YEAR BOOK

YEAR BOOK OF ORTHOPEDICS® 1989

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The Year Book of ORTHOPEDICS®

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Acta Chirurgica Scandinavica

Acta Orthopaedica Scandinavica

Acta Radiologica

American Journal of Medical Sciences

American Journal of Clinical Nutrition

American Journal of Neuroradiology

American Journal of Obstetrics and Gynecology

American Journal of Roentgenology

American Journal of Sports Medicine

American Journal of Surgery

Annals of Emergency Medicine

Archives of Orthopedic and Traumatic Surgery

Archives of Surgery

Arthroscopy

Bone

British Journal of Plastic Surgery

British Journal of Radiology

British Medical Journal

Calcified Tissue International

Canadian Journal of Surgery

Cancer

Clinical Nuclear Medicine

Clinical Orthopaedics and Related Research

Clinical Radiology

Developmental Medicine and Child Neurology

Foot and Ankle

French Journal of Orthopedic Surgery

Injury

International Orthopaedics

Journal of Arthroplasty

Journal of Bone and Joint Surgery (American volume)

Journal of Bone and Joint Surgery (British volume)

Journal de Chirurgie

Journal of Clinical Endocrinology and Metabolism

Journal of Computer Assisted Tomography

Journal of Hand Surgery (American)

Journal of Nuclear Medicine

Journal of Occupational Medicine

Journal of Oral and Maxillofacial Surgery

Journal of Orthopaedic Research

Journal of Orthopedic Trauma

Journal of Pediatric Orthopedics

Journal de Radiologie

Journal of the Royal College of Surgeons of Edinburgh

Journal of Trauma

Journal of Vascular Surgery

Medical Journal of Australia

Medicine and Science in Sports and Exercise

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Neurosurgery

New England Journal of Medicine

Orthopedics

Pain

Pediatric Infectious Disease

Radiology

RoFo: Fortschritte auf dem Gebiete der Rontgenstrahlen und der

Nuklearmedizin

Scandinavian Journal of Plastic and Reconstructive Surgery

Scandinavian Journal of Rheumatology

Schweizerische Medizinische Wochenschrift

Semaine des Hopitaux

Skeletal Radiology

Southern Medical Journal

Spine

Surgical Pathology

Virchows Archiv A: Pathological Anatomy and Histopathology

Publisher's Preface

We are delighted to welcome Clement B. Sledge, M.D., and his associates, Robert Poss, M.D., Robert H. Cofield, M.D., John W. Frymoyer, M.D., Richard H. Gelberman, M.D., Paul P. Griffin, M.D., Sigvard T. Hansen, M.D., Kenneth A. Johnson, M.D., and Dempsey S. Springfield, M.D., as editors of the YEAR BOOK OF ORTHOPEDICS.

This team is carrying on the tradition of distinguished editorial direction for this YEAR BOOK, commencing with the 1989 edition. We congratulate them and extend our appreciation for their superb work with the YEAR BOOK.

Introduction

This edition of the YEAR BOOK OF ORTHOPEDICS represents the first edition in 13 years that has not been under the able editorship of Mark B. Coventry. Some acts are difficult to follow; this one cannot be followed. Few people have the encyclopedic knowledge of orthopedics that Mark Coventry's years of experience have produced. The most obvious corroboration of that fact is the editorial reorganization that has been put in place to replace him. As I take over the editor's job, I am fortunate to have the able assistance of co-editor Robert Poss, M.D. (Adult Reconstruction, Lower Extremity and Metabolic Bone Disease) and associate editors Robert H. Cofield, M.D. (Shoulder, Arm, and Elbow), John W. Frymoyer, M.D. (Spine), Richard H. Gelberman, M.D. (Hand), Paul P. Griffin, M.D. (Pediatrics), Sigvard T. Hansen, Jr., M.D. (Trauma [fractures, healing, amputations, prosthetics]), Kenneth A. Johnson, M.D. (Foot and Ankle), and Dempsey S. Springfield, M.D. (Musculoskeletal Neoplasia). You can see how many people it takes to replace a renaissance orthopedist like Mark Coventry.

C.B. Sledge, M.D.

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1 Pediatrics

Introduction

Pediatric orthopedic conditions continue to arouse controversy with regard to etiologies and the preferred methods of treatment. Articles reporting on a number of such entities are included in this section, notably, conditions such as slipped capital femoral epiphysis, fracture of the tibial

spine, and congenital dislocation of the hip.

