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INSTRUCTIONS FOR AUTHORS

Any article relevant to orthopaedic surgery, orthopaedic science and the teaching of either will be considered by *The Iowa Orthopaedic Journal* for publication. Articles will be enthusiastically received from alumni, visitors to the department, members of the Iowa Orthopaedic Society, residents and friends of the University of Iowa Department of Orthopaedics. The journal will be published annually in May or June. The deadline for receipt of articles for the 1993 journal is February 1, 1992.

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1. The **original manuscript complete with illustrations.** The corresponding author must be clearly identified with address and telephone number. Manuscripts of accepted articles will not be returned.
2. A **bibliography,** alphabetical and double-spaced, or references made in text only. Refer to bibliographies in this copy of *The Journal* and follow style exactly.
3. **Legends** for all illustrations submitted, listed in order and typed double-spaced.
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EDITOR'S NOTE

The primary purpose of this Journal is education, and those who participate in its production undoubtedly learn the most. We have considered it a privilege to produce the Iowa Orthopaedic Journal and have gained a valuable experience in doing so. However, we hope that each reader is able to learn something new from the Journal and benefit from reading it.

As the demands on physician's time for clinical and administrative duties increase, the time available for research and education will decrease. Therefore, we greatly appreciate the efforts put forth by the authors in this Journal. We were particularly happy with the strong showing of the recent alumni, indicating continued support for the Journal and its purpose.

This is the 12th edition of the Iowa Orthopaedic Journal. The production of the Journal involves solicitation of manuscripts and advertising, editing, organizing and distributing the "final product". The resident editors are responsible for all of these duties with guidance from faculty advisors. We hope that it reflects the attitude of the Department of Orthopaedic Surgery at the University of Iowa and will help serve a model for future journal publications.

The Editors of the Iowa Orthopaedic Journal would like to thank our faculty advisors Dr. Reginald Cooper and Dr. Charles Clark. The Journal could not be produced without the excellent organizational and secretarial assistance provided by Laura Cole. The Journal is published under the administrative guidance of Paul Etre and Mrs. Kay Redlinger-Phillips.

This Journal includes publications in basic science, clinical research, review articles and case reports. We hope you as a reader will take interest in its content and will let it serve as a forum for discussion. We welcome your response and criticism.

Robert L. Bass, M.D.
Brian D. Mulliken, M.D.

JOURNAL DEDICATION TO DR. COOPER

Dr. Reginald R. Cooper

July, 1992 will mark Reginald R. Cooper's thirtieth year of service to the University of Iowa Department of Orthopaedics. For this devotion and much more we have chosen to dedicate this edition of the Iowa Orthopaedic Journal to Dr. Reginald R. Cooper.

Dr. Cooper was born on January 6, 1932 in Elkins, West Virginia. He graduated from Harman High School, Harman, West Virginia in May 1948. His undergraduate education included studies at Potomac State College of Keyser, West Virginia, and West Virginia University in Morgantown, West Virginia. Dr. Cooper attended West Virginia University School of Medicine and Medical College of Virginia, receiving his medical degree in May, 1955. Following graduation he began his general surgery internship in July 1955. The editor's appreciate the service provided during residency training, so perhaps we should be celebrating Dr. Cooper's thirty-seventh year of service to the Department of Orthopaedics! Dr. Cooper completed his orthopaedic residency training at Iowa in July, 1960. Subsequently, Dr. Cooper was an attending Orthopaedic Surgeon at the United States Naval Hospital in Pensacola, Florida from July, 1960 to July, 1962. He returned to the University of Iowa as an Associate in Orthopaedics in July, 1962. From September, 1964 to September, 1965, Dr. Cooper was a Research Fellow in Orthopaedic Surgery at John Hopkins Hospital in Baltimore, Maryland.

Dr. Cooper's academic ascension at Iowa included appointments as Assistant Professor of Orthopaedics in July, 1965, Associate Professor of Orthopaedics, July, 1968, Professor of Orthopaedics, July, 1971, and ultimately Chairman of the Department on November 12, 1973. Under his leadership, the department has flourished and the residency program has maintained its standard of excellence. During his tenure five department members have received the Kappa Delta Award and another five received ABC traveling fellowships.

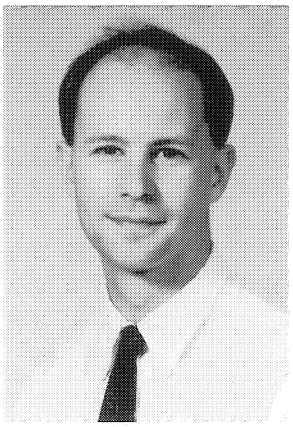
Dr. Cooper's academic achievements are many. He was awarded the Kappa Delta Award of Outstanding Basic Research in 1970 for his detailed electron microscopic evaluation of immobilization atrophy and regeneration of skeletal muscle in cats. Other landmark contributions included the electron microscopic investigation of the ultrastructure of compact bone in dogs, which lead to our current understanding of osteonal morphology. His other studies have included work on the ultrastructural morphology of secondary ossification centers and the mucopolysaccharidoses as well as much more.

Dr. Cooper has received more than 40 honorary degrees and visiting lectureships. He has been honored as an American Orthopaedic Association Exchange Fellow, President of the Orthopaedic Research Society, and a member of the Board of Directors of the American Academy of Orthopaedic Surgeons. His contributions to ensuring the current state and future of our profession culminated in the Presidency of the American Academy of Orthopaedic Surgeons, during 1986-1987. Although it would seem appropriate to list all of Dr. Cooper's contributions in this dedication, we would have double the size of this volume to accommodate the contents of his curriculum vitae. However, there is a great deal more to Dr. Cooper's commitment to Orthopaedics than could be justly written here. The pursuit of attaining excellence in orthopaedics and passing on that experience is an integral factor that makes Dr. Cooper the man and Chairman we have all come to know and respect. The editors, residents, faculty, and alumni wish to take this opportunity to express our thanks to Dr. Reginald R. Cooper for his many years of service and leadership.

1992 GRADUATING SENIOR RESIDENTS

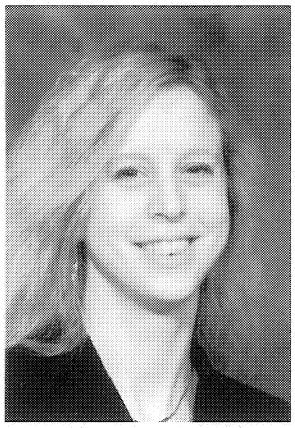
Thomas K. Wuest, M.D.

Thomas K. Wuest, M.D. was born in Cedar Rapids, IA on December 16, 1959. He lived in the Amana Colonies and Marshalltown, IA before entering the University of Iowa in 1977. There he earned B.S. degrees in Microbiology and Biology, studies in the Biology graduate program, and received his M.D. degree in 1987. Tom, his wife Megan, and daughter Veronika will travel to Great Britain for a one year fellowship at the North Staffordshire Royal Informary in Stoke-on-Trent. There he will be a Visiting Registrar in Orthopaedic Trauma studying under Professor John Templeton.



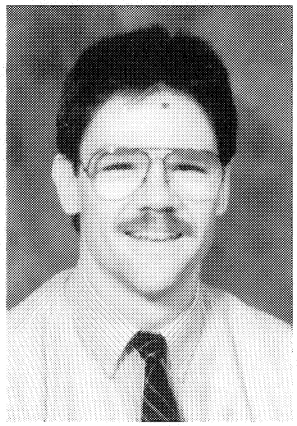
Devon D. Goetz

Devon was born in Kansas City, MO, on 12-14-60. His hometown is Creston, IA. Devon attended The University of Iowa, receiving his B.S. in 1983 in General Science and M.D. in 1984. Also to be congratulated are Kathy his wife and two lovely daughters, Katelyn and Kirsten. Next year Devon will be the William H. Harris Fellow in Adult Reconstruction at Massachusetts General Hospital/Harvard Medical School.



J. Sybil Biermann

Sybill received her B.S. at Cornell University and M.D. at Stanford University School of Medicine. Her plans for next year include a fellowship in Orthopaedic Oncology with Dr. Michael Simon at the University of Chicago.



Kenneth E. Newhouse

Ken was born in Cleveland, OH and raised in Chagrin Falls, OH. He graduated with a B.A. in Chemistry from Miami University in 1983. Subsequently he attended Yale University School of Medicine, receiving his M.D. in 1987. Next year Ken will be a fellow in Arthroscopy and Sports Medicine at the Salt Lake Knee and Sports Medicine Center under the guidance of Drs. Paulos and Rosenberg. After his fellowship Ken will practice Sports Medicine and general orthopaedics in Pocatello, Idado with Dr. Mickelson. He will be a member of medical staff caring for the athletes of Idaho State University.

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Curtis Steyers
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James Nepola
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James Weinstein
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Barbara Campbell
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William Pontarelli
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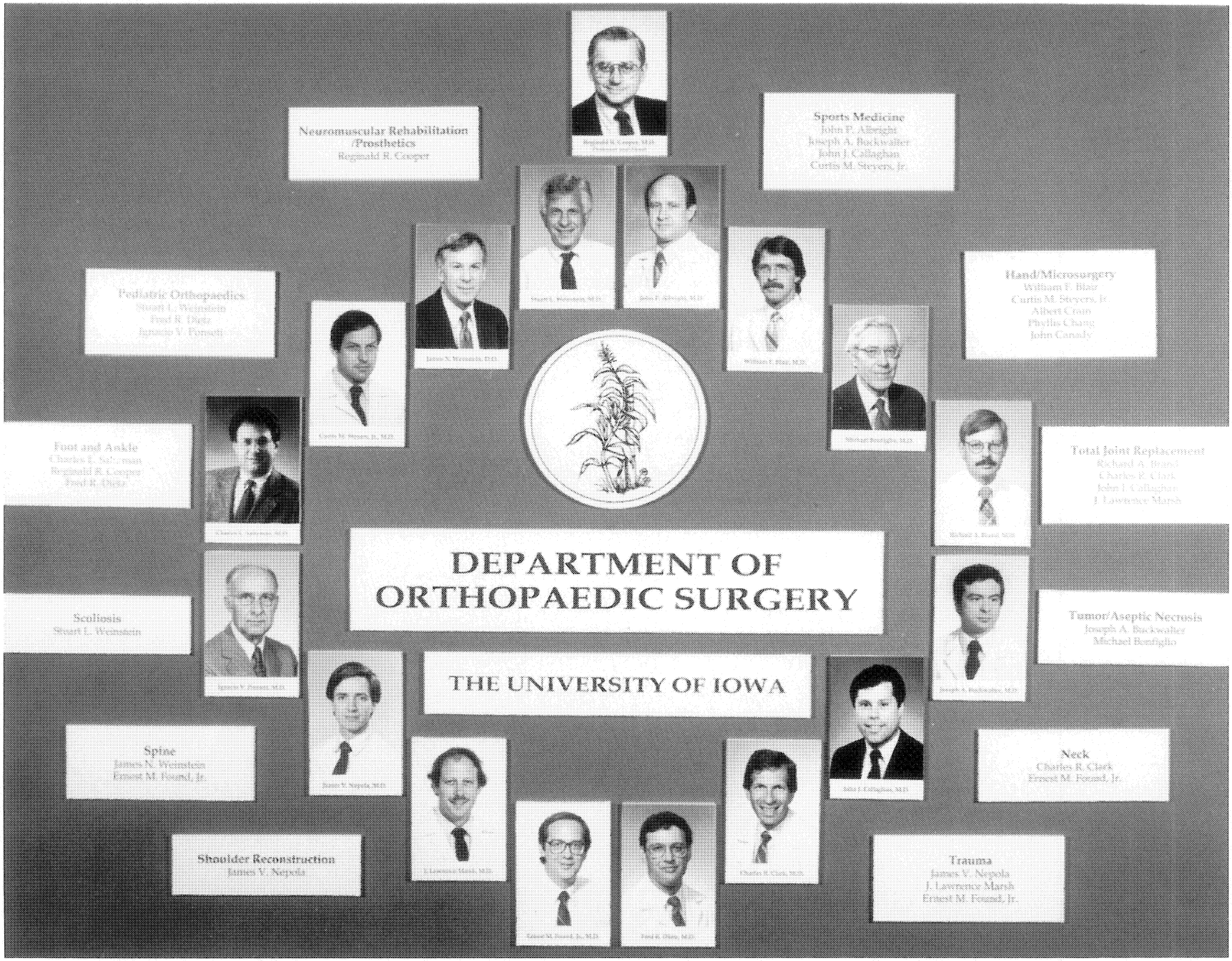
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Second Row (L to R):	Kary Schulte, Tom Wuest, Mark Creighton, Tim Ballard, Gene Thornburg, Doug Cooper, Joe Martin, Ken Newhouse, John Albright, Reginald Cooper
Third Row (L to R):	Curtis Steyers, Stuart Weinstein, Margaret Fehrle, Kristi Weber, George Emodi, Sybil Biermann, Scott Meyer, John Durham, Brian Mulliken, John Callaghan, Fred Dietz
Not Pictured:	J. Lawrence Marsh, Ernest Found, William Blair, Ken Noonan, Tom Fyda

SCHEUERMANN'S JUVENILE KYPHOSIS

*Aurelio G. Martinez, M.D.
†Stuart L. Weinstein, M.D.

From The Department of Orthopaedics,
University of Iowa

Juvenile kyphosis is defined as an abnormal increase in the posterior convexity of the thoracic or thoracolumbar spine, arising in puberty and characterized by marked rigidity and radiographic changes in the affected vertebrae.[1] It was first described by Scheuermann in 1920. The incidence of Scheuermann's kyphosis is thought to be about one percent with a slight female predominance.[1,4,6] Even though the condition was described more than 70 years ago, very little is known about its natural history.

Normal thoracic kyphosis is between 20 and 45 degrees, and increases with age. Kyphosis at the thoracolumbar junction should always be considered abnormal.[4]

The diagnosis of Scheuermann's kyphosis is usually made during the adolescent growth spurt and is rarely made under ten years of age.[1] The incidence of spondylolysis and spondylolisthesis is reportedly increased in patients with Scheuermann's kyphosis, as well as a reported 20 to 30% incidence of scoliosis in the region of the kyphosis.[4,9]

The cause of Scheuermann's juvenile kyphosis remains unclear. Many etiologies have been proposed including vascular, mechanical, metabolic, hereditary and endocrinologic factors.[1,4,6]

There are two types of thoracic Scheuermann's kyphosis[1]: one with the apex at the T7 to T9 level, and one with the apex in the lower thoracic spine at the thoracolumbar junction (T11-T12). There is generally an associated compensatory lumbar hyperlordosis. Another less common condition is the so-called "Lumbar Scheuermann's" with the apex at L1-L2. This condition is generally more common in young male athletes. It is thought to have a traumatic origin.[2]

Clinically, most patients with thoracic Scheuermann's kyphosis present with deformity. The incidence of pain in the adolescent is quite low, although about 20% of the patients will have some discomfort in the region of the kyphosis. In patients with the lumbar Scheuermann's the chief complaint is most often pain (80%). The pain is usually intermittent and characterized as dull or aching, activity related, and relieved by rest.[4]

The physical examination reveals the typical rigid kyphotic deformity. This is best demonstrated in the forward flexed position. The rigidity of the kyphosis can be demonstrated either by having the patient hyperextend from a prone position or having the patient sit hyperextended on a chair with their hands held behind their head. Lack of flexibility indicates the structural nature of the kyphotic deformity in contrast to patients with flexible "postural kyphosis". These patients also have a hyperlordosis in the lumbar spine.[1,4,14]

In the low thoracic Scheuermann's disease the kyphosis is at the thoracolumbar junction. There may be hypokyphosis above the thoracolumbar junction and hypolordosis in the lumbar spine.[4]

Hamstring tightness may be present in these patients. Because of the high association of scoliosis with Scheuermann's kyphosis this too must be assessed. The thoracic Scheuermann's patient may have tenderness to palpation above or below the apex of the kyphosis. In the lumbar variety tenderness to palpation is generally in the region of the curve apex.[4]

Each patient should have a careful neurologic examination. Although rare, with an extreme degree of kyphosis, neurologic deficit can occur. In addition, there is an association of epidural cysts causing spastic paraparesis in patients with Scheuermann's kyphosis.[4]

The diagnosis of Scheuermann's kyphosis is confirmed on a standing lateral radiograph of the spine. The radiograph should be taken with the arms parallel to the floor and the patient resting on a support. It is important to see the entire spine to measure the thoracic kyphosis, lumbar lordosis, and any secondary cervicothoracic curves which may accompany the kyphosis. Spondylolysis and spondylolisthesis should also be sought. The kyphosis is measured by determining the angle between the maximally titled end vertebrae (similar to the Cobb method for measuring scoliosis). A PA scoliosis film should be obtained to detect the presence and/or magnitude of any

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associated scoliosis. Also any evidence of interpedicular widening should be noted because of the association of this finding with epidural cysts.[1,4]

The radiographic diagnosis of Scheuermann's kyphosis is made by the presence of irregularities of the vertebral endplates, anterior vertebral wedging, Schmorl's nodes, and decreased intervertebral disc space height. In older patients degenerative changes may be evident.[1,4,6] There is some discrepancy in the literature as to the number of consecutive vertebrae that need to be wedged to make the diagnosis of Scheuermann's kyphosis. By one criteria (Sorensen) there should be wedging in three or more adjacent vertebrae of more than five degrees.[11] In other studies the diagnosis is made by the presence of only one wedged vertebrae of more than five degrees.[3,4,9] This compounds the problem of determining the natural history.

