of bones. *J Can Assoc Radiol* 29:7–21
39. Ratliff AHC (1970) Complications after fracture of the femoral neck in children and their treatment (abstr). *J Bone Joint Surg* 52B:175
40. Botting TDJ, Scrase WH (1965) Premature epiphysal fusion at the knee complicating prolonged immobilization for congenital dislocation of the hip. *J Bone Joint Surg Br* 47:280–282

Developmental Dislocation of the Hip

Perthes Disease

41. Barnes JM (1980) Premature epiphyseal closure in Perthes' disease. *J Bone Joint Surg* 62B:432–437
42. Shapiro F (1982) Legg-Calve-Perthes disuse: a study of lower extremity length discrepancies and skeletal maturation. *Acta Orthop Scand* 53(3):437–444

Osteomyelitis

43. Caffey J (1970) Traumatic cupping of the metaphysis of growing bones. *Am J Roentgenol Radium Ther Nucl Med* 108:451–460

Slipped Capital Femoral Epiphysis

44. Betz RR, Steel HH, Emper WD, Huss GK, Clancy M (1990) Treatment of slipped capital femoral epiphysis. Spica cast immobilization. *J Bone Joint Surg Am* 72(4):587–600
45. Meier MC, Meyer LC, Ferguson RL (1992) Treatment of slipped capital femoral epiphysis with a spica cast. *J Bone Joint Surg Am* 74(10):1522–1529

Chemically Induced Immobilization

46. Osborne AC, Lamb KJ, Lewthwaite JC, Dowthwaite GP, Pitsillides AA (2002) Short-term rigid and flaccid paralyses diminish growth of embryonic chick limbs and abrogate joint cavity formation but differentially preserve pre-cavitated joints. *J Musculoskelet Neuronal Interact* 2(5):448–456

Diaphyseal Fracture

31. Beals RK (1990) Premature closure of the physis following diaphyseal fractures. *J Pediatr Orthop* 10:717–720
32. Chapchal G (1979) Late complications of fractures in children. *Reconstr Surg Traumatol* 17:130–138
33. Gonzalez-Lopez JL (2000) Letter to the editors. *J Pediatr Orthop* 20:826
34. Hresko MT, Kasser JR (1989) Physal arrest about the knee associated with non-physal fractures in the lower extremity. *J Bone Joint Surg Am* 71:698–703
35. Hunter LY, Hensinger RN (1978) Premature monomelic growth arrest following fracture of the femoral shaft. *J Bone Joint Surg Am* 60:850–852
36. Morton KS, Starr DE (1964) Closure of the anterior portion of the upper tibial epiphysis as a complication of tibial-shaft fracture. *J Bone Joint Surg Am* 46:570–574

Physeal Injury Other Than Fracture

Peterson, H.A.

2012, XXV, 428 p. 109 illus., Hardcover

ISBN: 978-3-642-22562-8