The association of abnormal levels of growth hormone and testosterone in patients with a slipped epiphysis and no clinical evidence of an endocrinopathy is reported for the first time, but levels of these hormones were normal in another report of patients with a slipped epiphysis. From the evidence submitted I cannot explain the difference between these two studies. Such differences come from either sampling techniques or methods of laboratory measurements. Because the production of growth hormone is diurnal, the method of sampling is important. Measurement of somatomedin-C is more dependable in evaluating the physiology of growth hormone, as somatomedin-C is protein bound and more stable. Further studies will be necessary to clarify the relationship of hormonal abnormalities and slipping of the capital femoral epiphysis. The mechanical factor in slipping of the capital femoral epiphysis is addressed in this section. Forces across the physis are influenced by weight and the epiphyseal neck and shaft relationship. Retroversion significantly increases the shear forces across the physis.

For years there have been varying opinions about the effectiveness of closed and open treatment of fractures of the tibial spine. A new classification is evaluated and appears to address the issue of the success in re-

duction techniques and prognosis.

Use of the Pavlik harness, acetabular development, and surgical treatment of congenital dislocation of the hip are discussed in several abstracts in this section. The criteria for use of the Pavlik harness and an aggressive surgical approach to treatment in the older child are reported in great detail.

Paul P. Griffin, M.D.

Hormonal Studies in Patients With Slipped Capital Femoral Epiphysis Without Evidence of Endocrinopathy

Mann DC, Weddington J, Richton S (Cook County Hosp, Chicago) J Pediatr Orthop 8:543–545, September–October 1988

1 - 1

Several investigators have supported the concept that a hormonal imbalance caused by an endocrine disturbance may cause or contribute to

Average and Range of Investigated Hormones								
Hormone	M (11)	F (9)						
T4	9.14 (6.1–12.6)	8.7 (6.6–10.3)						
T3RIA T3RU	168 (82–243) 28.23 (25–30.2)	193 (128–259) 28.8 (27.1–30.8)						
TSH	2.3 (0.5-4.1)	2.69 (0.2-5.2)						
Growth hormone Somatomedin-C	2.12 (0.1–9.3) 2.1 (0.57–3.0)	0.36 (0.1–1.0) 1.72 (0.49–3.0)						
Testosterone	1.39 (0.1–5.2)	Not applicable						
Cortisol	13.8 (5.1–23.7)	16 (3.4–25.3)						
Ca	(9.6–10.4)	(9.5–10.3)						
Phos Alk phos	(3.9–6.2) (117–391)	(4.9–6.1) (265–517)						

Abbreviations: TSH, thyroid-stimulating hormone; Ca, calcium; Phos, phosphorus, Alk phos, alkaline phosphatase.

(Courtesy of Mann DC, Weddington J, Richton S: J Pediatr Orthop 8:543-545, September-October 1988.)

the occurrence of slipped capital femoral epiphysis (SCFE) during the adolescent growth spurt. However, the only available prospective study of growth hormone levels among adolescents with SCFE reported abnormal hormone levels but found no evidence of endocrinopathy. A prospective study was undertaken to identify any occult endocrinopathy among 20 adolescents with confirmed SCFE.

Plasma levels of triidothyronine (T₃), thyroxine, thyroid-stimulating hormone, cortisol, testosterone, growth hormone, somatomedin-C, calcium, phosphates, and alkaline phosphatase were measured in 11 boys (average age, 13 years) and in 9 girls (average age, 11 years), including 8 who were premenarchal, with clinical and radiographic evidence of SCFE. None of the patients had a history or physical evidence of endocrinopathy. In all, 190 laboratory values were determined, 130 of which were hormonal values.

Only 1 alkaline phosphatase value and 4 hormonal values were abnormal, including 1 low cortisol value, 1 elevated growth hormone value, and 2 elevated T₃ values (table). However, none of the patients with abnormal values had any clinical evidence of endocrinopathy, and none of the patients had more than 1 abnormal laboratory value each. Moreover, the abnormal values were all close to the normal ranges and were considered outlying ranges.

An SCFE is not associated with any identifiable endocrine abnormality. Routine sampling of these hormones in patients without clinical evidence of endocrinopathy is not warranted.

▶ It is known that endocrinopathies such as hypothyroidism, hypogonadism, and hypopituitarism are associated with an increased incidence of SCFE. Patients without endocrinopathies had not been found to have abnormal levels of testosterone or growth hormone until the report by Weiner and Leighley (1). The difference between these 2 reports needs further investigation as to techniques for sampling and laboratory analysis.—P.P. Griffin, M.D.