In the lumbar Scheuermann's, irregularities of the vertebral endplates are usually present as are Schmorl's nodes. The intervertebral disc spaces are normal and there is no evidence of vertebral wedging.[2]

In a patient with an apparent exaggerated kyphosis, the differential diagnosis includes postural round back. In postural round back there is a slight increase in the thoracic kyphosis. However, the kyphosis is flexible as demonstrated on the prone or sitting hyperextension tests. On the standing lateral radiograph there are no structural changes as noted for Scheuermann's kyphosis. The kyphosis is usually in the range of 45 to 60 degrees. On a hyperextension lateral view, the deformity is totally flexible.[1,3,4,6] The question remains as to whether a postural kyphosis, if left untreated, may progress and get secondary bony changes resembling Scheuermann's disease. Postural kyphosis should be treated by exercise.

Thoracic hyperkyphosis is also seen in patients with various skeletal dysplasias such as spondyloepiphyseal dysplasia and Morquio's disease. These conditions can usually be diagnosed by the history, clinical examination and other radiographic features. Ankylosing spondylitis may present a similar picture, but 97% of these patients will be HLA B27 positive. Kyphosis may also be present in patients who have had a laminectomy prior to skeletal maturity and patients who have had radiation to the spine for a regional tumor. Kyphosis may also be seen with eosinophilic granuloma. Type II congenital kyphosis (failure of segmentation) may be confused with Scheuermann's disease. In such cases, it may be necessary to get polytomograms to identify the anterior failure of segmentation seen in this condition. Infectious spondylitis and trauma may also result in an abnormal thoracic kyphosis.[1,4]

There is a paucity of natural history data available on Scheuermann's kyphosis. Many think that pain, if present, will subside with growth and there are few adverse long term sequelae of the condition.[8] Others postulate that the incidence of pain with Scheuermann's kyphosis increases throughout life, as may the deformity.[3,5] The pain in adults with Scheuermann's is generally described as the feeling of "tiredness" in the back. They may have pain in the hyperlordotic lumbar spine, or at the apex of the kyphosis because of ankylosis.[5]

In a recent review of our experience at The University of Iowa[8], we studied 67 patients with an average follow-up of 31.7 years; average age at follow-up was with 53 years with an average kyphosis of 71 degrees. Compared to controls no differences were found in work disability, work attendance, marital status, assertiveness, social function or recreational activities. Pain incidence was no greater than the general population but pain levels were mildly enhanced and tended to occur for longer periods. Patients often complained of back "tiredness" but neurologic compromise was rare. Pain did not correlate with the degree or level of kyphosis. Scoliosis was common, being found in 72% of the patients, with an average curve of 16 degrees. In contrast to the literature, spondylolisthesis was uncommon. Pulmonary function was above normal for curves up to 85 degrees, with restrictive disease occurring only when curves approached 110 degrees. Thus for most of these patients with Scheuermann's kyphosis, the natural history was generally benign and conservative management was satisfactory.

TREATMENT

Because of the paucity of natural history data it is difficult to formulate treatment plans. Some authors feel that the natural history of thoracic Scheuermann's kyphosis is generally benign and therefore rarely needs treatment.[8] Others report increasing pain with progression of the deformity.[3,5] Treatment of Scheuermann's kyphosis in the skeletally immature patient is generally recommended in hopes of preventing excessive deformity which may cause pain and cosmetic concerns.

The objectives of treatment in Scheuermann's kyphosis are to: 1) improve cosmesis 2) relieve pain 3) prevent increasing kyphosis. Not all patients with Scheuermann's kyphosis require treatment. For example, an asymptomatic skeletally mature patient, with an acceptable degree of kyphosis, should be observed.[6]

Factors to consider in treatment are: 1) age of patient 2) degree and rigidity of the deformity 3) location of the posterior convexity 4) presence or absence of pain 5) presence of scoliosis and 6) psychosocial factors (cosmesis).[3,9]

Treatment options include:
A) Milwaukee Brace
B) Antigravity Corrective Plaster Followed by Brace
C) Underarm Braces
D) Surgery
E) Exercises
F) Electrical Stimulation

Only the first four alternatives have demonstrated efficacy in the management of this problem. However, indications for treatment remain unclear, since little is known about the natural history of the disease; there is insufficient data concerning the response of pain to current methods of treatment, and significant loss of correction after conservative and surgical treatment is common.[8]

A) MILWAUKEE BRACE. This is the most common form of treatment in North America.[3,9] A good Milwaukee brace should maintain pelvic tilt to eliminate lumbar lordosis. It has two posterior kyphosis pads and a neck ring that is centered above the thorax.[6] The most common mistake is to have the neck ring forward, perpetuating the kyphosis. This is usually used in conjunction with an exercise program. After a few weeks, any pain associated with the condition usually disappears and significant height is gained, requiring adjustment of the orthosis. The deformity is usually corrected in four to eight weeks if it is flexible. After this, the goal is to maintain the correction and allow the soft tissues and the vertebrae to "readjust" and grow more normally. The typical patient requires one year (and preferably 18 months) of full time (23-24 hours a day) use, combined with a supervised physical therapy program to maintain and improve thoracic extensors and to decrease lumbar lordosis.[14] A lateral standing view of the whole spine in the brace is recommended every four to six months to allow objective measurement of the correction and appropriate brace adjustment. At the end of one year of full time brace wear, weaning can gradually be instituted (2 to 4 hours a day), if the deformity remains corrected, even though full growth may not have been reached. Some authors recommend starting the weaning period only after the vertebral wedging is less than five degrees in order to prevent loss of correction.[4] Weaning can be continued as long as correction is maintained. The periods of time out of the brace should be increased until the brace is only used at night for four to six months. If there is a loss of correction at any time, the weaning process should be slowed. In general, the weaning process is similar to the one utilized for the treatment of idiopathic scoliosis.[4]

Bradford and associates[3] treated 75 patients with a brace for an average of 34 months. They reported a 40% average correction of the kyphosis, 41% average correction of the vertebral wedging and 36% improvement of lumbar lordosis. Loss of correction was only six degrees after 34 months. However, in a more recent long term follow-up study from the same Center, a more than ten degree loss of correction was seen after an average follow-up of eight years.[9] In another report, Montgomery and Erwin[7] noted a 15 degree loss of correction after 18 months of brace wear. There are currently no studies on part time bracing in Scheuermann's kyphosis.

B) CAST TREATMENT - THE LYON METHOD (Proposed by Stagnara), involves the correction of the kyphosis by antigravity plaster casts and its maintenance with a plexidur brace. Several antigravity hyperextension casts are applied, usually during a total period of four to 12 months, followed by full-time wear of a hyperextension orthosis until the wedging of the vertebrae is corrected. Then a similar weaning program as the one mentioned for the Milwaukee brace is followed.[4]

Hyperextension casts may be applied in two stages. In the first stage the lordosis is corrected by positioning of the patient bent forward while the lower part of the cast is applied; in the second stage, the patient stands erect, extending the cast to the upper part of the trunk, correcting the kyphosis. Another method involves applying the cast on a Risser scoliosis table with the patient supine. The lower part of the cast is applied first with the hips flexed to correct lumbar lordosis. Then the upper part of the cast is applied with a vertical suspension strap at and below the kyphosis. After the desired correction with casts has been achieved a plastic hyperextension orthosis is used.[4]

The results obtained by these two methods (Milwaukee brace and Cast treatment) are similar. The average correction of the kyphosis is between 30 and 40%, and that of the vertebral wedging is between 40 and 50%. However, more than 60% of the correction has been lost after 15 years of follow-up in the cases of De Mauroy and Stagnara using the cast treatment.[1] This is similar to the experience of Sachs, Bradford and co-workers after an average eight year follow-up using the Milwaukee brace.[9] Since both methods achieve similar results the use of the Milwaukee brace is recommended because it is cosmetically more acceptable, it can be removed for bathing, hygiene, sports or certain special events and the patient can be gradually weaned.

C) UNDERARM BRACES: These may be effective in the treatment of thoracic kyphosis. They are useful in the management of kyphosis with an apex below T-9, and particularly useful for thoracolumbar and lumbar kyphosis.[6] The main advantage of this type of brace is that they are more cosmetically acceptable to the patient and their families and compliance may be increased. No studies have yet been published on this method.

D) SURGERY: Surgery is rarely if ever necessary in Scheuermann's kyphosis. Indications for surgery remain

unclear.[5,12] Surgery may be recommended for an absolute curve value such as 65 or 75 degrees or vertebral wedging of more than 10 degrees, with expectations that the curve may progress and cause future problems.[12] Indications considered by other authors include progressive kyphosis above 60 degrees in spite of brace treatment, pain unresponsive in spite of brace treatment, pain unresponsive to all conservative methods and adults who present with residual deformity.[14]

Posterior fusion alone leaves the fusion mass under tension and hence is associated with a high rate of complications (loss of correction and pseudoarthrosis). The recommended surgical approach is a combined anterior and posterior spinal fusion. The operation consists of an anterior release of the thickened and hypertrophied anterior longitudinal ligament and removal of the intervertebral discs at the apex of the convexity. The posterior annulus and the posterior longitudinal ligament are left intact. Small corticocancellous bone grafts are packed into the disc spaces. Two weeks later posterior spinal fusion and instrumentation is performed. Some surgeons may prefer, depending on the conditions of the patient, doing the combined surgery in the same setting. The classic form of instrumentation has been two heavy Harrington compression rods. If there is significant associated scoliosis, a distraction rod is used on the concave side and a compression rod on the convex side.[14] Other methods of instrumentation currently used are: Harrington plus sublaminar wiring, Luque segmental instrumentation, and more recently Cotrell-Dubousset, Texas Scottish Rite Hospital (TSRH), and other newer instrumentation systems. Postoperative immobilization depends on the type of instrumentation, the quality of bone and the surgeons preference. Some authors recommend either cast or brace immobilization for a period of six months or until a solid arthrodesis has been achieved.[12]

E) EXERCISES: Exercises are not effective if used alone. They should be used in combination with brace treatment and are aimed at decreasing the lumbar lordosis through pelvic tilt exercises, increasing thoracic muscle tone, improving posture and decreasing hamstring tightness.

F) ELECTRICAL STIMULATION: There is not enough data supporting the effectiveness of electrical stimulation.

In summary, Scheuermann's juvenile kyphosis is a relatively benign condition. It occurs in about 1% of the general population, with a slight female predominance. It is characterized by a fixed thoracic kyphosis and specific radiographic changes. A slight increase in the duration and intensity of back pain may be expected in the adult life, as well as residual hyperkyphosis compared to that found in the general population. Associated mild scoliosis is common. Juvenile kyphosis can generally be managed successfully with conservative measures. The most common form of treatment in North America is the Milwaukee brace with a supplemental exercise program.

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LIGAMENT HEALING
A REVIEW OF SOME CURRENT CLINICAL AND EXPERIMENTAL CONCEPTS

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INTRODUCTION

Injuries to ligaments in and around virtually all major joints are common and remain a source of concern to the surgeons who must treat them. Treatments vary from non-operative methods to surgery, depending on a number of variables including which joint is injured, which ligament is injured, and the willingness of both the patient and the surgeon to undertake operative procedures without a guarantee of success. Surgeons often lower the expectations of the injured patient to less-than-perfect results after serious ligament injuries, so that long term functional deficits are not misinterpreted as "therapeutic mistakes". Some failures are expected even in the most expert hands[10,11,18,26] and many experts recognize that early "successes" may slowly deteriorate.[11] Surgeons must therefore use words like "variable" and "unpredictable", when attempting to explain that a given procedure may be indicated.

Variation in clinical approaches and ambiguous qualifications of results would clearly not exist if ligament healing were completely understood, predictable and controllable. While this is still not the case, there is a substantial and steadily increasing body of knowledge about ligament structure and function,[1-3,6] and why ligaments appear to heal in some circumstances and not in others.

Here, we present a brief review of some clinical and experimental concepts of ligament healing, as currently understood. References to several key articles are provided so that the reader may better understand the history and evolution of this interesting area. For a more complete background, several reviews are recommended which devote more space to detailed descriptions of the structure and function of normal and healing ligament.[1-3,6,13,14]

Clinical Concepts

Historically, ligament injuries have been recognized as a source of clinical concern, resulting in a number of reviews of pathology and outcomes.[25,26] Isolated ligament injuries have a better chance of functional recovery than do combined ligament injuries. This "clinical principle" has been rediscovered several times,[26] and is one of the major determinants in predicting outcome. Beyond the seemingly obvious reason of "increased tissue damage" resulting in "impairment of the quality or quantity of the healing process", there are no clear explanations why combined injuries should "heal more poorly". In a subsequent section we will introduce some specific measures of healing quality and quantity: keys to translating clinical outcomes into definable and testable terms.

TABLE I

Clinical Concepts in Ligament Healing

1. Combined injuries have poorer prognosis.
2. Different ligaments heal differently.
3. Ligament healing is variable and unpredictable.
4. Ligament healing is slow and can fail late.

A second "clinical principle" of ligament healing is that not all ligaments have equal healing potential. Injury to the anterior cruciate ligament (ACL) of the knee, now recognized more frequently due to improved physical examination, MRI and arthroscopic capabilities,[3] is the classic example of a ligament which has a particularly poor prognosis for healing. Numerous reviews of ACL injuries have shown a predisposition for recurrent knee injury, progressive intra-articular meniscal and hyaline cartilage damage, progressive joint instability and, in some cases the gradual development of osteoarthritis.[4,11] This progression has been called "the natural history of the ACL deficient knee", and is attributed to the inability of the ACL to heal. Another less-considered possibility is that an ACL healing response may be present, but inadequate to reestablish the normal function of the ACL. Contrasted with this is the ability of the medial collateral ligament (MCL) of the knee to heal and provide adequate knee joint stability. Either the MCL heals or its function is somehow replaced by other knee structures, such that the "natural history" of untreated MCL injuries is much more favorable.[10,18] Clinical evidence supports a combination of

these two factors (superior healing plus better compensation) in explaining the superiority of outcomes of MCL injures versus ACL injuries. Valgus instability appears to be better tolerated than rotatory instability of the knee, suggesting that joint dynamics are an important factor in judging ligament healing. "Equal healing" of an MCL and an ACL, measured by scar quality and quantity for example, may not give "equal joint symptoms" or equal joint stability, due to potential compensation. Definitions of "clinical healing" must therefore be separated from actual "ligament healing" at the tissue level.

A third "clinical principle" of ligament healing stems from the observation that the same apparent ligament injury (eg. a complete or grade III, isolated MCL injury) does not heal equally (or predictably) in different people. Many grade III MCL injuries result in excellent joint stability and function, even with relatively simple "aggressive conservative" measures of bracing and therapy.[10,18] However, some do not. Even in those series reporting a high incidence of early clinical success, some failures of healing were found for reasons which were not clear.[10,18] Conversely, most isolated ACL injuries have resulted in relatively poor outcomes. Apparent healing in ACLs is certainly a relative exception, but it has been reported.[25] It is tempting to explain these apparent "failures of MCL healing" or "successes of ACL healing" on either misdiagnoses of original injuries, misinterpretation of clinical outcomes, or on rare peculiarities of healing in some individuals. While all of these explanations are tenable, a fourth possibility could also be entertained—that some set of environmental and/or physiological factors are important to prevent healing of some ligaments or to allow healing of others. Such factors may be present and potentially controllable in every case.

Differences in apposition of torn ends has been speculated to cause important qualitative differences in ligament healing since O'Donoghue's classical works in the 1950s and 1960s.[25-28] Differences in the location of injury in ligament complexes and differences in mechanical loading conditions during the healing phase have also been proposed. Recently, it has been postulated that neurophysiological feedback loops may or may not be preserved after ligament injuries, providing variable protection of the joint and of healing ligaments.[3,4]

The last "clinical principle" is that ligament healing is a slow process which can also fail slowly. Early "good results" after a ligament repair can be misguided. Failure of ligament repairs have been reported up to 3-5 years from the time of surgery, because the healing process may actually "fail over time". Other possibilities include the potential of failing "compensation" by other structures or for delayed detection. It is almost impossible at the present time to separate these possibilities clinically.

EXPERIMENTAL CONCEPTS

I. Background

Translating clinical results into experimental terms is difficult but essential in attempting to understand ligament healing. First, it is important to recognize that "clinical results" must be subdivided into two general dimensions —physiologic and non-physiologic. The non-physiologic dimension refers to the psychological, sociological and patient-specific variables which collectively determine the ability to "recover completely" from an injury. Patients' expectations regarding their recovery from an injury and psychological adjustments to injury may be unrealistic. We will not address these crucial factors in interpreting clinical studies, other than to point out that such factors exist and contribute very strongly to the subjective scoring scales that attempt to rate successes or failures of ligament healing.

"Physiologic measures" of ligament healing refer to those biological, physiological, biochemical, morphological, or biomechanical measures which have a quantitative or qualitative meaning in defining how a ligament has healed, how the joint functions, and how the limb function has "recovered". We define "healing" of a ligament not in the dictionary sense of "restoring the integrity of the original", but in completely replacing important normal functions of that ligament. While normal ligament structure (ie. morphology, biochemistry) would clearly achieve this goal, we accept that alternate structures, or alternate adaptive mechanisms (eg. remodeling of an entire joint, altered neuromuscular reflex patterns) may result in normal function without having a normal ligament. It is equally critical to maintain an appropriate perspective of the experimental measures of ligament healing which focus only on healing tissue, rather than on the functioning of the joint or of the individual. The vast majority of historical studies on ligament healing in animal models have concentrated on the reductionistic definition of success—ie. normality of the healing ligament itself.

More than just the ligament in question changes over time after a ligament injury. Perhaps virtually every other structure in the affected joint changes its function in response. Sometimes this is manifested as gross pathological changes (eg. meniscal tears) but probably more often as microscopic alterations. This spectrum of changes supports the need for a spectrum of research approaches. We will pursue this concept that "no structure works in isolation", suggesting that virtually all structures in and around a joint may adapt after even the most "isolated" injury.

Due to limitations in space, we will now define and discuss only those biomechanical concepts that have been used to quantify ligament healing. Again, the reader is encouraged to review comprehensive works

on other measures to put this form of measurement of ligament healing outcome into its proper perspective.[1,3,7,9,12,16,19,28,32]

i. Joint Laxity or Ligament Laxity

In clinical terms "joint laxity" refers to a subjective impression of the movements of one bone relative to the other when that joint is manipulated or displaced by intrinsic muscle forces and moments. Laxity is a relative term and is often based on a comparison with the contralateral joint or normal external controls (other individuals). In some cases, laxity can be quantified, if a single direction of displacement can be related to the application of a specific force or moment.[21] Such displacements of a joint under low loads are thought to correlate with the patients' feelings of instability, and with the development of joint damage and possibly osteoarthritis.

In biomechanical terms, "joint laxity" can be a quantitative measure of joint motion into one (or more) of six independent degrees of freedom of motion and relates these motions to specific forces or moments which are causing a displacement. In a whole joint there are many structures which contribute to its force-deformation characteristics in any given direction. Some structures are obviously more critical in resisting certain displacements than others. The MCL, for example, is the major structure which resists valgus forces applied to the knee.[19,21,32] In its absence, the ACL becomes more important in resisting valgus forces.[17,33]

For the purpose of assessing ligament healing, it is important to account either for the relative contribution of that structure or to "isolate" a structure such that it is the only structure resisting a load. Due to the number of unknowns and the potential for changes in contributions of "other structures" over time in biological experiments, most experiments employ the "isolation" technique.[4-9,16,17,20,27,29-31] Only a few have quantified whole joint stiffness.[16,33] In all cases, the quantitative measure being compared can be referred to as a "low load stiffness". Generally, laxity is measured when loads are very low (as compared with failure load of a structure) in the so-called "toe region" of the load-displacement curve (Figure 1).

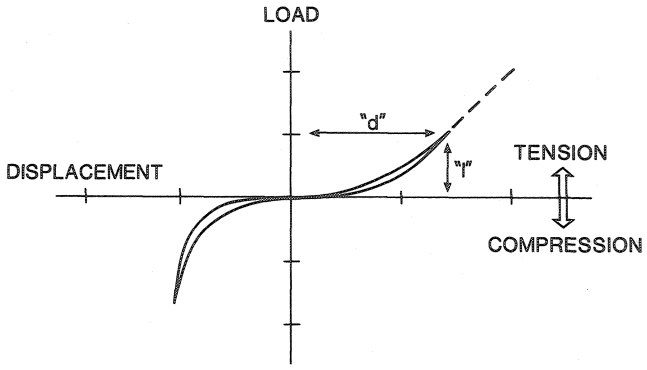

Figure 1
"Laxity" is a measure of low load stiffness of the joint or tissue. The dashed line shows higher load stiffnesses that cannot be tested safely and without destruction in vivo.

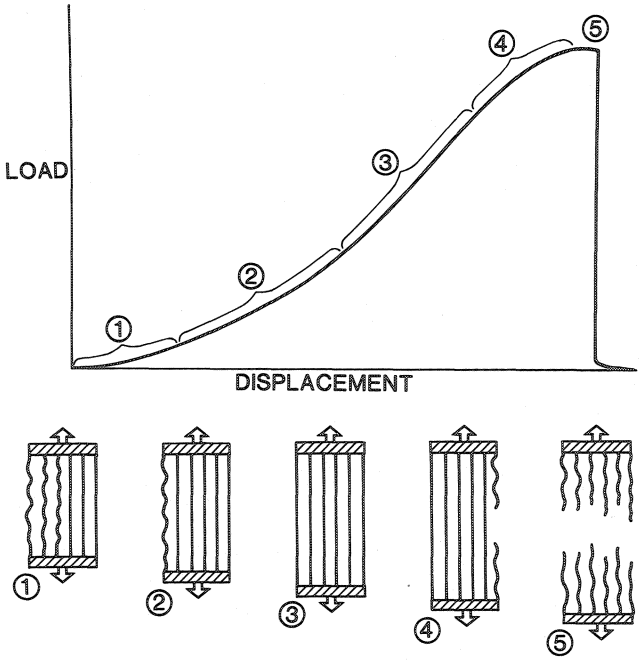

Figure 2
Tensile testing of a joint or of a tissue shows non-linear load-deformation behavior. At least part of the reason for this non-linearity is the phenomenon of "recruitment". Slack or "crimp" of fibers is removed during displacement until all fibers are loaded. These recruitment patterns are joint angle- and test-specific.

ii. Structural Stiffness and Structural Strength

These are terms which refer to higher load stiffnesses of tissues and their failure behaviors. At low loads, tensile behaviors of ligaments are non-linear, attributed to the phenomenon known as "fiber recruitment". This recruitment is shown conceptually in Figure 2. The term "structural" refers to the behavior of the entire structure (tissue or joint) and is not normalized for the size of the specimen. A large specimen of the same material would be stiffer (require more load to cause an equal deformation and deform less under a given load) and stronger than a small specimen of that material. Structure also relates to the ligament "complex" which consists of bony insertions as well as midsubstance ligament tissue. Failure of this "structure" may therefore occur in any of the structural regions (ie. insertion site or midsubstance).

Structural strength refers to the load at which the entire structure being tested fails. In a typical midsubstance failure, fibers fail progressively as the structure begins to yield and then are completely disrupted by continued

displacement (Figure 2). Less is known about the mechanism of "histological" failure at the ligament insertion sites.

iii. Material Behavior

Material behaviors of a tissue are structural behaviors which have been normalized for the size of the specimen and restricted to the "material" of interest (ie. midsubstance). For example, a tensile stress-strain curve describes the characteristics of a portion of a ligament loaded in tension where its cross-sectional area and the tensile strain can be measured.

iv. Viscoelastic Behavior

Many materials exhibit stress-strain behavior which is time and load-history dependent. Ligaments, for example, demonstrate both creep and load relaxation behavior as they are loaded in tension. "Creep" refers to increasing deformation over time under a constant load, while "load-relaxation" refers to a decrease in load in a tissue which is being held at a constant deformation. Similar changes take place when the tissue is cycled in tension, but at different rates. For a more complete description of these phenomena in ligaments, the reader is encouraged to pursue specific references.[2,6]

v. Ligament "Function"

While all these mechanical descriptors of ligament function are useful under certain circumstances, they do not necessarily reflect properties which are relevant or vital in vivo. Our current speculation, for example, is that "high load behaviors" (ie. any failure descriptors, high load stiffnesses) are relatively meaningless—since we, like others, believe that many ligaments are rarely loaded to those extremes. Instead, ligaments more likely function normally over relatively low loads—ie. in the so-called "toe region" of the load-deformation or stress-strain curves. In those ranges, the viscoelastic behavior is unique and tends to be more viscous-dominated. Measurement of loads and strains becomes difficult in these ranges in vitro, let alone in vivo. Due to our fundamental belief that these measures and better definitions of "functional laxity" are critical in understanding normal (and healing) ligament, we plan to put considerable effort in this area.

Experimental Concepts

With this background, several important concepts are now reviewed which have emerged in experimental work on ligament healing. Much of this work must be qualified as being both ligament- and model-specific, and dependent on the test conditions utilized. Nevertheless, the number of consistencies in the results suggests that there are, in fact, some "principles" of ligament healing. These will, of course, need to be tested in various ways with other models to establish their validity and reliability.

i. Gaps in Disrupted Ligaments Heal with "Scar Tissue"

Since the earliest experiments on ligament healing,[20,23,24] there has been abundant evidence that gaps in ligaments can be filled with tissue which is grossly, histologically, biochemically and biomechanically similar to "scar tissue". Descriptions of scar tissue in skin and tendon are the basis of this observation, with consistent patterns of formation, maturation and remodeling.

Briefly, gaps in any of these tissues fill first with blood. An inflammatory phase follows, in which inflammatory cells are recruited and fibroblasts begin to proliferate. Torn ends become revascularized and the gaps are filled with scar tissue. Cell proliferation is then further enhanced. Cells produce a matrix which is composed of various types of collagen, proteoglycans, other proteins and glycoproteins which are produced and assembled in different proportions, at different times during the healing process. This proliferative phase merges into a very long (months to years) remodeling phase in which these matrix elements are rearranged or removed, as the ligament ends become more distinctly "reconnected". The weak load carrying ability of the disorganized scar slowly evolves to a stronger and more discrete load-carrying ligament structure. The morphologic and biochemical details are described in detail elsewhere.[3,7,12]

ii. Gap Size Influences at Least the Rate of Scar Maturation

Several references have stated that minimizing the gap between ligament ends appears to modify the healing process. O'Donoghue[27] described this as a more "orderly process", with "collagenization" rather than "scar formation".

We have recently compared gap healing to healing of a Z-plasty (minimized gap) in the midsubstance of an adult rabbit MCL, and found that there are some mechanical advantages to having the cut ends in contact.[7] Improvements in structural strength parallel those in structural stiffness, with the Z-plasty configuration being about 25-35% stronger at all intervals of healing to 40 weeks (Figure 3A). There is a slight material advantage to contact healing as well, particularly at later healing intervals (Figure 3B). Laxity measures, as defined earlier, showed that both contact and gap healing ligaments were similar after 6 weeks and were within contralateral control limits by 40 weeks.[7] Longer term follow-up of these parameters is currently underway as no "endpoints" have yet been truly identified.

We speculate that structural differences are due to the presence of more frequent and/or larger "defects" in scars

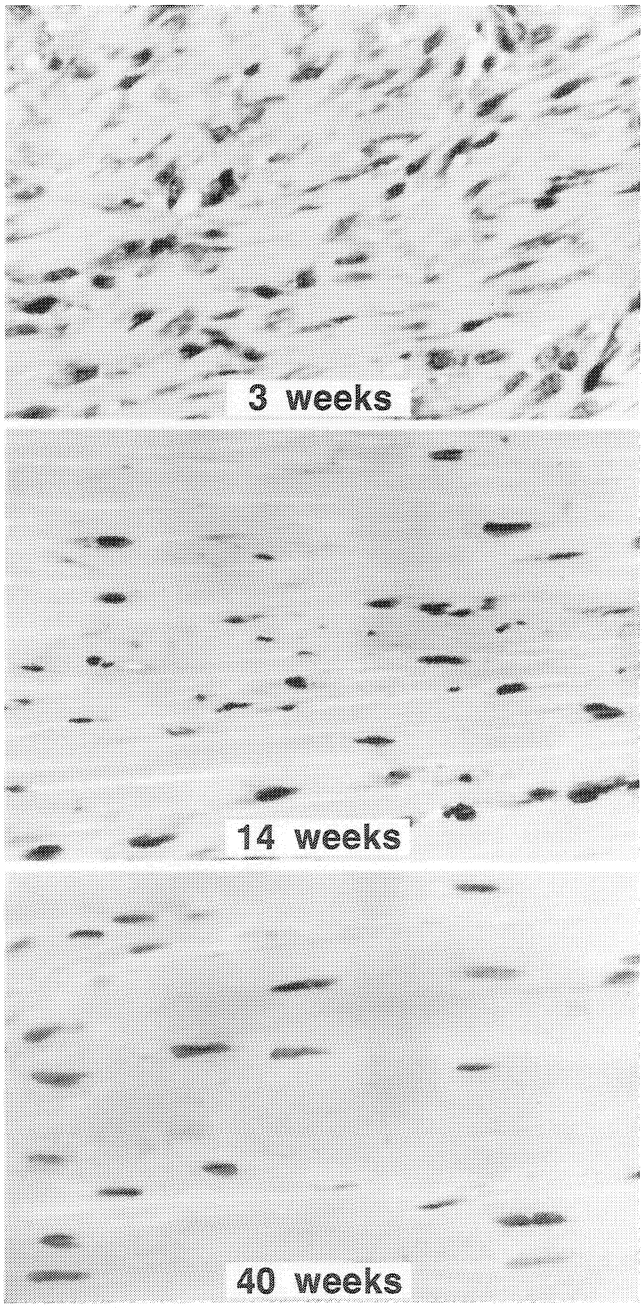

Figure 3A
A comparison of healing strengths of 4 mm midsubstance gaps with contact Z-plasties in adult rabbit MCLs. Note some structural advantage to the contact healing at each interval.

from gap healing ligaments. We are presently attempting to define and quantify such abnormalities in matrix organization that would likely represent early sites of stress concentration and thus scar failures, at least at high loads.

iii. Different Ligaments Heal Differently; At Least Quantitatively

Not all ligaments heal at the same rate, in the same way, or to the same endpoints. The best example of two

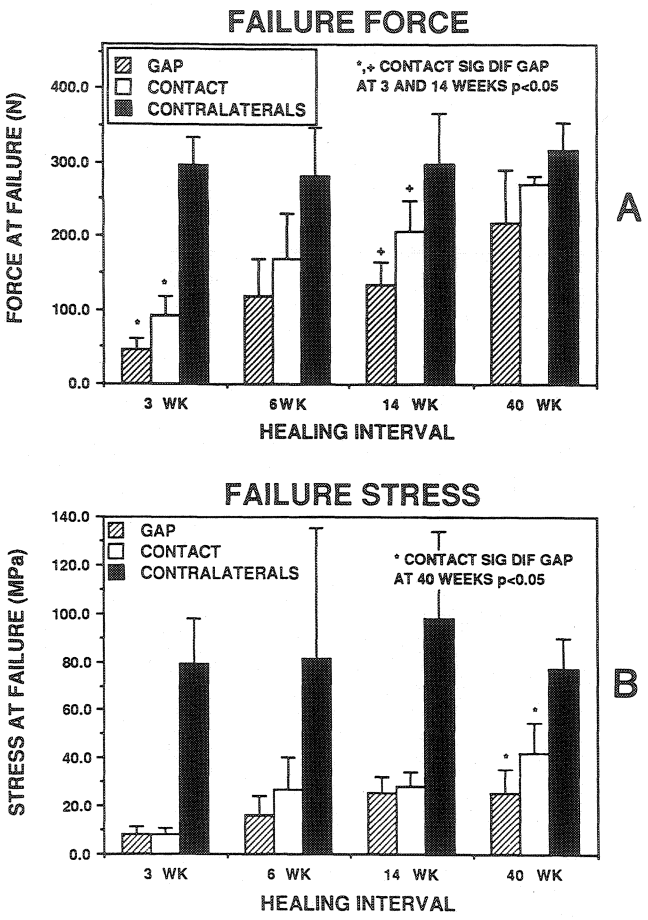

Figure 3B
A comparison of stresses at failure of these same groups.

extremes are the MCL and the ACL of the knee joint. The MCL has been shown to heal at different rates in a number of animal models, using a number of outcome measures.[4,5,17,19,29,30] In general, the healing MCL in the rabbit does not achieve all normal biomechanical behaviors within the first year after injury. However in the dog, nearly "complete" healing can occur as early as 12-24 weeks after injury.[16] The MCL's progressive improvement toward normal in different models suggests that "adequate" healing of that ligament does eventually occur in many species.

The ACL, on the other hand, heals much less predictably. Various animal models have shown that complete transections of the ACL have a very low chance of healing.[4,22,28,33] Instead, cut ACL ends are often resorbed. In some cases, however, *some* healing of the ACL *has* occurred.[28,29] As with the MCL, the quantity and quality of this healing response appears to be structurally inferior to the normal ACL. To our knowledge the "quality" of this healing (as expressed by material properties) has not been defined.

A quantitative comparison of material healing qualities between the ACL and MCL would help determine whether they heal by similar processes or not and how each responds to different therapeutic manipulations (biological, environmental and/or mechanical).

iv. Healing Ligaments are Affected by the Presence or Absence of Joint Motion

As with normal dense connective tissue,[13] healing ligaments responds to joint motion by forming more scar tissue—causing them to be structurally stronger and stiffer than an immobilized counterpart.[4,5,17,31] This "principle" appears to apply to extraarticular ligaments such as the MCL where containment of the scar may be a prerequisite to cellular proliferation. Joint motion appears to distract the torn ACL and may actually prevent scarring. Whether such distraction of ends prevents scarring, or the forces cause disruption of scar, or whether the ACL forms inferior scar tissue remains unknown.

The effect of motion on the qualities of scar tissue is more controversial. Extrinsic forces have long been thought to be required for "remodeling" of ligament scars.[30] While this is likely true in some circumstances, we have recently found that matrix alignment is independent of joint motion, at least in the immature rabbit MCL.[15] We are reluctant to speculate any further on this phenomenon until the processes of matrix deposition and alignment are better understood.

v. Combined Ligament Injuries Heal with Inferior Rates and/or Quality than Isolated Injuries

Several animal models have shown that there is an "interaction" between ligament injuries; combined injuries do not heal as quickly as isolated injuries. Combined injuries potentially have different healing endpoints, and most of these endpoints remain to be defined quantitatively.

Our experience in a rabbit model[4,5] has shown that combined ACL/MCL injuries result in inferior structural and material properties of the healing MCL compared with those of the "stable" model (Figure 4). This probably relates to a combination of at least two factors: relative immobility of animals with painfully unstable knees or subluxed joints (there was a high incidence of patellar dislocations in ACL/MCL injured rabbits) and excessive forces placed on the MCL healing tissue in the ACL deficient knee. Each of these speculations requires experimental proof and quantification.

SUMMARY AND CONCLUSIONS

An overview of the clinical and experimental concepts of ligament healing shows many important similarities. Ligaments show some healing potential in both humans and in animal models. Different ligaments do have different healing potential and combined ligament injuries do not heal as quickly as isolated injuries, due to a speculated combination of biological and biomechanical reasons. Distance between torn ends is one factor which appears to affect ligament scar quality, and joint motion may affect both quality and quantity.