Reference

1. Weiner DS, Leighley CJ: Pediatr Orthop 8:180-196, 1988.

Maturation Factors in Slipped Capital Femoral Epiphysis

Wilcox PG, Weiner DS, Leighley B (Akron Children's Hosp, Akron, Ohio) J Pediatr Orthop 8:196-200, March-April 1988

1 - 2

The causes of slipped capital femoral epiphysis (SCFE), a disease of adolescence, are unclear. Of various theories proposed, hormonal imbalance and biomechanical factors seem most likely. The records of 191 patients were reviewed retrospectively to examine the relationship between an SCFE and the following factors: bone age, thyroid function, sex and growth hormone levels, and height and weight.

In 38% of the patients skeletal age was delayed as compared with chronologic age. Twenty-one percent had accelerated bone age. Although most patients had normal thyroid function, 7 had clinical evidence of hypothyroidism with marked decreases in triiodothyronine (T₃) or thyroxine and the free thyroid index. Of 80 patients, 20 had low levels of T₃. Of 64 patients tested, 76% had depressed testosterone levels, and 87% had low growth hormone levels.

Data were available on height and weight for 138 patients. Ninety-nine were above the 80th percentile for weight, with half of these at or above the 100th percentile. Sixty measured at or above the 80th percentile for

height.

The height and weight excess seen in this group suggests a relative hormonal imbalance, as does the evidence for delayed sexual maturation. Slipping of the capital femoral epiphysis seems to result from the combination of delicate hormonal imbalance and biomechanical stress induced by obesity in the young adolescent.

▶ The etiology of SCFE has to be multifactorial, with a combination of biologic factors that cause weakness in the physis and mechanical changes that cause sufficient shear forces to displace the epiphysis. It is interesting that the majority of patients studied in this series had low levels of growth hormone, because SCFE has been reported after the institution of treatment with growth hormone for disorders of the pituitary. Others have not found these abnormal hormone levels (1).—P.P. Griffin, M.D.

Reference

1. Mann DC, Weddington J, Richtors S: J Pediatr Orthop 8:543-545, 1988.

Open Reduction of the Severely Slipped Upper Femoral Epiphysis

Broughton NS, Todd RC, Dunn DM, Angel JC (Black Notley Hosp, Braintree, England)

J Bone Joint Surg [Br] 70-B:435-438, May 1988

Results of 70 Operations for	r Chronic Slip With an	Open Growth Plate	(50 boys, 20 girls)
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	Good	Fair	Poor
Subjective Clinical Radiographic	62 59 50	1 3 10	7 8 10
50 Normal Early complications (13%)		natic, full range, fai	
2 Avascular necrosis alone 1 Avascular necrosis and chondrolysis 5 Chondrolysis alone	olysis		
1 Subluxation after trivial injury 95% confidence interval 3.4-14.6 (5% to 21%)		osteoarthritis after	

Note: Two patients had pain of unknown cause.

(Courtesy of Broughton NS, Todd, RC, Dunn DM, et al: *J Bone Joint Surg [Br]* 70-B:435-438, May 1988.

Management is difficult when the angulation between the severely slipped upper femoral epiphysis and the femoral neck is more than 50 degrees. Open reduction of the epiphysis as the best way to avoid hip deformity in adolescence and prevent osteoarthritis in later life was evaluated. In all, 115 open reductions were performed on 110 patients.

On lateral radiography, the average angulation of the epiphysis to the femoral neck was 61 degrees, with a range of 33–90 degrees; only 15 hips had an angle of less than 50 degrees. Seventy hips had a chronic slip with an open growth plate, 38 had an acute-on-chronic slip, and 7 had a chronic slip with a partially fused growth plate. Patients were followed for an average of almost 13 years.

The incidence of complications was low in hips with a chronic slip and an open growth plate: in 5 patients chondrolysis developed; 2 others had avascular necrosis; 1 patient had both complications (table). Patients with acute-on-chronic slip had more complications: 6 had avascular necrosis, and 1, chondrolysis; 3 had both complications. Only 1 patient with a partially fused plate did well. Complications developed within the first year in all cases; however, osteoarthritis developed in 3 hips within 10-20 years after surgery.

Open reduction of the chronic severely slipped upper femoral epiphysis with an open growth plate is an acceptable procedure. Results in acute-on-chronic cases are similar to those with other procedures. Open reduction is not recommended for patients with a partially fused growth plate; a Griffith-type osteotomy is preferable.

▶ The high incidence of vascular necrosis and chondrolysis after open reduction or cunieform osteotomy is well recorded in the literature and is considered risky at least. Long-term results after treatment of a slipped upper femoral epiphysis are best in patients who do not have reduction of the slip (1). Fish (2) is the only author recently to publish good results from cuneiform osteotomy. It is not an operation for the uninitiated, and in situ fixation followed later by a

proximal femoral osteotomy, if needed, is more likely to be successful in most instances.—P.P. Griffin, M.D.

References

- 1. Boyer DW, et al: J Bone Joint Surg 63A:1153-1168, 1985.
- 2. Fish IB: I Bone Joint Surg 66A:1153-1168, 1984.

Mechanical Factors in Slipped Capital Femoral Epiphysis

Pritchett JW, Perdue KD (Univ of Washington; Arizona Children's Hosp, Phoe-

J Pediatr Orthop 8:385-388, July-August 1988

1 - 4

Several theories have been advanced to explain slipped capital femoral epiphysis (SCFE), but those that involve mechanical stress seem the most logical. Three-dimensional force analysis was performed and the expected sheer force was calculated during various activities to determine the point at which mechanical failure of the growth plate is likely to occur.

Fifty normal adolescents and 50 with an SCFE were studied. All of the latter patients had normal height, but 23 were overweight. The shear strength and load required to produce shear failure of the growth plate were calculated. Shear stress was calculated for slow walking, fast walking, and running.

In normal patients the calculated shear load to produce failure of the

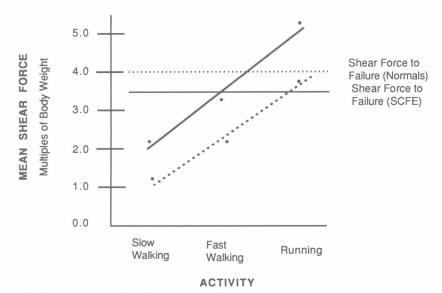


Fig 1-1.—Increase in shear force on proximal femoral growth plate as function of activity for patients with normal (dotted line) and slipped epiphyses (solid line). SCFE, slipped capital femoral epiphysis. (Courtesy of Pritchett, JW, Perdue KD: J Pediatr Orthop 8:385-388, July-August 1988.)

growth plate ranged from 3.7 to 5.5 times body weight. The mean shear force in slow walking was 1.4 times body weight; in fast walking it was 2.2 times body weight, and for running it was 3.6 times body weight (Fig 1–1). In patients with an SCFE the shear load to produce failure of the growth plate ranged from 2.6 to 4.3 times body weight, a range 15% lower than that for normal patients. Study patients also had a decreased neck shaft-plate shaft angle. By assuming a 10-degree relative retroversion, a mean shear force of 2.2 times body weight was calculated for slow walking. Fast walking generated a shear force of 3.3 times body weight, and running had a shear force of 5.1 times body weight. Fast walking generated a shear force above the critical level for a slip in some patients, and when running, all patients were at risk for a slip.

Patients with a slipped epiphysis apparently have subtle anatomical variations that reduce the growth plate's resistance to shear forces. Mechanical factors alone can cause shear failure in these patients during

running. Obesity is a contributing factor.

▶ Mechanical forces are part of the etiology of an SCFE. The shear forces across the growth plate are increased by retroversion. Patients with a SCFE generally have decreased anteversion (1). In obese children anteversion rapidly diminishes during growth and many have retroversion (2). However, not all obese children have an SCFE. I have found that those that do have an SCFE have less internal rotation of the hip in flexion than in extension before the slip occurs, as shown by careful examination on the normal hip in unilateral slips. The cause of this limited motion must contribute to the slip. Obesity and retroversion do not explain the limitation before slip.—P.P. Griffin, M.D.

References

- 1. Gelberman RH, et al: J Bone Joint Surg 68A:1000-1007, 1968.
- 2. Kelsey JL: Am J Dis Child 124:276-281, 1972.

The Predictability of Acetabular Development After Closed Reduction for Congenital Dislocation of the Hip

Brougham DI, Broughton NS, Cole WG, Menelaus MB (Royal Children's Hosp, Melbourne)

J Bone Joint Surg [Br] 70-B:733-736, November 1988

1 - 5

The development of the acetabulum may not be satisfactory after closed reduction of a congenital dislocation of the hip (CDH). An early, reliable radiologic guide for predicting subsequent acetabular dysplasia would enable early surgery on acetabula with a poor prognosis, thus avoiding unnecessary operations. A study was done to determine which pelvic radiographic measure can reliably predict adequate acetabular development after closed reduction. Serial radiographs of 63 hips in 53 children who had received treatment by closed reduction for CDH were reviewed. All of the children had been followed for more than 7 years. At the last review 34 hips were dysplastic.