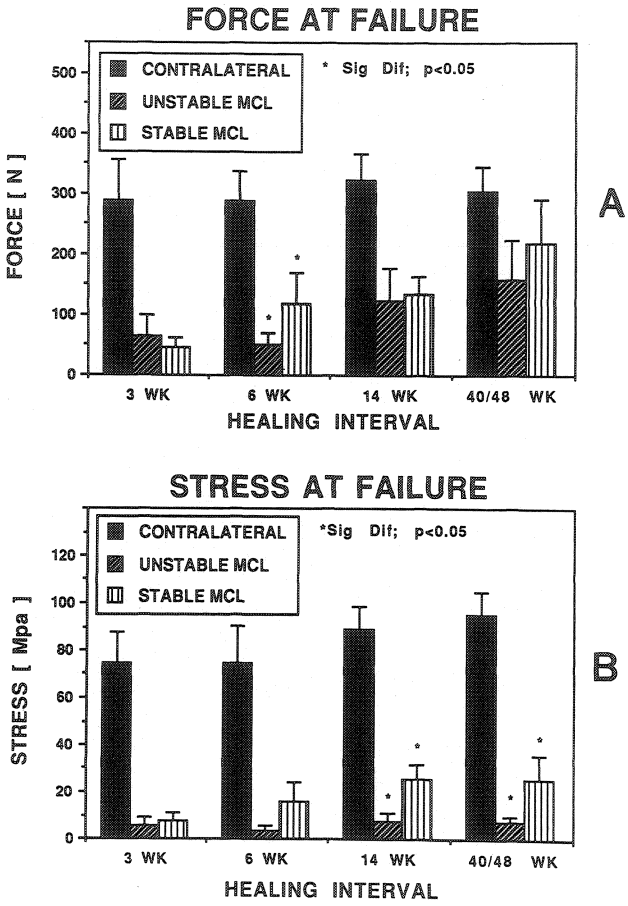

Figure 4
A comparison of structural and material failure properties of gap healing MCLs in "stable" (MCL only injured) versus "unstable" (MCL/ACL injured) rabbit knees at four intervals of healing.

Despite these "principles" being defined in gross terms in both humans and in animal models, there remains a significant gap between the clinical and experimental definitions of "successful ligament healing". Clinical assessments rely too completely on global joint function while experimental results may be too focused on individual tissue characteristics. These fields are converging with clinicians beginning to isolate and test individual structures, while animal investigators are beginning to address the impact of ligament healing on whole joint function. However, a significant effort is still clearly required by both groups of investigators to standardize terminology and to design appropriate experiments to test not just the

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phenomena of ligament healing, but also the mechanisms behind the "principles" noted above.

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DISTAL FIBULAR STABILIZATION WITH A MODIFIED KNOWLES PIN

George A. Brown, M.D.
Amanda Copus

ABSTRACT

Intramedullary fixation of the distal fibula has been used routinely by the first author (G.A.B.) for fifteen years. Twenty patients with ankle fractures fixed with a modified Knowles pin (Howmedica®) in the intramedullary canal of the distal fibula were evaluated as a follow-up study to one conducted in 1984 on thirty-three patients. The 1984 study showed successful stabilization of the injured ankles by use of the modified Knowles, Deyerle, and Steinman pins. No patient had a measurable change in fibular fracture alignment or position of the intramedullary device. There was a 100% union rate and no infections. The modified Knowles pin appears to be a successful device for internal fixation of the lateral malleolus and allows early return to full function.

INTRODUCTION

Anatomic reduction and maintenance of the reduction until healing are essential steps in restoration of normal function to fractured ankles.[1,2,3,4,9,10] Significant residual talar displacement leads to unsatisfactory results.[1,5,17] Only one mm of lateral displacement may cause a 42% reduction in the area of contact between the tibia and talus, as demonstrated by Ramsey.[14] Further, the stress per unit area increases as the total contact area decreases.

Current literature stresses the importance of open reduction in displaced ankle fractures,[7,8] and the importance of fibular reduction in fractures involving the lateral malleolus. The fibula is the key to diagnosis and treatment of ankle fractures.[1,6,8,11,12,15,18] Yablon demonstrated that the talus faithfully follows the displaced lateral malleolus in unstable bimalleolar fractures. Consequently, the talus will be accurately positioned if the lateral malleolus is anatomically reduced.[19]

An ideal fibular fixation device should be easily implanted, rigid enough to afford very early mobilization, maintain reduction accurately without migrating, have a perfect rate of union with minimal infection rate, and not have to be removed. Plating the fibula, as recommended by Miller, et al and the AO group,[11] has been shown to work well. However, the small amount of subcutaneous tissue over the plate may lead to lateral ankle discomfort.[16,17] Additionally, we feared an increased infection rate when plating long bones, and had concern about screw loosening in osteopenic bone. Plate breakage can occur, especially in comminuted fractures using the tubular plate. Intramedullary fixation with the Rush pin leaves a protruding portion of the pin at the tip of the fibula and a Steinman pin may be difficult to remove.

During the past fifteen years, the first author (G.A.B) has used the modified Knowles pin and the Deyerle pin for displaced distal fibular fractures. Both devices were originally designed for internal fixation of femoral neck fractures, but offer significant advantages for fibular fixation: (1) small diameter (1/8 inch) with adequate lengths (3-6 inches); (2) self-tapping for easy insertion; and (3) a small head that would allow removal but does not protrude significantly from the tip of the fibula. Fibular fixation using these devices was a technique originally performed at the University of Iowa in 1975. A follow-up study from 1976 to 1981 showed the modified Knowles, Deyerle, and Steinman pins were effective intramedullary devices for fibular fixation. There was 100% union, no infections, and no detectable loss of reduction in thirty-three patients with short follow-up (Table 1).

This paper involves twenty additional patients with an average follow-up of four years. Sixteen patients were followed more than two years and all were followed to stable fracture union.

METHODS & MATERIALS

The modified Knowles (Howmedica®) pin is an intramedullary device originally intended for use in femoral neck fractures. The pin is composed of a chrome-cobalt alloy (Vitallium®) and has the proper diameter to fit the intramedullary canal of the fibula. It has enough flexibility to contour with the medullary canal, yet it is strong enough to maintain fracture alignment. It is also self-tapping, and comes in 3, 4, 5 and 6 inch lengths with a standard 1/4 inch hexagonal head and a detachable drive shaft so it can be easily inserted with a hand drill. Only the 1/4 inch socket wrench is needed to remove the device.

Surgical Technique

The patient is placed in the supine position. Betadine is used to prep the skin and tourniquet control of bleeding is utilized. A posterolateral incision is made over the fracture site. For pin insertion, a separate incision or an extension of the primary incision distal to the fibula is used, depending on the location of the fracture. Because accurate stable reduction of the fibula is required, the fracture site must be exposed and a stable reduction obtained before insertion of the intramedullary device. Small Lane self-locking bone clamps are helpful. A site at the tip of the fibula is

TABLE 1

		1984	Current Study
1.	CAUSE		
	Falls	25	15
	Accidents	5	2
	Sports	4	3
2.	FRACTURE TYPE		
	Bimalleolar	15	6
	Trimalleolar	13	10
	Distal Fibula	5	4
	Tear Syndesmosis	4	2
3.	PIN		
	Deyerle	21	0
	Steinman	4	0
	Modified Howmedica Knowles	8	20
4.	AGE		
	15-19	1	0
	20-29	11	0
	30-39	5	5
	40-49	4	8
	50-59	6	1
	60-79	6	6
5.	SEX		
	Male	19	13
	Female	14	7
6.	LENGTH OF FOLLOWUP		
	< Year	17	0
	> Year	16	20
7.	RESULTS		
	No Problems	25	16
	Minimal Pain	8	3
	Mild Loss of Flexion	0	3
8.	RADIOGRAPHIC EVIDENCE OF FRACTURE UNION	33	20
9.	POSTOPERATIVE INFECTION	0	0
	DETECTABLE CHANGE IN POSITION OF PIN OR FX		
10.	ALIGNMENT AT FOLLOWUP	0	0

aligned with the proximal medullary canal. It is important to stay directly over the tip of the fibula. The self-tapping modified Knowles pin will go through the soft medullary portion of the distal fibula. If accurately directed, the pin will go easily up the proximal canal. It is important to invert the ankle and protect the skin margin. The procedure is straight-forward and we have found that the image intensifier is not always necessary. As long as the threads go in easily, it will bend with the intramedullary canal, as demonstrated on postoperative x-rays (Fig. 1). If turning becomes difficult, the cortex may become perforated. Realigning the pin or using a shorter length pin is preferable, though experience has shown proximal pin penetration has not compromised results if a stable fixation is obtained. The pin is tightened until the head is adjacent to the tip of the fibula, but not so tight that it would become a lag screw. With bimalleolar fractures, performing fixation of the fibula first will make the medial malleolar reduction much easier.

It is important to do a stress view. If separation of the fibula from the tibia is present, the syndesmosis can be stabilized using a transverse malleolar AO screw adjacent to the previously inserted modified Knowles pin. There is usually a sufficient diameter of the fibula to fit the screw past the pin and stabilize the syndesmosis (Fig. 2). The posterior malleolus is also reduced through this lateral incision when indicated. The skin is approximated, followed by application of Adaptic and soft roll. A sugar tong ankle splint is then applied. Immediate ankle plantar flexion and dorsiflexion are encouraged as well as touch weightbearing with crutches. At three weeks, the patient is changed to a thin, light short leg cast with the forefoot free to allow full activity, including full weightbearing. This cast can fit into a stretched tennis shoe. At six weeks, the patient is given an aircast to wear for approximately two weeks. We have followed these patients with three weekly x-rays to be sure that the fracture has maintained its alignment and that the syndesmosis has not widened.

We conducted a retrospective study on patients with the modified Knowles pin in the intramedullary canal of the fibula (Table 1). Knowing that Fairbanks, Alaska has a very mobile population, we contacted only the patients who were treated for displaced ankle fractures between 1982 and 1989, and who were in Fairbanks for the summer of 1990. Of these twenty patients, all were willing to participate in the study. Several factors were analyzed for uniformity in sampling: length of follow-up, age, sex, type of fracture, postoperative splint types and durations, times to partial and full weightbearing, radiographs and range of motion. Patients were called individually for an office visit and completed a questionnaire modified from that used by W.A. Phillips, et al.[13] Items included the level of pain, limitations of activity, gait, stair climbing and descending ability, walking aids, reinjury, medication usage, progress, present performance and satisfaction. Dorsiflexion and plantar flexion measurements were taken according to methods used by Segal.[17] A range of motion rating was assigned to each patient.

Anterior-posterior, mortise, and lateral x-rays were taken. The mortise view was used to determine medial clear space and talar tilt. The medial clear space is the distance from the lateral border of the medial malleolus to the medial border of the talus at the level of the talar dome. A distance greater than 4 mm is considered abnormal.[5] Talar tilt is determined by examining the width of the joint space between the subchondral bone of the tibia and the talus in the medial and lateral parts of the joint on an anterior-posterior radiograph. A difference of 1-2

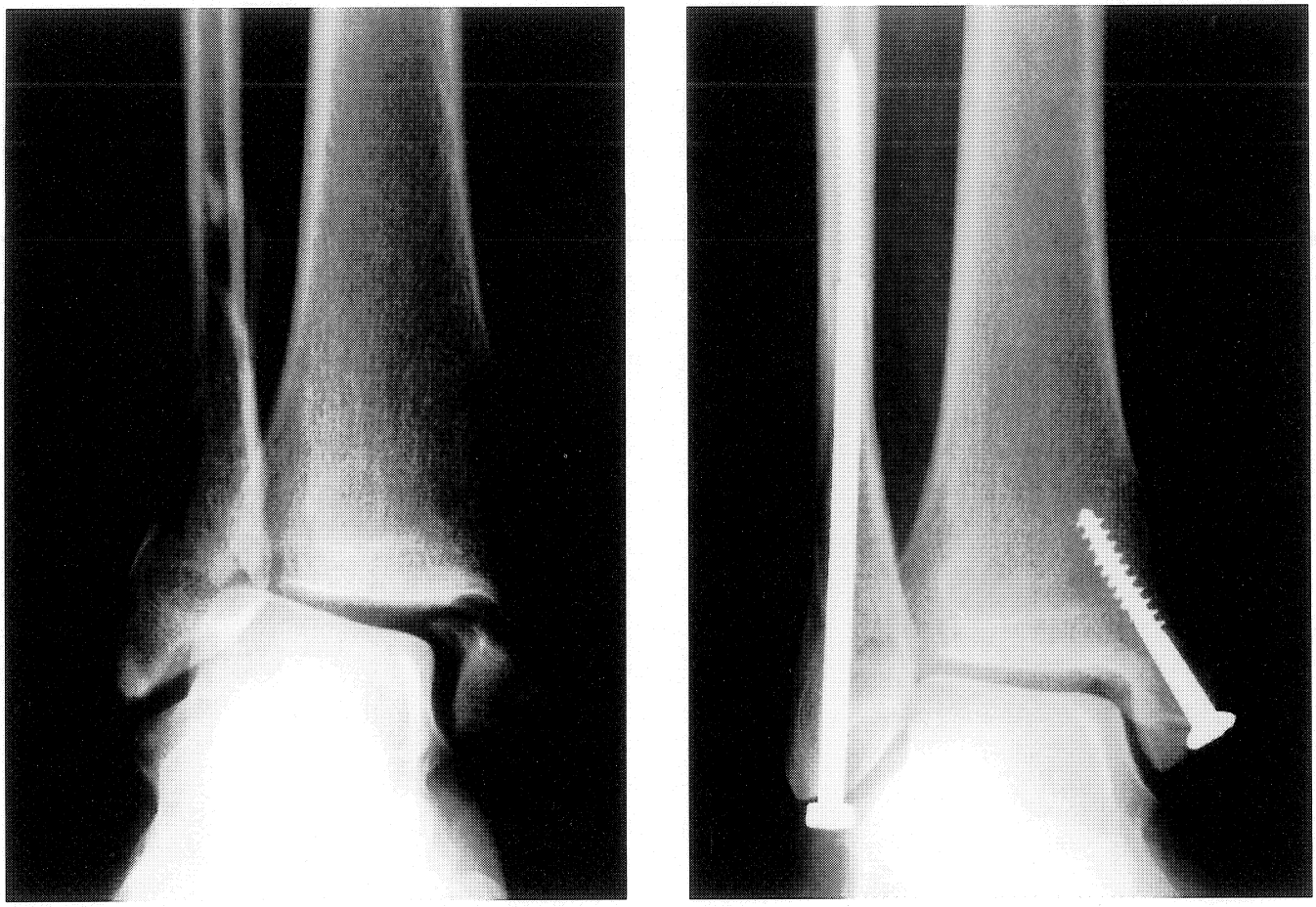

Figure 1 (A, B)
Bimalleolar fracture with the fibula internally fixed using a modified Knowles pin. Note the slight bend in the pin proximally. Stabilization is accomplished without pre-drilling the medullary canal.

mm is considered normal.[5] The anterior-posterior view was also used to measure the ankle syndesmosis and talar subluxation. Syndesmosis A is the measurement of the tibiofibular clear space from the lateral border of the posterior tibial malleolus to the medial border of the fibula. The distance is normally less than 5 mm.[12] Syndesmosis B is the measurement of the overlap from the medial border of the fibula to the lateral border of the anterior tibial prominence. The normal measurement is 10 mm or greater.[12] The talar subluxation was determined by assessment of the congruity of the tibial articular surface and talar dome. Any incongruity is considered abnormal.[5] The lateral views are used to measure lack of apposition or angulation of the fibula.

RESULTS

Follow-up ranged from one to eight years, with an average of four years. The study included seven males ranging in age from 33-73 years, with an average age of 49. The thirteen females ranged in age from 30 to 74 years, with the average age of 51. The Danis-Weber classification of ankle fractures was used. Three patients (15%) were Type A with transverse or avulsion fractures of the distal fibula distal to the syndesmosis, fifteen (75%) were Type B with oblique fractures distal to the syndesmosis, and two (10%) were Type C with fibular fractures proximal to the syndesmosis and syndesmosis rupture. In seven of the twenty patients, the talus was completely dislocated from the ankle mortise. Four of the twenty had a subluxed talus within the mortise, and in nine patients the initial x-ray did not demonstrate subluxation of the talus, though the fibula was displaced. In four of the twenty fractures, the fibula was the only bone fractured, six were bimalleolar fractures and ten were trimalleolar fractures. Injuries occurred from falls in fifteen patients (slipping on ice, tripping, or falling down stairs), two were involved in motor vehicle accidents, and three injuries occurred in sports-related activities. All patients had modified Knowles pins fixation of the fibula. Ten patients had the 6″ pin, eight had the 5″ pin, and one each a 4″ and a 3″ pin. In addition seven patients had internal fixation of the medial malleolus using one, two or three 4.0 mm AO

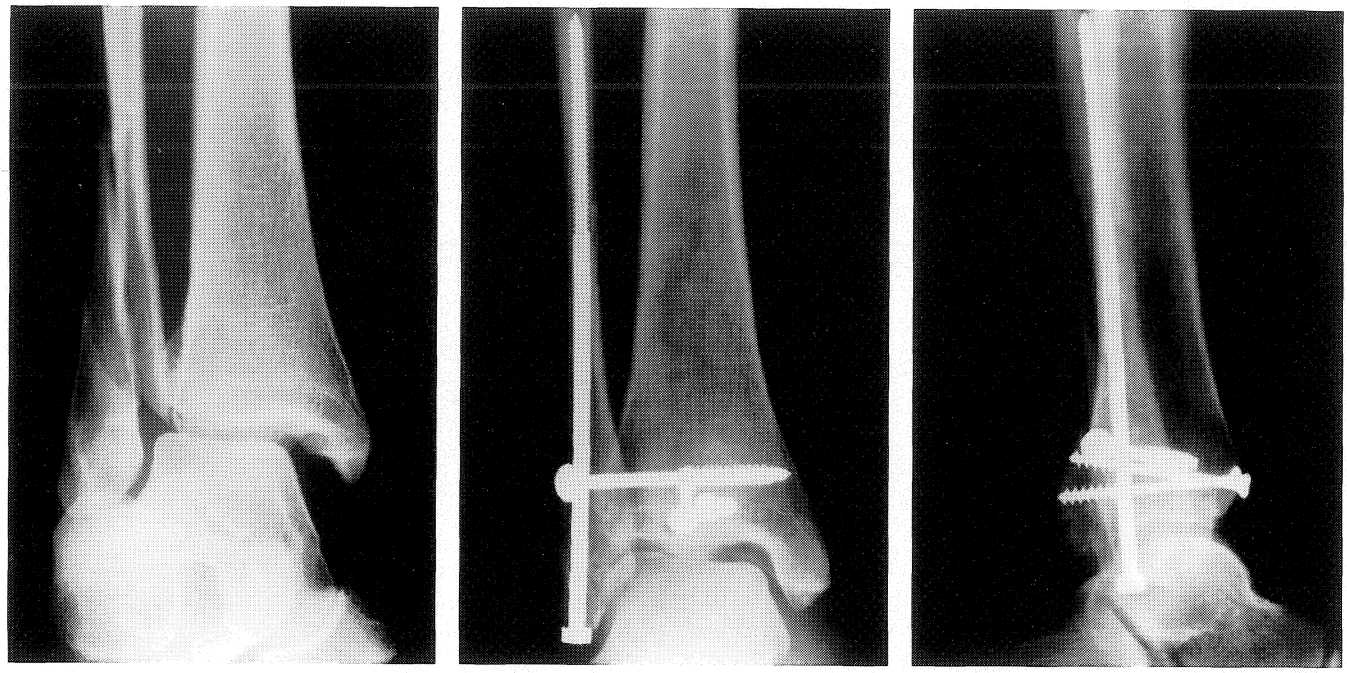

Figure 2 (A,B, C)
Pre and postoperative roentgenograms of an ankle fracture with an unstable syndesmosis and a posterior tibial fracture fragment. Internal fixation of the fibula with the modified Knowles pin still allows room for a transverse syndesmosis screw.

lag screws. One patient had a posterior malleolar fracture large enough to require fixation with a single AO screw. The average hospital stay was 3.5 days. The average total casting time was six weeks. The mean time until initial weightbearing was two weeks (range from 1 to 3-1/2 weeks), and the mean time until full weightbearing was six weeks (range from 4 to 12 weeks). Nine patients did not need physical therapy and were full weightbearing at six weeks. Nine patients did therapy on their own, including swimming, riding an exercise bicycle, and circular dorsiflexion and plantar flexion motions of the ankle. Two patients required a formal physical therapy program three times a week for approximately three months. All patients walked with a normal gait and were able to climb and descend stairs. None used walking aids.

The questionnaire revealed the patient's perception of his/her pain, functional performance, and satisfaction. Three of the twenty patients reported having transient pain after heavy activity, one experienced a dull ache occasionally after heavy activity, and one experienced occasional stiffness after a long day of activity. Eight (40%) patients had their pins taken out due to discomfort over the lateral pin head site after two to sixteen months, with an average of nine months. Decreased discomfort and increased range of motion commonly occurred after pin removal. Of the eleven patients with pins remaining internal, three complained of similar discomfort and plan to have the pin removed. The remaining eight patients have had no trouble with their pins and plan to leave them in indefinitely. One (5%) patient who was painfree had the pin removed. Fourteen (70%) patients felt their progress had plateaued and six (30%) reported continued improvement with time, even as late as five years post injury. None have become worse with time. Sixteen of the twenty reported their performance level had returned to 90-100% of their pre-injury state and were very satisfied. Three patients moderately satisfied with their treatment complained of loss of flexion. One patient was dissatisfied at one year post surgery, and felt the ankle should be less swollen. However, she thought her performance had returned to 90%, and the ankle was still improving. One patient used occasional aspirin for pain control.

A range of motion rating was assigned to each patient. Seventeen rated excellent (no loss of motion), and three had good range of motion (no more than five degrees loss of dorsiflexion). There were no patients with fair or poor ankle motion.

Radiograph ratings were determined post-operatively and on follow-up radiographs. Eighteen (90%) patients had excellent ratings post-operatively, and remained excellent at final follow-up. Two (10%) rated good post-operatively and on follow-up radiographs, due to fibular angulation in lateral projection only. Migration of the modified Knowles pin was also studied radiographically. No patient showed evidence of pin migration at final follow-up or in x-rays taken at or close to the time of pin removal. There were no infections. There were no nonunions or delayed unions,

and no change of position of the fracture once reduced. There were no refractures and no cases of progressive joint space narrowing.

COMPLICATIONS

Eleven of twenty patients (55%) felt discomfort at the fibula or tip of the pin. Pin complaints were mild in all cases, such that three patients with pain have not had the pin removed. However, patients with residual ankle symptoms, and particularly with pain at the tip of the fibula reported pin removal as worthwhile. Pin removal was accomplished without complication in nine patients.

One patient had slight lipping of the anterior tibia, and one had slight lipping of the medial malleolus. Five patients had ossification in the deltoid ligament attachment site, which was classified as injury and repair, not as a sign of arthritis. No patient had evidence of joint space narrowing. One patient underwent a second surgical procedure at day three to attach a posterior malleolar fracture fragment that was large enough to allow slight posterior subluxation of the talus. Initially it was felt to be small enough to be treated without internal fixation.

Post-traumatic arthritis may take years to develop, and cannot be accurately assessed by this short-term review. Anatomic reduction of the talus within the ankle mortise, as has been demonstrated by this review, should minimize the chances of ankle arthritis.

DISCUSSION

Modified Knowles pin internal fixation of the distal fibula is nearly ideal, but like AO plating and perhaps intramedullary rodding of other long bones, any exposed metal at the bone entrance site is at least detectable by approximately half the patients. If the head of the pin could be completely buried at the fibula, perhaps the device would be painfree and not need to be removed. Further modification of the modified Knowles pin might allow this.

It has not been determined how early these patients can do vigorous exercise after surgery. Instruction for early mobilization within the splints obviated the need for formal physical therapy in all but two patients. The device feels very secure even in quite osteopenic patients and may permit very early full weightbearing. Osteopenia in older patients, higher degrees of fracture comminution, the distance from the tip of the fibula to the fracture site, and complex fracture patterns have not compromised the results. The method of fibular stabilization continues to be used routinely.

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THE MORPHOLOGY OF THE LONG BONE PHYSIS IN HYPOCHONDROPLASIA AND COMPARISON WITH OTHER GROWTH DISORDERS

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ABSTRACT

The morphology of fibular physes from three growth disorders (achondroplasia, hypochondroplasia, and pseudoachondroplasia) were compared with normal physes. All growth disorders' physes were abnormal with the most consistent abnormality occurring in the hypertrophic zone (HZ). Marked shortening of the HZ was seen in the hypochondroplast and the achondroplast. The shortening of the HZ was more severe in the achondroplast compared to the hypochondroplast. Good column formation was present in most of the physis in both of these disorders. Disorganization of the entire physis was present in pseudoachondroplasia.

These observations are consistent with the hypothesis that physeal elongation is driven by HZ cell enlargement directed longitudinally by the extra cellular matrix. These observations suggest that abnormal HZ function may be the final common pathway of growth disorders.

Hypochondroplastic dwarfism was clearly delineated from achondroplasia in the late 1960's,[1,43] although descriptions of the condition were published in 1913 by Ravenna[31] and in 1924 by Leri and Linoussier.[21] Hypochondroplasia results in disproportionate short limbed dwarfing without rhizomelia.[1,35,38,43,45] Trunk height is normal or near normal and "stocky" build is typical. The head and facies are normal without the frontal bossing or the depressed nasal bridge seen in achondroplasia. A moderately increased lumbar lordosis is usually present. The hands and feet are short and broad without the trident hand seen in achondroplasia. Bowing of the legs is common (Fig. 1).

Radiographic features of hypochondroplasia include short, relatively broad long bones with metaphyseal flaring.[1,35,38,43,45] The ulnar styloid process is frequently long as are the fibulae. Young hypochondroplasts may have a mild "V" shaped physis although this is less pronounced than in achondroplasts. The pelvis is small but of fairly normal shape with normal flaring of the iliac crests. Narrowing of lumbar interpedicular distance is present in only 15% of hypochondroplasts and is milder than that seen in achondroplasts.[45] A key radiographic feature of hypochondroplasia is the relatively normal skull. The markedly shortened base of the skull, small foramen magnum, enlarged neurocranium and frontal prominence seen in achondroplasia are absent (Fig. 2).

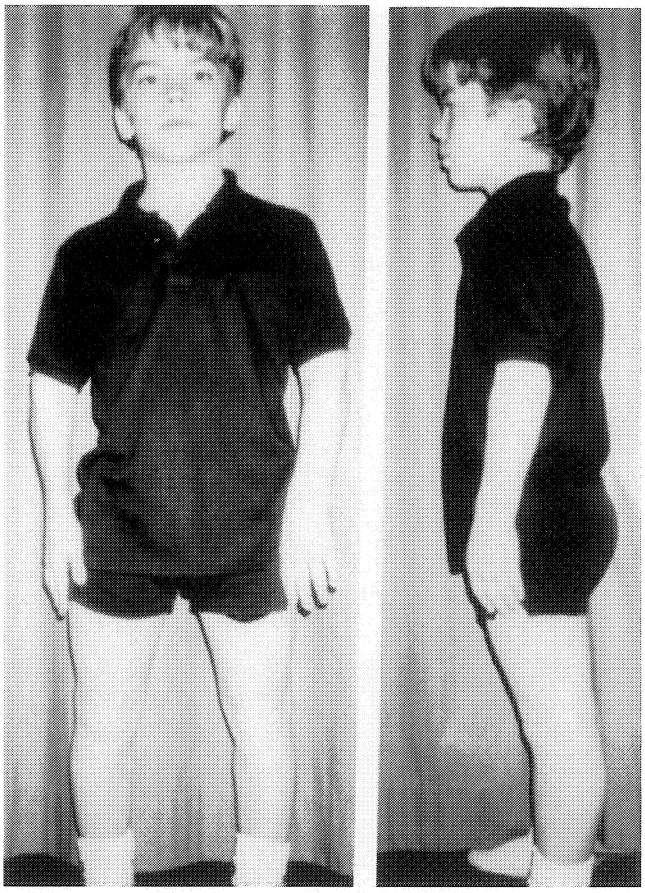

Figure 1
The patient at 14 years of age—typical features of hypochondroplasia are present including disproportionate short limbed dwarfing and normal head and facies. Mild residual varus of legs persists 3 years after fibular epiphysiodesis.

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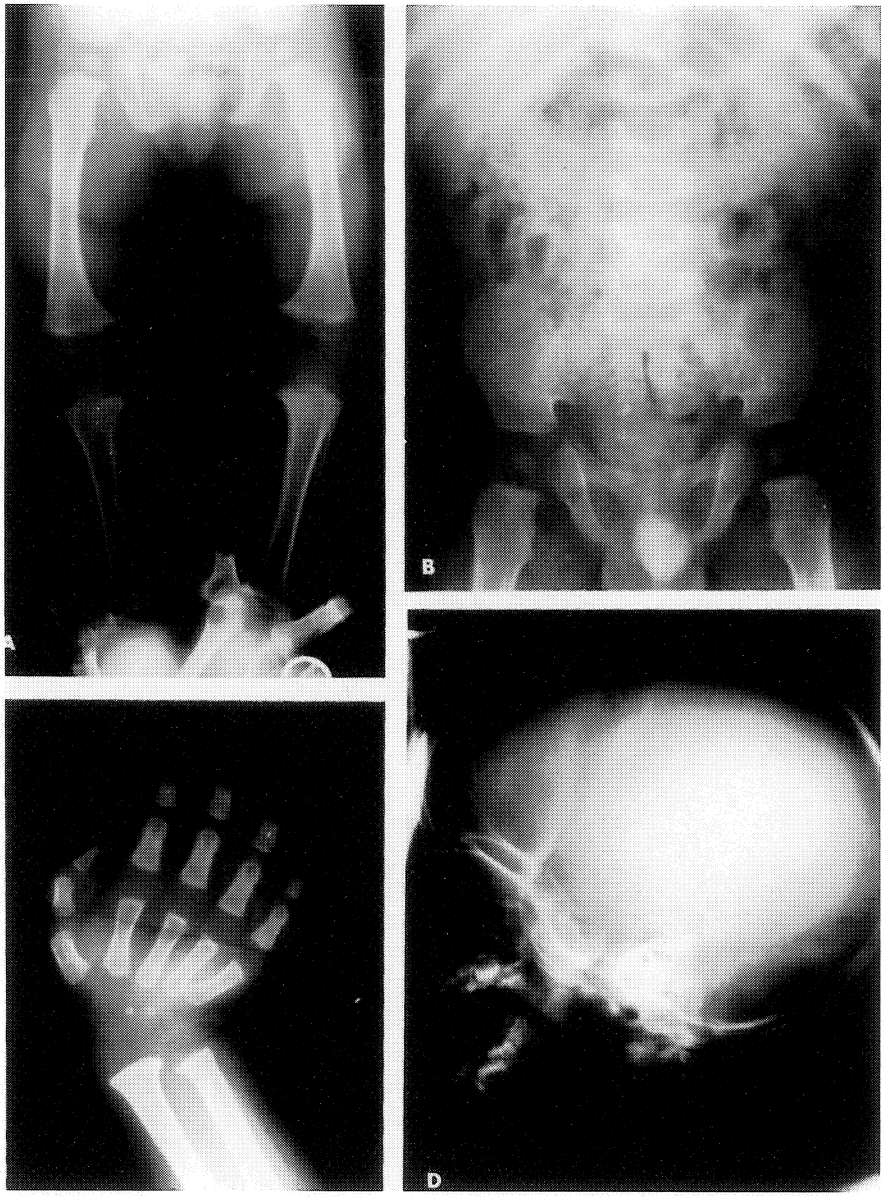

Figure 2
A) Short, broad long bones with metaphyseal flaring are apparent at 2 years of age. B) The normal pelvis with normal flaring of iliac wings is shown at 1 year of age. Narrowing of the lumbar interpedicular distance is absent. C) Short, broad tubular bones of the hand are seen at 1 year of age. D) The relatively normal skull as compared with achondroplasia is shown, also at 1 year of age.

Hypochondroplasia is estimated to be 1/12 as common as achondroplasia and is inherited as an autosomal dominant trait with most cases being new mutations.[38]

The distinction of hypochondroplasia as separate from achondroplasia is clearly illustrated by matings between achondroplasts and hypochondroplasts.[20,43] Their progeny are phenotypically either typical achondroplasts or hypochondroplasts rather than an intermediate appearance. The pathophysiology of hypochondroplasia is unknown. The only report of physeal morphology was by David Rimoin in 1976.[34] Rimoin studied iliac crest biopsies from seven patients and felt the morphology was entirely normal. Normal physeal morphology in this disorder is suspect for three reasons. First, no other described dwarfing condition has entirely normal long bone physeal morphology. Second, iliac crest apophyses are morphologically quite variable in normals and the radiographic appearance of the ilium in hypochondroplasia is normal. Finally, normal or near normal morphology of the iliac apophysis has been reported in achondroplasts, who have distinctly abnormal long bone physes.[23,28,32]

We are reporting the microscopic and ultrastructural morphology of the fibular physis in an 11 1/2 year old boy with hypochondroplasia and comparing this specimen with fibular physeal specimens from a normal child, an achondroplastic dwarf and a pseudoachondroplastic dwarf.

CASE REPORT

An 11 1/2 year old boy presented at the Orthopaedic Clinic for evaluation of progressive bow leggedness. He

was diagnosed with hypochondroplasia based on typical clinical and roentgenographic findings (Figs. 1 & 2).

His standing height was 115 cm. His weight was 26.5 kg. According to the growth chart for boys his standing height was well below the 5th percentile. His upper to lower segment ratio was 1.75. He had full range of motion of all joints and no complaints of pain. He had a 13° varus deformity of the right tibia and a 16° varus deformity of the left tibia.

Radiographs revealed short broad long bones with apparent metaphyseal flaring. Lumbar interpedicular narrowing was not present. The pelvis and skull were radiographically normal.

Proximal and distal fibular epiphysiodeses were indicated for correction of the varus deformities of the tibiae.

MATERIAL AND METHODS

Proximal and distal fibular physes were obtained with adjacent epiphyseal and metaphyseal bone and prepared for light and electron microscopy. Light specimens were fixed in 10% neutral buffered formalin, decalcified in 5% formic acid, dehydrated in alcohol, embedded in paraffin, sectioned and stained with 1) hematoxylin and eosin, 2) alcian blue and 3) safranin-0. Montages of the entire width of the excised portion of the growth plate were compared using magnified photographs. The montages were examined qualitatively to assess typical areas and variability.

Specimens for electron microscopy were fixed in 2.5% glutaraldehyde in 0.05 M cacodylate buffer (ph 7.4) at 4° C and post fixed in 1% osmium tetroxide in 0.05 M sodium cacodylate with 1.5% potassium ferrocyanide. They were then dehydrated in ethanol and acetone, and infiltrated with and embedded in Spurr embedding medium. Thin sections were then cut, stained with uranyl acetate and lead citrate and examined on an Hitachi H-7000 electron microscope at 75 kv.

RESULTS

Morphologic Observations of the Physis in Hypochondroplasia

The physis was abnormal in both the proliferative zone (PZ) and the HZ (Fig. 3). The morphology of the resting zone (RZ) and the zone of provisional calcification appeared normal. The PZ showed mild to moderate disruption of the normal columnar arrangement in the majority of the physis. However, the height of the PZ appeared normal. The most striking finding was narrowing of the HZ. Although some variability existed, the majority of the HZ was only 2-3 cells thick. The size of individual HZ cells appeared normal. Electron microscopic assessment of the chondrocytes appeared normal in each zone. Cell degeneration, abnormal matrix, and intracellular inclusions were not found as in some other dwarfing conditions.