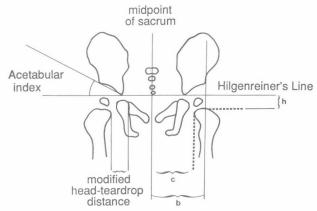


Fig 1–2.—Radiologic measurements used in assessment of pelvic radiograph. (Courtesy of Brougham DI, Broughton NS, Cole WG, et al: *J Bone Joint Surg [Br]* 70-B:733–736, November 1988.)

Failure to obtain concentric reduction or its loss by femoral head migration within 1 year of the surgery were the best predictors of persisting acetabular dysplasia and were best quantified by the h/b ratio (Fig 1-2). The median acetabular index at closed reduction in hips with acetabula that developed normally was 40 degrees and in those with persistent dysplasia, it was 43 degrees—a nonsignificant difference. Dysplasia tended to be more common in hips in which the acetabular index decreased less than 10 degrees in the first year after closed reduction, but this difference was also nonsignificant. The average age at which the acetabulum stopped developing was 5 years, with a range of 17 months to 8 years (Fig 1-3).

Failure to obtain a concentric reduction and early migration of the femoral head, as evidenced by an abnormal h/b ratio within 1 year of closed reduction, are the most accurate predictors of inadequate acetabular development. The acetabular index was not helpful in predicting per-

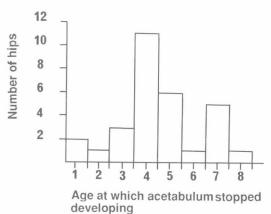


Fig 1-3.—Age at which acetabulum stopped developing in 30 children who had regular radiographs and no acetabular surgery. (Courtesy of Brougham DI, Broughton NS, Cole WG, et al: J Bone Joint Surg [Br] 70-B:733-736, November 1988.)

sistent dysplasia, whether the index was used at reduction or 12 months later.

▶ Acetabular development in CDH is critical for a successful result. Much controversy and uncertainty persist as to the age at which the acetabulum stops developing. I believe that the critical factor in CDH is stability. In the presence of instability the acetabulum will not improve. The ratio of h/b, described by Smith in 1968, is an excellent indicator of prognosis, because it reflects either failure of reduction or progressive upward and lateral displacement, either of prevents acetabular development and requires surgical intervention.—P.P. Griffin, M.D.

Neonatal Screening for Congenital Dislocation of the Hip: A Prospective 21-Year Survey

Hadlow V (Taranaki Base Hosp, New Plymouth, New Zealand) J Bone Joint Surg [Br] 70-B:740-743, November 1988

1 - 6

In 1948 Ortolani described an abduction test for diagnosing congenital dislocation of the hip (CDH) in otherwise normal children and adults. In 1962 Barlow devised an additional, slightly different abduction test for use in neonates. However, opinions about the reliability of these tests have varied widely. In 1964 a prospective screening program for CDH in neonates was initiated in New Plymouth, New Zealand. Findings in this 21-year survey were reviewed.

During the study period 20,657 newborns were examined for CDH by an orthopedic surgeon within the first 7 days of life. Both the Ortolani and Barlow tests were used for screening. Initially, 672 infants (3.2%) were considered to have CDH, but in 341 of these the hips stabilized completely within 5 days. The remaining 331 infants (1.6%) required splintage for instability or frank dislocation. Because early in the study 2 hips were still unstable after 6 weeks of splinting, the splinting period was extended to 8 weeks. One infant later found to have been splinted in the unreduced position eventually required open reduction at age 12 months. One child had established unilateral dislocation of the hip at age 15 months and another had dislocation at 18 months. Both children received treatment with closed reduction and pelvic osteotomy and had acceptable results. Both cases had been missed at initial examination by an inexperienced examiner. Thus there was 1 splintage failure and 2 diagnostic failures.

None of the infants with stable hips at neonatal examination had CDH during the first year of life, and no late cases have been reported. Of the 172 infants splinted during the last 10 years of the survey, 87 (50.5%) had bilateral instability and 85 (49.5%) had unilateral instability. Twenty-four (7.1%) of 336 breech presentations required splinting, an incidence of nearly 5 times that for vertex presentations (1.5%).

Although neonatal screening for CHD in neonates is simple to do, detection of CHD requires considerable expertise. This may account for the