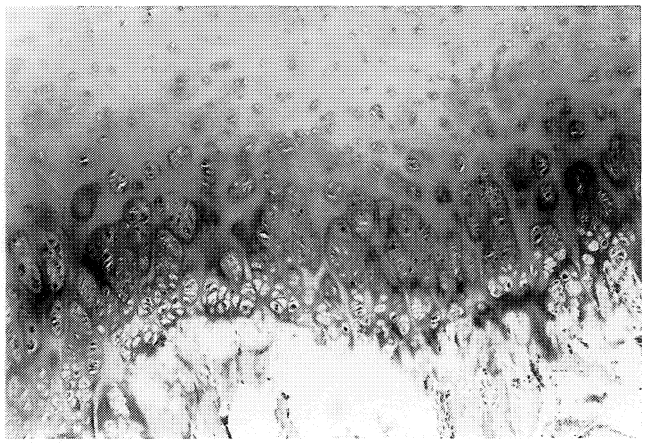

Figure 3
This photomicrograph is a 100X magnification of the fibular physis. Note the moderate disruption of PZ columns and the narrowed HZ consisting of only a few cells.

Comparative Physeal Morphology of Hypochondroplasia, Achondroplasia and Pseudoachondroplasia

The physeal morphology of the proximal fibula from this 11 1/2 year old boy with hypochondroplasia was compared with proximal fibular physes from a 10 year 5 month old girl with achondroplasia, a 14 year old boy with pseudoachondroplasia, and children with normal physes aged 12 year-11 months, 12 years and 14 years (Fig. 4). One of the normal specimens was obtained at autopsy after trauma, one at epiphysiodesis for contralateral hemiatrophy and the other at epiphysiodesis for leg length discrepancy due to a mild contralateral congenital spastic hemiparesis.

Resting zone abnormalities were present only in pseudoachondroplasia. The abnormalities were fewer cells, cell enlargement due to the rough endoplasmic reticulum (RER) inclusions, and some cell degeneration.

Proliferative zone abnormalities were present in all dwarfing disorders. Pseudoachondroplasia had complete disorganization of the normal column arrangement. The majority of the achondroplasia physis had a normal appearing PZ with mild columnar disorganization. Areas of complete disorganization previously referred to as "cluster areas" were uncommon.[23] The hypochondroplasia PZ had more disorganization than did the achondroplasia PZ but columns were still well maintained in comparison with pseudoachondroplasia.

Hypertrophic zone abnormalities were striking in all conditions. Narrowing of the HZ was present in hypochondroplasia and achondroplasia. The hypochondroplasia HZ showed 2-3 cell thickness with normal enlargement of cells. The achondroplasia HZ had only 1-2 cells that were clearly in the HZ and these cells showed less enlargement than in hypochondroplasia or the normal specimens. The

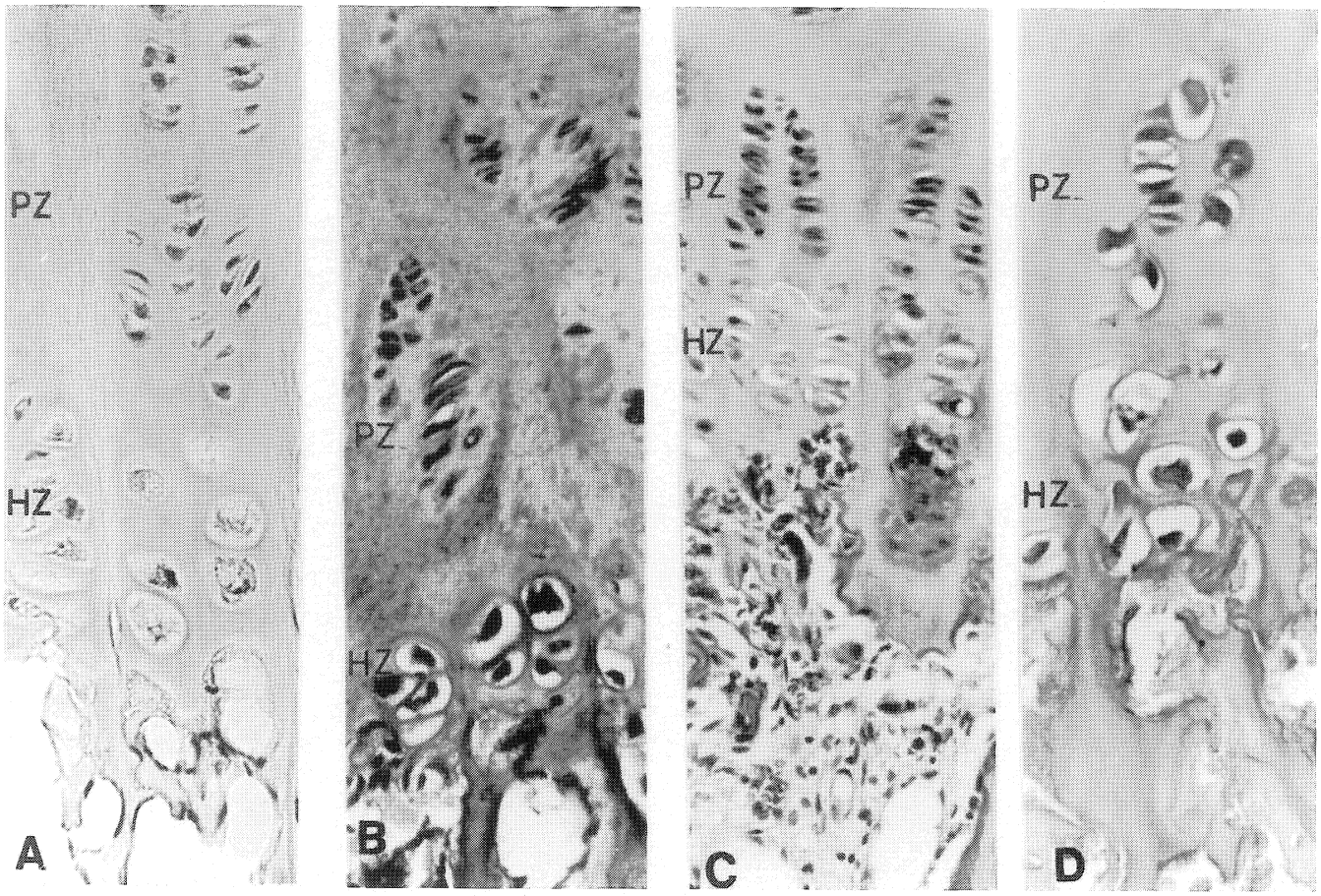

Figure 4
These photomicrographs are all fibular physes magnified 250X. a. is a normal physis from a 12 year 10 month old female with mild contralateral hemiparesis, b. is from a 11 year 6 month old male with hypochondroplasia, c. is from a 10 year 5 month old female with achondroplasia, d. is from a 14 year old male with pseudoachondroplasia. Compared with the normal (a) the HZ is narrowed in the hypochondroplast (b)and the achondroplast (c). The lowest cells in the achondroplast's physis never attain the normal polygonal shape of HZ cells. The enlargement of the HZ cells of the hypochondroplast is near normal but with fewer HZ cells than normal. The pseudoachondroplast (d) has good cell enlargement in the HZ but marked disorganization. The columnar organization is better preserved in the achondroplast than the hypochondroplast.

pseudoachondroplasia HZ had complete disorganization which made it difficult to identify a true HZ in some areas. Enlargement of the HZ cells that were present was as great or greater than normals due to the large RER inclusions.

Discussion

Qualitative assessment of the long bone physis in a hypochondroplast dwarf reveals definite abnormalities. Mild disruption of the normal columnar orientation is seen in both the proliferative and hypertrophic zones although columns are well maintained compared with pseudoachondroplasia. The hypochondroplast's HZ had fewer hypertrophic cells per column compared to normal but was not as diminished as the achondroplast's HZ.

Interpretation of these observations depends on one's view of the mechanism by which the normal physis elongates. Cell proliferation, matrix synthesis and chondrocyte hypertrophy have all been thought to contribute to physeal elongation.[3,14,16,17,18,36] A recent hypothesis suggests that the key mechanism of physeal elongation may be matrix directed swelling of HZ cells.[8] This hypothesis is consistent with the major morphologic abnormalities we observed in dwarfing conditions.

Evidence for matrix directed HZ cell swelling as the major determinant of physeal elongation comes from work with animal physes.[4,5,7,11,14,16,17,19,44] This work has demonstrated that relatively few PZ chondrocytes continue to proliferate.[14,19] PZ cells are more appropriately described as flattened chondrocytes.[14,19] The volume of these cells increased sixfold from the upper PZ to the lower HZ.[4,7] Approximately 90% of this volume increase is swelling (imbition of water) not hypertrophy (synthesis of new macromolecules and organelles)[5,7] and this swelling is directed longitudinally by the collagenous vertical matrix columns.[39,42] The flattened chondrocytes of the PZ are

four times wider than tall whereas these cells become polygonal with an equal height to width by the lower HZ, which reflects this longitudinal swelling.[4] The great majority of the cell volume increase occurs in the lower 20% of the physis.[44] These facts suggest that HZ cell swelling may be the major contributor to physeal elongation. Furthermore, both increased rate of growth and slowing of growth are strongly correlated with HZ cell volume and height[6,16,44] and the amount of HZ swelling seems largely independent of the rate of cell proliferation and cell death.[11,44] Matrix directed cell swelling is a much simpler and more energy efficient mechanism to obtain physeal elongation than cell replication and/or hypertrophy and is similar to the established mechanism of rapid growth in plants.[42]

Evidence that HZ events may be key in dwarfing conditions is supported by a recent morphometric study of osteochondrodysplasia in Scottish deer hounds.[2] Breur et al. found a dwarfing condition in these puppies characterized by RER lamellar inclusions with the same lamellar periodicity as found in human pseudoachondroplasia. Comparison between affected and normal littermates revealed no difference in the RZ. Proliferative zone cells were equal in number and only slightly smaller in the affected puppies. Hypertrophic zone cell volumes, however, were one-half as large in affected puppies and cell shape was radically different. Affected dogs' HZ cells were much more spherical, hence the enlargement that did occur was not directed longitudinally. Polarized light microscopy showed disorganization of the normally vertically oriented collagen fibers.

The architecture of the fibular physes in achondroplasia, hypochondroplasia and pseudoachondroplasia supports this hypothesis: Morphologically, the RZ is not abnormal in achondroplasia and hypochondroplasia. The PZ is abnormal in all the dwarfing conditions with mild column disorganization in achondroplasia and hypochondroplasia and complete disorganization in pseudoachondroplasia. The HZ is the most abnormal and seems to correlate best with the severity of dwarfing. Achondroplasia and hypochondroplasia both have shortened HZ with mild disruption of longitudinal orientation. The HZ in the achondroplast is shorter than the HZ in the more mildly stunted hypochondroplast and less enlargement of HZ cells occurs in achondroplasia than hypochondroplasia. The pseudoachondroplast has more enlarged cells in the HZ than either of the other dwarfing conditions but has complete loss of longitudinal matrix direction.

It seems plausible that a final common pathway of dwarfing conditions exists, regardless of the underlying biochemical etiology. This pathway may be a disruption of normal HZ events that consist of matrix directed cell swelling. Cell swelling and/or matrix direction may be abnormal in varying proportion in different disorders. The abnormalities in growth disorders are clearly not limited to the HZ as morphologic abnormalities of the PZ are commonly seen. Nevertheless the extent of failure of longitudinal growth may best correlate with the extent of disruption of normal HZ events. The RZ and PZ may participate primarily by failing to create the necessary conditions for matrix directed HZ cell swelling.

The dwarfing conditions we studied had two basic patterns of HZ abnormality. Hypochondroplasia and achondroplasia showed a narrowed HZ, while pseudoachondroplasia had HZ disorganization. This prompted us to review the reported physeal morphology in other growth disorders. All reported disorders have abnormalities in the HZ (List 1). Growth disorders of known or probable etiology producing short columns or diminished HZ cell enlargement include hypopituitarism,[40] pseudohypoparathyroidism,[40] Hurler syndrome,[40] spondylometaphyseal dysplasia,[41] and asphyxiating thoracic dysplasia.[37] All are caused by an endocrine abnormality or metabolic defect in the chondrocyte. Growth disorders of known or probable etiology with a disorganized physis include pseudoachondroplasia,[27,41] Kneist syndrome,[9,29, 30,41] metaphyseal chondrodysplasia,[24] achondrogenesis I and II,[9,12,13,15] opismodysplasia[41] and fibrochondrogenesis[41] and some variants of spondyloepiphyseal dyplasia.[9,25,26] All of these disorders are associated with a defect in proteoglycan or type II collagen synthesis. It seems possible that a failure of production of sufficient cells or a failure of HZ cell enlargement is the cause of growth retardation in the endocrine/metabolic disorders and is reflected morphologically by shortened columns and especially a narrowed HZ. Growth retardation in disorders with abnormal matrix production may result ultimately from a failure to direct HZ cell swelling longitudinally and is reflected morphologically in disorganized physes.

Growth disorders of unknown etiology such as achondroplasia, hypochondroplasia and multiple epiphyseal dysplasia have mainly short columns with mild disorganization of columns.[22,24,32,34,37,40] If this speculation is correct, the cause of these disorders may be an endocrine or metabolic defect primarily affecting the physeal chondrocytes' intrinsic cell function rather than abnormal matrix synthesis. Conversely, many types of spondyloepiphyseal dysplasia, thanatophoric dysplasia, chondrodysplasia punctata, pykondysostosis, and short rib polydactyly syndrome, which have disorganized physes, may be more likely to have a matrix synthesis defect.[22,33,34,37,41]

The boundary between short and disorganized columns is not always distinct. One would expect that cells with an underlying metabolic disorder would not produce normal quantities of matrix. Thus most dwarfing conditions with short columns have some disorganization of columns when

compared to normal physes. Conversely, cells with disordered matrix production are probably not healthy in all other respects and some diminution of size or numbers of cells may be expected. Nonetheless the differences between short and disorganized physes are relatively clear in many disorders. The classification set forth in this paper gives a new framework for considering the etiology of growth disorders that may be correct in many cases and may help direct research efforts into the most productive areas of investigation. Finally, these observations lead support to the hypothesis that matrix directed cell swelling is the normal mechanism of physeal elongation.

LIST 1

Short Columns

Pituitary Dwarf[40]
Pseudohypoparathyroidism[40]
Hurler Syndrome[35,40]
Spondylometaphyseal Dysplasia (Kowolski)[37,41]
Asphyxiating Thoracic Dysplasia[37]
Spondyloepiphyseal Dysplasia (some variants)[37]
Multiple Epiphyseal Dysplasia[40]
Long Limbed Compomelic Dysplasia[37]
Diastrophic Dysplasia[34,37,41]
Achondroplasia[22,24,32,34,37,40]
Hypochondroplasia[This Report]

Disorganized Columns

Pseudoachondroplasia[10,27,41]
Metaphyseal Chondrodysplasia[24,34,37]
Kneist Dysplasia[37,41]
Achondrogenesis Types I & II[13,15,22,33,37]
Opismodysplasia[41]
Fibrochondrogenesis[41]
Spondyloepiphyseal Dysplasia (some variants)[34,41]
Chondrodysplasia Punctate[34,37]
Homozygous Achondroplasia[33,37]
Thanatophoric Dysplasia[22,33,37]
Pyknodysostosis[41]
Short Rib Polydactyly Syndrome[37]
Chondroectodermal Dysplasia[22,37]

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MANAGEMENT OF BONE DEFECTS
A Review of Available Techniques

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INTRODUCTION

Trauma, neoplasm, congenital defects, infection and failed arthroplasty are all capable of creating large bony defects. Achieving the goals of osseous reconstruction of these defects, namely, sustaining bone length and securing bony union, remains a challenge to the orthopaedic surgeon.

Primary limb shortening, which is generally well tolerated in the upper limb without significant effects on cosmesis or function, and various amputations of both the upper and lower extremity are among many techniques in the surgeon's armamentarium. The goal of this article, however, is to review reconstructive procedures available once the surgeon has chosen to salvage the limb and maintain skeletal length.

CLASSIFICATION

Various techniques are individually categorized in Table 1. The entire reconstructive effort, however, may employ combinations of several methods, e.g., prosthesis + allograft ("alloprosthesis"), bone transport + autograft, etc. This discussion will focus on cancellous autogenous grafting, structural autogenous grafting (particularly with reference to various uses of the fibula) and distraction osteogenesis. In addition, various uses of allograft, xenograft, biomaterials and prosthetic implants will be reviewed.

TABLE 1
TECHNIQUES FOR RECONSTRUCTION OF BONE DEFECTS

I. Grafting
  A. Autograft
    1. Cancellous
      a. open
      b. closed
    2. Structural
      a. fibular transfer
      b. non-vascularized bone
      c. vascularized bone
    3. Autoclaved
  B. Allograft
  C. Xenograft
II. Distraction Osteogenesis
  A. Bone transport
    1. Monofocal
    2. Bifocal
    3. Fibular transfer
  B. Compression-distraction
III. Biomaterials
  A. Demineralized bone matrix
  B. Ceramics
IV. Prosthetic Implants

GRAFTING TECHNIQUES

Cancellous Autografting

Early surgeons initially used cortical bone,[1] but after half a century of surgical practice there seemed to be near universal agreement that cancellous bone was superior. Nonvascularized cortical autograft, while it may provide immediate bony support, has the disadvantages of slow union, high incidence of fatigue fracture, and the probability that total revascularization and replacement may never occur. Conversely, cancellous graft has greater capacity to induce osteogenesis, revascularizes easier, and matures and remodels faster. Though stress hypertrophy occurs slowly, the new segment can be augmented by supplementary grafting.[30]

The fragments of cancellous bone should have one dimension less than five mm so that nutrients can diffuse to all the osteoblasts.[4] These fragments can be used in either an open[33] or closed technique. With the open technique, union is obtained first and the skin defect is allowed to epithelialize spontaneously or with the aid of a split thickness graft. With the closed method stable soft tissue coverage is obtained prior to bone grafting.

Open Cancellous Grafting (Papineau)

For both techniques the early steps of treatment are the same and include meticulous debridement of all necrotic bone and soft tissue and stabilization of the skeleton (e.g., external fixator, intramedullary nail, plate, tibiofibular synostosis). In the open technique, a bed of granulation tissue allowed to form. Cancellous graft is then packed into the skeletal defect without soft tissue coverage. The bed of granulation tissue revascularizes the bone graft and the defect is allowed to epithelialize spontaneously or a split thickness graft is applied. (Figure 1A-F).

Green and Dlabl[15] used this technique in fifteen patients

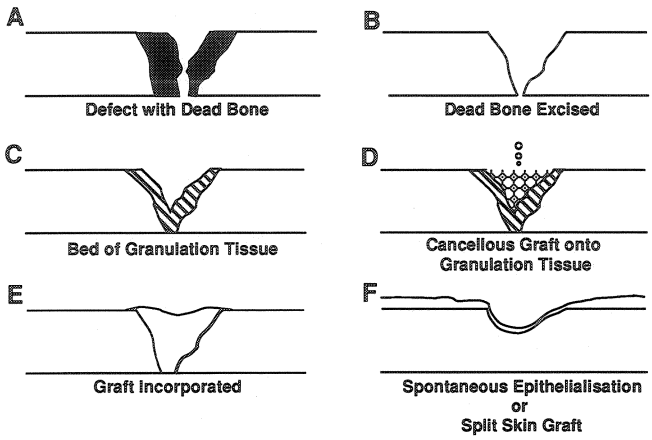

Figure 1
Open cancellous grafting

aged twenty-one to sixty-seven for septic nonunion of various long bones. Following adequate debridement, resultant bone defects ranged from 1.5 to eight cm. Union was established in thirteen of fifteen cases; two eventually required amputation. Important technical points stressed by the authors included: 1) thorough debridement, 2) adequate skeletal stabilization (biplaner external fixation was recommended), 3) waiting until all exposed bone is covered with granulation base prior to grafting (average time: four weeks in this series), 4) maintenance of the viability of the transplanted bone tissue by immersing it in a physiologic irrigating solution, and 5) inserting a bone graft tissue mass larger in diameter than the bone being replaced. The authors concluded that this technique is not suitable for defects greater than nine cm since a limited volume of cancellous bone is available for reconstruction.

Malkawi, et al,[28] reported on a similar series of thirteen patients managed with open cancellous grafting. Defects, following debridement, averaged 4.3 cm (range two to ten cm). Union and eradication of infection were achieved in all patients, though functional results were considered poor in four and fair in four due to shortening or poor ankle motion. The authors concluded this technique would achieve good results even in such unfavorable conditions as those encountered in infected segmental bone defects, providing a well vascularized bed and adequate stability are achieved prior to grafting.

Although the open technique is less demanding and is associated with less potential morbidity than other techniques, disadvantages do exist. In general, open grafting is poor for defects: 1) over four cm, 2) with poor surrounding muscle and, 3) with total loss of bone substance (diaphysectomy).[37] The procedure will not work if the local blood supply is inadequate to support formation of granulation tissue. Finally, the technique provides less durable skin coverage and intrinsic bone strength is slow to develop.[31]

Closed Cancellous Grafting

Following debridement, stable soft tissue coverage in the form of rotational, cross leg pedicle or free flaps is obtained prior to grafting. Free tissue transfer is best for management of large defects created following debridement after trauma or infection.[13,31] Free tissue, because of its potential bulk and unlimited mobility, allows for much more radical debridement of devitalized or infected tissue. Unlike local flaps, free tissue can cover massive defects, is not limited by the muscle's arc of rotation, and does not further compromise adjacent recipient tissue. Also, a transferred tissue flap with microvascular anastamoses is highly vascularized as opposed to local flaps, which are rendered relatively ischemic in their distal portion. In contrast to cross leg flaps, in which increased blood flow is only transitory, free flaps add permanent supply to the region.[39]

Regardless of the soft tissue coverage chosen, increased vascularity in the recipient bed aids in rapid incorporation of cancellous grafts. Successful healing has been demonstrated in several case series where diaphyseal defects averaging five cm,[28] 8.8 cm,[30] and ten cm[8] have been successfully reconstructed in multi-staged procedures which include obtaining stable soft tissue coverage followed by massive autogenous grafting. Drawbacks of this technique include the need for multiple operative procedures (usually three to five) and potential soft tissue and bony donor site morbidity. Recent reports seem to favor closed over open grafting, though the best results have a healing index* of only one cm per month.

$$\text{*healing index} = \frac{\text{length of defect (cm)}}{\text{treatment time (months)}}$$

STRUCTURAL AUTOGRAFTING

Various uses of the fibula have been employed in the management of segmental defects. These techniques, including fibular transfer, free nonvascularized fibular autograft, tibiofibular synostoses and free vascularized fibular autograft, provide useful models for discussing various uses of structural autograft. Fibular transfer was first described by Hahn in 1884 for management of congenital pseudoarthroses. Huntington then modified the technique to a two stage transfer in 1905 for the management of traumatic defects.[30] This modification was later employed by Carrell[6] for use in tumor surgery. (Figure 2). Fatigue fracture and nonunion, which may compromise further efforts at reconstruction, were frequent problems with these techniques. Also, these methods cannot be considered when the peroneal vascular pedicle is the sole supporting artery to the lower limb and foot.[31]

Tibio-fibular synostoses

Multiple techniques have evolved which attempt to bridge a tibial defect by creation of a proximal and distal

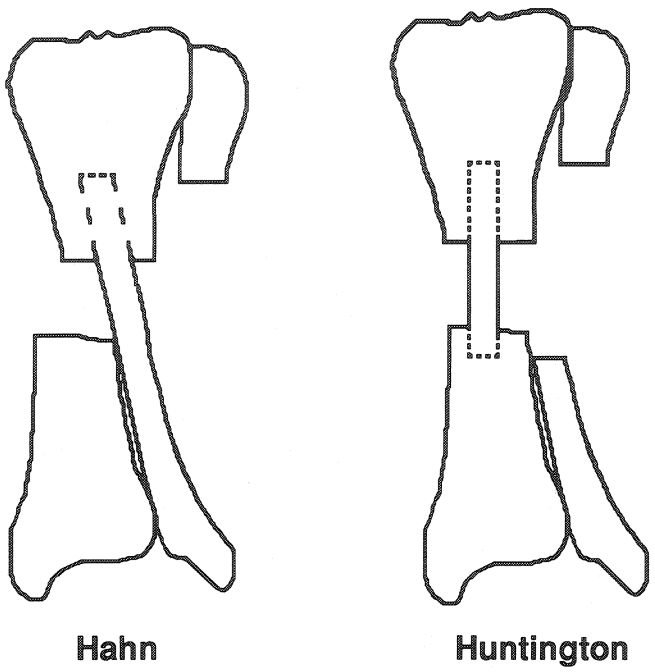

Figure 2
Hahn's fibular transfer and Huntington's two stage modification

tibio-fibular synostosis (Figure 3). Nonunion, unremitting infection and amputation were common sequelae following many of the early single stage procedures. Subsequent staged procedures have achieved much higher success rates. Basic principles, as outlined by Maurer and Dillon,[30] include: 1) control of infection and stable soft tissue coverage, 2) stabilization of the tibia by transfixing the fibula proximally and distally with screws, and, in large defects (> 12 cm), spanning the tibial gap with a plate, 3) increase structural strength of the construct by bridging the tibial defect with cancellous graft or reinforce the fibula spanning the defect with cancellous graft, and 4) prolonged, protected weightbearing in a brace.

Employing these principles, defects ranging five to fifteen cm were successfully bridged in patients who had severe soft tissue trauma, local sepsis, and were otherwise candidates for amputation. Five to seven procedures, separated roughly by three months, were required for each patient. No implant failures, fatigue fractures or chronic infection were noted at a mean follow-up of 5.5 years. The tibio-fibular synostosis, while avoiding areas of sepsis, functions as an internal splint and provides a stable environment for incorporation of cancellous graft in the tibial defect and/or around the bridging segment of fibula. This technique is suitable for defects greater than three cm, provided that a stable soft tissue cover is obtained first. In general, defects under three cm are more easily handled with a simpler posterolateral graft.[14]

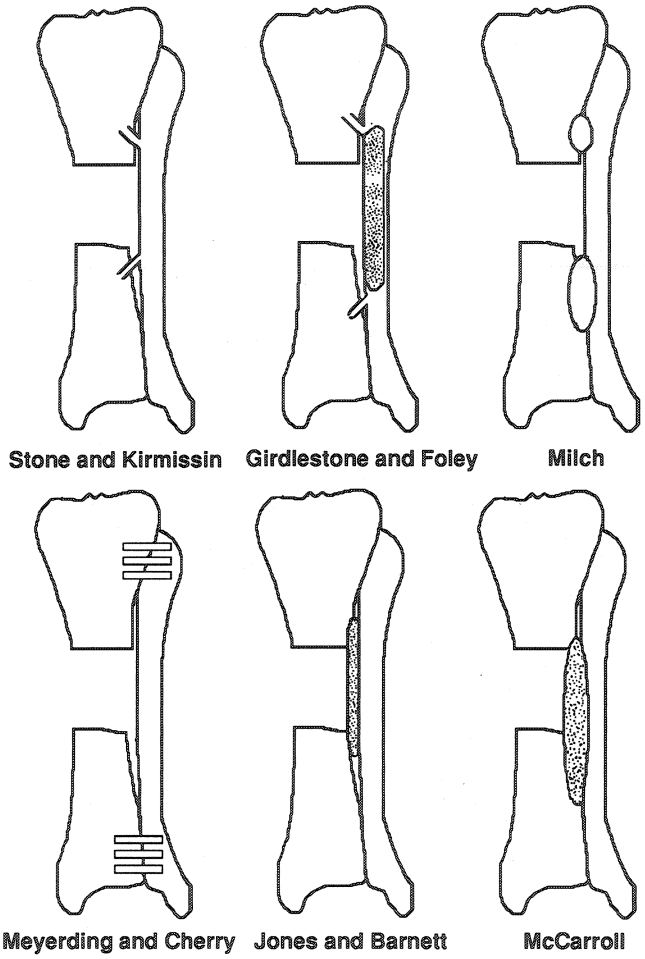

Figure 3
Various tibio-fibular synostoses. Stone and Kirmissin split the fibula and transferred the split portion onto holes in the tibia. Girdlestone and Foley created proximal and distal "flying buttress" grafts between the tibia and fibula and reinforced the fibular shaft with cancellous graft. Milch split the outer tibial cortex and laid it on the interosseous membrane. Meyerding and Cherry used bone pegs to create proximal and distal tibiofibular synostoses. Jones and Barnett recommended a posterolateral graft; McCarroll advocated an anterolateral graft.

Free non-vascularized graft

Free non-vascularized fibular graft, as well as free tibial and iliac graft, has been employed in both inlay and onlay fashion with a variety of fixation techniques to bridge bony defects.[25] While a whole fibular transplant is almost ideal for bridging defects of the radius or ulna, elsewhere single fibular transplants are limited by their size. The graft needs prolonged support and will continue to weaken as it revascularizes, reaching a maximum weakness in twelve to forty-eight weeks after transfer.[31] In addition, these grafts are too small and prone to fatigue fracture in the adult tibia; however, in children, fibular grafts may hypertrophy enough to approach the size and strength of a normal tibia.[9]

One exception to the application of free nonvascularized fibula in the adult lower extremity is its use in dual fashion.[41] Significant tibial and femoral gaps of up to twenty-four cm have been successfully bridged in adults following intercalary resection of a low grade tumor. Nonunions and fatigue fractures occurred occasionally and universally healed after local autogenous grafting. This technique appears to be simple, readily available, reliable, less time consuming and economical compared to some other techniques (no need for a bone bank, microvascular expertise, expensive implants, etc.)

Free vascularized graft

Free vascularized bone graft was first described by Taylor in 1975. Orthopaedic literature has largely dealt with application of free vascularized fibulae in the management of congenital pseudoarthrosis in the tibia. Generally, excellent results are reported.[35,40] Vascularized fibular pedicle grafts[13] and cross leg composite pedicle grafts[12] have also been described for management of this condition.

Free vascularized bone grafting has been successfully applied to segmental bone defects of up to fifteen cm secondary to infection, trauma or following tumor resection.[24,38,39] Donor sites employed most frequently are the fibula (most suitable for reconstitution of long bone defects), rib and iliac crest, the latter being part of an osteocutaneous transfer.

Such free grafts retain their intrinsic nutrient blood supply so that osteocytes in the graft can survive.

Healing of the graft to recipient bone will be facilitated without the usual replacement of the graft itself by creeping substitution. Advantages include quicker union at the graft-host junction, shorter immobilization, improved graft incorporation and graft hypertrophy. The cortical nature of the bone also allows for stable internal fixation and early progressive weight bearing.[31] In addition, vascularized grafts may be suitable for transfer into poorly vascularized beds.[30] Disadvantages include potential donor site morbidity, prolonged and complicated surgery, and need for microvascular expertise and equipment.

Autoclaved Autograft

This technique has been employed for limb salvage following wide local resection for low grade and certain high grade malignant lesions.[22] Following excision of the lesion, gross tumor is debrided and the remaining bone is autoclaved for five minutes to kill microscopic tumor deposits. The bone is then reimplanted with some form of internal fixation and/or prosthetic implant. Segmental defects ranging from ten to eighteen cm of the distal femur and ten to thirteen cm of the proximal tibia have been reconstructed with a long stem total knee prosthesis supplemented by autoclaved autogenous bone graft. Infection, delayed wound healing or tumor recurrence have not been problems. The autoclaved graft is easily obtained and always has a perfect fit. Though the technique is appealing, in vivo biopsies of the graft have revealed no incorporation of dead bone and, experimentally, autoclaved autograft heals slower than fresh autograft or fresh frozen allograft.

Allograft

The first large series of allograft transplantation was reported in 1908 by Lexor who performed whole and hemijoint transplants about the knee with limited success. It was not until the 1960's, when it was discovered that the immunogenicity of the graft could be reduced by freezing, that interest in allograft transplantation was rekindled.[29]

The use of allograft bone in the reconstruction of segmental defects may offer some distinct advantages. The supply of bone is relatively unlimited and available in a variety of sizes and shapes. Allografts do not require sacrifice of normal structures nor is there donor site morbidity. With cryopreservation techniques, articular surfaces can be retained in the anatomical reconstruction of the joint (osteoarticular allograft), or allograft can be used in conjunction with a prosthesis ("alloprosthesis"). Conversely, autograft bone is of limited supply, and the shape of the material, its relative lack of strength, and the absence of articulating surface often make it impossible to construct massive defects.[29]

Observations on massive retrieved human allograft[11] have demonstrated consistent union at the graft-host junction. Internal repair is slow and confined to the ends and superficial surface of the graft, and involves approximately 20% of the graft by five years. Deep unrepaired portions of the graft retain their architecture. When an implant was used, no evidence of resorption or loosening was noticed. Soft tissues of the host attached to the graft by deposition of a thin seam of new bone on the surface of the graft. In osteoarticular autografts, necrotic cartilage functioned well for as long as five years. This method is generally employed following tumor resection, though has been used for non-neoplastic conditions including failed total hip arthroplasty,[32] extensive fibrous dysplasia or Gaucher's disease.

Despite enthusiasm in many published reports, use of allograft bone has certain disadvantages. Revascularization is even slower than that of autogenous cortical bone. Fractures, infection, nonunion, and host versus graft reaction have remained problems. In addition, allograft bone must generally be avoided in patients with infection because any residual organisms can remain on the allograft, which acts as a large sequestrum.[31]

Xenograft

There is very little recent literature about this new rarely applied technique, though bovine graft at one time had numerous proponents. Later series showed bovine bone was poorly tolerated by the host and condemned its use in orthopaedic surgery.[18] More recently, freeze dried baboon bone combined with an autogenous component of cancellous bone has been used to reconstruct large diaphyseal defects in humans following trauma and tumor resection. The technique uses cross segments (rings) of implant material stacked on an intramedullary nail. At two years, the xenograft showed radiologic and histologic incorporation. Advantages, like allograft, include an unlimited supply of bone available in a variety of shapes and sizes.[23]

DISTRACTION OSTEOGENESIS

Ilizarov, expanding on traditional mechanical considerations of bone formation under compression, has demonstrated bone formation under tension loading.[3] This "distraction osteogenesis" or "callotasis" offers enormous potential in reconstruction of both skeletal and soft tissue defects. The technique utilizes corticotomies of varying geometry and a flexible ring external fixation unit which allows for distraction of bone segments (Figure 4). Several months are needed to regain skeletal length and to allow for maturation of woven bone formed during distraction so that unprotected loading may commence. For pure lengthening, the frame is on an average of one month (thirty days) for each one cm (ten mm) of lengthening.

Though the device is cumbersome, technically difficult to apply, and requires complex inventories of equipment and frequent manipulations during treatment, potential advantages are appealing. The technique often can be done in one stage without the need for additional bone grafting or soft tissue coverage. Skeletal length and alignment can be manipulated simultaneously, and the size of the osseous defect, in theory, presents no impediment.[31]

BIOMATERIALS

Demineralized Bone Matrix

Based on animals studies, several authors[5,10] have considered the use of demineralized bone matrix (DMB) as a graft in clinical situations for repair of segmental defects. These studies have consistently demonstrated osteoinduction (chemotaxis of progenitor cells and their attachment to demineralized matrix), followed by formation of a cartilagenous matrix (which undergoes calcification, replacement by osteoid and mineralization). Advantages of DMB include ease of preparation, abundant supply, long storage life, and, at least experimentally,

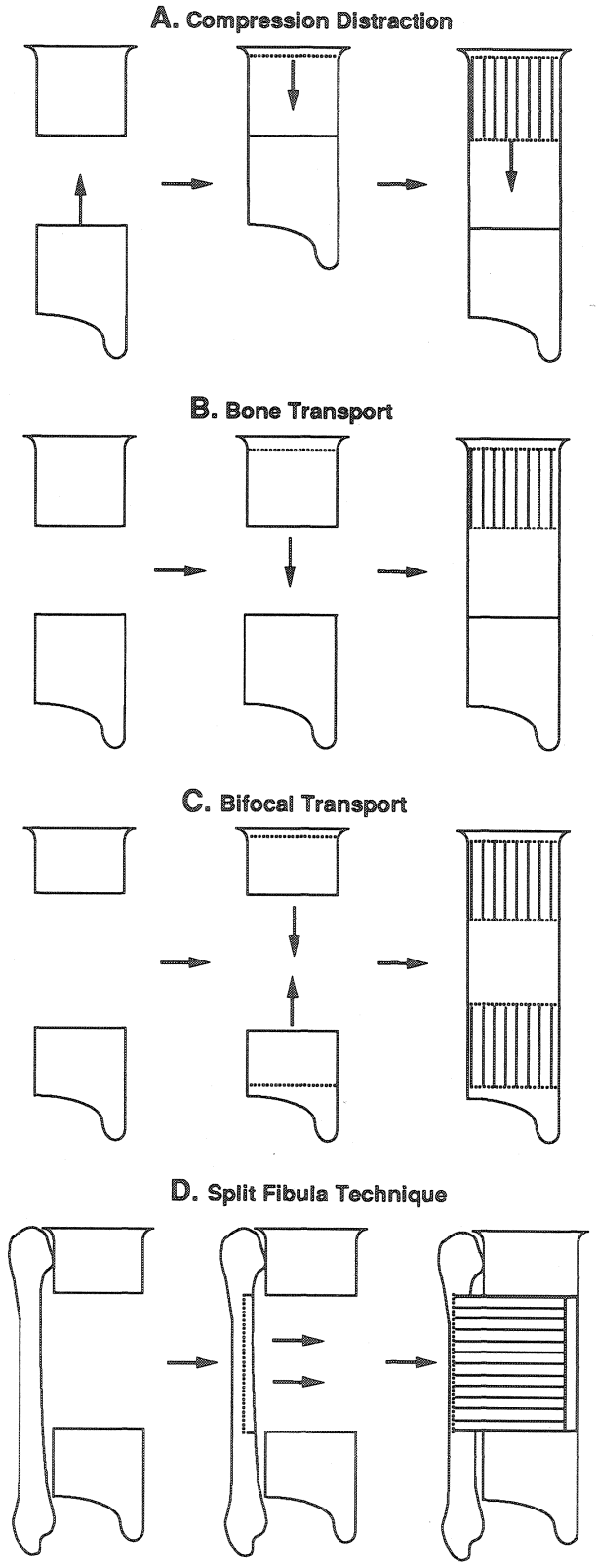

Figure 4
Distraction osteogenesis. Dashed lines represent corticotomy sites; lined regions depict new bone trailing the transported segment(s).

induction of bone that biomechanically behaves like bone observed in the early stages of normal fracture repair[10] or bone formed after autogenous cancellous grafting.[5] Use of DMB would reduce the morbidity associated with autogenous graft harvest and have a likely advantage over bulk autograft is probably never fully incorporated by the host. No series have described the use of DMB for reconstruction of human diaphyseal defects, though DMB has been used successfully in human craniofacial reconstruction and spinal fusion.

Ceramics

Alumina ceramics have good biocompatibility and high mechanical strength. These materials have been used for articulating surfaces in total hip arthroplasty, segmental replacements of long bone defects, and vertebral body replacements. These ceramics do not form a chemical bond with osseous tissue and are prone to loosening at the bone-implant interface. Calcium phosphate ceramics (hydroxyapatite, tricalcium phosphate) form tight chemical bonds with osseous tissue. Their low mechanical strength has limited their use to non-weightbearing structures, filling defects, or coating of hip prostheses.

A glass ceramic containing apatite and wollastonite has been created which has high mechanical strength as well as ability to form a chemical bond with bone. This material has been successfully applied to long bone defects experimentally created in a rabbit model[36] but, like DMB, no series have described its clinical application to human long bone defects.

Prosthetic Implants

Noncoated, cemented, custom and modular implants have been widely used about the knee following segmental tumor resection. Because all ligament constraint at the knee joint is removed, a hinged prosthesis has to be used in the basic design. Fixation to bone of the long component remains the most critical problem, and coated implants (e.g., fiber metal, hydroxyapatite) may provide more satisfactory long term fixation.[36]

Failure of total knee prostheses, particularly fixed hinged devices, may result in substantial bone loss. Revision components can include elongated femoral or tibial stems to make up for lost bone stock. Revision to another hinged device resulted in high complication rates (fracture, subsequent loosening) in one study. The authors recommended use of uncemented or partially cemented nonfixed hinges that are compatible with bone grafting to restore lost bone stock.[21]

Anderson[2] demonstrated successful segmental replacement of long bone defects in baboons using an intercalary fiber titanium implant. Uninterrupted bone formation occurred along the full length of the replacement segment in 70% of the defects created by extraperiosteal resection and supplemental bone graft and in 71% of the subperiostially resected defects without grafts. No bone formation was noted in any defects after extraperiosteal resection without bone grafts.

Kuo[27] applied a similar titanium fiber metal implant for human segmental defects following tumor resections about the knee. The procedure was done in one or two stages and included supplemental cancellous bone grafts packed around the fiber mental implant. The technique, used as a diaphyseal segmental arthroplasty or knee arthrodesis for defects ranging from 9.5 to 18 cm, resulted in a stable weight bearing extremity in sixteen of seventeen patients.

DISCUSSION

Research and new technology have provided an array of effective techniques for the reconstruction of long bone defects. A fundamental question, however, must be addressed prior to embarking on often technically difficult, time consuming, expensive reconstructions that may be associated with high complication rates and patient dissatisfaction: Is the limb worth saving? Multiple factors including etiology of bone loss, age of the patient, site and extent of bone and soft tissue damage, state of distal limb including vascularity and innervation, as well as psychosocial and economic factors are all important. The surgeon also must view aims of treatment from the patient's perspective. These include creation of stable, painless, discharge-free limb that has satisfactory joint motion, muscle power, sensation and, in the lower extremity, length. Treatment time should be minimal and weight bearing should commence early. Clearly, each candidate for reconstruction must be individualized based on careful assessment of all variables.

Retrospective studies have provided certain guidelines for limb salvage (versus amputation) with respect to underlying pathology. Various scoring systems based on duration of ischemia, age, preexisting disease, shock, level of arterial injury, degree of bony/soft tissue injury, etc., have been useful in decision making in massive lower extremity trauma.[16,19,20] Certainly strong arguments favoring primary amputation of the Type IIIC open tibial fracture exist.[7,17] With respect to primary sarcomas of the extremities, limb sparing technology should in no way affect the ultimate survival of the patient compared to traditional amputation. At the same time, the patient should be left with a functional capacity equal to or better than the same patient with an amputation and prosthetic limb.[22] In certain congenital defects, efforts at saving the limb may be inadvisable. For example, amputation may be indicated in management of congenital pseudarthrosis of the tibia after repeated operative failures, when the prognosis is poor, when significant deformity exists or when there is excessive leg length discrepancy.[34]

The decision, therefore, is often one between amputation and early prosthetic fitting versus reconstruction of soft tissue and bone. When informed of the potential duration of treatment and associated risks, some patients elect amputation rather than reconstruction. If reconstruction is chosen, it is important to emphasize to the patient early in the course of management that failure can occur at any stage, and other options, including further efforts at reconstruction or amputation, may be elected.

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MECHANICAL INJURIES OF ARTICULAR CARTILAGE

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Normal synovial joint function depends on the smooth, low friction gliding surface provided by articular cartilage. Although at most only a few millimeters thick, articular cartilage has surprising stiffness to compression and resilience. It has exceptional ability to distribute loads, thereby minimizing peak stresses on subchondral bone. Perhaps most important, it has remarkable durability and is able to provide normal joint function for 80 years or more in many people. Yet despite the durability of articular cartilage under normal joint loading, excessive joint loading can damage articular cartilage causing loss of joint motion, instability, deformity and pain.

Open articular surface injuries including osteochondral fractures, penetrating injuries and dislocations can be diagnosed easily, but probably occur much less frequently than closed injuries of articular cartilage (i.e., injuries that do not penetrate or expose the interior of the joint). Experimental work has shown that excessive joint loading and blunt trauma subject articular cartilage to intense compression and shear forces. These forces can damage the cartilage matrix and cells, altering the mechanical properties of cartilage without disrupting the tissue. They can also fracture or rupture the cartilage matrix.[1,2,3,4] Clinical observations suggest that progressive cartilage degeneration follows these injuries in at least some patients. Less frequently, acute closed injuries fracture cartilage and the underlying bone.[5,6,7,8,9,10,11,12,13,14]

The clinical presentations, frequencies and natural histories of cartilage injuries caused by mechanical forces are less well understood than those of injuries to the other musculoskeletal tissues including bone, tendon, ligament and muscle. The actual mechanisms of cartilage injuries have not been well defined. Closed articular surface injuries are difficult if not impossible to detect by physical examination and plain radiographs do not directly show cartilage injuries. Magnetic resonance imaging cannot at present provide clear demonstrations of cartilage injuries and sensitive clinical measures of cartilage mechanical function do not currently exist. Therefore, there is no reliable and clinically useful method of detecting articular surface injuries that do not cause visible disruption of the tissue. Injuries that do cause tissue disruption can only be reliably evaluated by direct inspection at arthroscopy or arthrotomy.[15,16,17]

Arthroscopic examinations of injured joints suggest that closed articular surface injuries occur frequently. One group of surgeons arthroscopically examined 85 knees with traumatic hemarthroses and no evidence of ligamentous instability. Twenty percent of these knees had chondral fractures or an articular surface defect.[15] In many patients, cartilage injuries are associated with injuries to other tissues of the synovial joint including menisci, ligaments, joint capsule and synovium.[15] The cartilage injury may be overlooked, and if identified it is difficult to distinguish the effects of the cartilage injury from the effects of the injuries to the other tissues.

Advancement in the understanding of mechanical injuries to articular surfaces and the development of better methods to diagnose and treat cartilage injuries depends on knowledge of the types of cartilage injury and the response of the synovial tissues to these injuries. This article first discusses the different types of closed mechanical injuries of articular cartilage, and subsequently describes the response of the synovial joint to these injuries.

DIFFERENCE AMONG MECHANICAL INJURIES OF ARTICULAR CARTILAGE

All mechanical injuries of articular cartilage result from forces applied to tissue. The injuries differ in the extent and type of tissue damage and the response of the tissue to the injury. Joint trauma can damage the matrix macromolecular framework and cells without causing mechanical disruption of the tissue or it can fracture or rupture the cartilage matrix causing visible splits in the articular surface.[5,6,7,8,18,19,20,21] Since cartilage lacks blood vessels, damage to the cartilage alone does not cause inflammation. If an injury disrupts cartilage and subchondral bone, the damage to bone blood vessels causes inflammation and initiates the fracture healing process.[4] The clot and repair tissue from bone can then fill the articular cartilage defect and follow the sequence of inflammation, repair and remodeling similar to the repair process in ligaments. Unlike the repair tissue in ligaments, the repair tissue that fills cartilage defects initially differentiates toward articular cartilage rather than toward dense fibrous tissue.

The extent of tissue damage separates mechanical injuries of cartilage into three types:[3,4,23,24,25] 1) disruption or alteration of the macromolecular framework, loss of matrix macromolecules or cell injury without visible tissue disruption, 2) disruption of articular cartilage alone (i.e., chondral fractures) and 3) mechanical disruption of articu-

lar cartilage and subchondral bone (i.e., osteochondral fractures). Each of these types of cartilage damage presents a different problem for repair and stimulates a different response (Table 1[23]), but the categories overlap. Progressive loss of matrix macromolecules or disruption of the organization of the matrix macromolecular framework eventually results in mechanical disruption of the tissue. Mechanical disruption of the cartilage and bone may release tissue factors that stimulate matrix degradation and loss of matrix macromolecules.

TABLE 1

CLOSED MECHANICAL INJURIES TO ARTICULAR CARTILAGE

Injury Type	Clinical/Radiographic Presentation	Tissue Response	Potential For Healing
Damage to matrix and/or cells without visible disruption of the articular surface	No known symptoms Direct inspection of the articular surface and current clinical imaging methods cannot detect this type of injury	Synthesis of new matrix macromolecules Cell proliferation?	If the basic matrix structure remains intact and enough viable cells can restore the normal tissue composition, the injury will heal. If the matrix and/or cell population sustains significant damage or if the tissue sustains further damage, the lesion may progress to alter the mechanical properties of the tissues
Cartilage Disruption	May cause mechanical symptoms, synovitis and joint effusions.	No fibrin clot formation or inflammation. Synthesis of new matric macromolecules and cell proliferation, but new tissue does not fill the cartilage defect.	The lesion may or may not progress depending on the size of the lesion, the structural integrity, stability, and joint alignment.
Cartilage and Bone Disruption (Osteochondral Fractures)	May cause mechanical symptoms, synovitis and joint effusions.	Formation of a fibrin clot, inflammation, invasion of new cells and production of new tissue.	The lesion may or may not progress depending on the size of the lesion, the structural integrity, stability, and joint alignment.

CARTILAGE INJURY WITHOUT TISSUE DISRUPTION

Experimental work has shown that acute or repetitive blunt trauma can damage cartilage (i.e., cause a decrease in matrix proteoglycan concentration and possibly alter the collagen meshwork and injure chondrocytes) without causing visible tissue disruption.[1,2,3,19,20,21,22,24,26] Other causes of this type of cartilage injury include traumatic or surgical disruption of the synovial membrane that leaves the articular surface exposed, prolonged joint immobilization, some medications, joint irrigation with some types of fluids, and synovial inflammation.[3,4,20,24,25,27,28,29] The intensity and type of cartilage loading that can cause tissue damage without visible disruption has not been well defined.[2] Physiologic levels of joint loading do not appear to cause cartilage injury. Impact loading above that associated with normal activities, but less than that necessary to produce cartilage disruption, can cause alterations of the cartilage matrix.[20]

Maintenance of normal cartilage structure, composition and function require a minimal level of joint loading and motion. Increased joint loading and motion, up to a certain level, may increase cartilage matrix synthesis relative to matrix degradation. In dogs, increased loading of a limb, due to cast immobilization of the opposite limb or moderate running exercise (4 km/per day), increased cartilage glycosaminoglycan concentration and thickness.[30] Yet, strenuous running (20 km/day) reduced cartilage thickness and glycosaminoglycan concentration in normal joints, suggesting that loading and motion above a certain level may adversely affect articular cartilage. Disturbances of neuromuscular control of joints, articular surface incongruities, or ligamentous instability can presumably increase

loading of at least some regions of the articular surface and thereby increase the probability of cartilage damage.[2,31]

Matrix damage without tissue disruption has not been studied extensively, but experimental evidence shows that the first signs of damage are a loss of proteoglycans or alteration of their organization (in particular, a decrease in proteoglycan aggregation). The loss of proteoglycans may be due either to increased degradation or decreased synthesis.[3,4,24,27,28,29] This decreases cartilage stiffness and increases its hydraulic premeability.[25,32] This may cause greater loading of the remaining macromolecular framework, including the collagen fibrils, increasing the vulnerability of the tissue to further damage from impact loading.[25]

Mechanical injuries may cause other matrix abnormalities such as a distortion of the collagen fibril meshwork or a disruption of the collagen fibril-proteoglycan relationship. They may also injure chondrocytes. Alterations in the cartilage matrix of dogs following impact loading included cartilage swelling, increased cartilage collagen fibril diameter and a disturbance of the relationship between collagen and proteoglycan.[20] Presumably, these changes represent more severe matrix damage than a decrease in proteoglycan concentration.

Currently there is no clinically applicable method of detecting alterations in cartilage matrix composition such as decreased proteoglycan concentration or increased water concentration. Nor is there a reliable, accurate method of clinically measuring cartilage mechanical properties. However, when probing the articular surface surgeons sometimes find regions of apparent "softening" that may have resulted from alterations in the matrix. It is not known if cartilage "softening" progresses to further matrix disruption.

The ability of chondrocytes to sense changes in matrix composition and synthesize new molecules makes it possible to repair the damaged macromolecular framework. Evidence suggests that following a loss of proteoglycans the cells increase synthesis of these macromolecules and restore the matrix concentration of proteoglycans toward normal. As a result, the material properties of the matrix return toward normal. Following significant depletion of proteoglycans, the process of repairing the matrix may require many weeks and possibly months.[4,27,28,29]

If the cells do not repair significant matrix macromolecular abnormalities, or if the loss of matrix molecules progresses, the tissue will deteriorate. It is not clear at what point this type of injury becomes irreversible and leads to progressive loss of articular cartilage. If the fibrillar collagen meshwork remains intact and enough chondrocytes remain viable, the chondrocytes can restore the matrix as long as the loss of matrix proteoglycan does not exceed what the cells can rapidly produce.[3,25] When these conditions are not met and the cells cannot restore the matrix, the chondrocytes will be exposed to excessive loads and the tissue will degenerate.

Immobilization, exposure of articular cartilage, and inflammation may predispose the articular surface to these mechanical injuries. Therefore, it seems advisable to minimize impact loading of cartilage in these situations.

CHONDRAL FRACTURES

Impact loading, twisting and direct blows to synovial joints occur frequently, especially during vigorous activity. The resulting compression or shear forces applied to an articular surface can rupture the cartilage matrix producing chondral fissures, flaps or fractures.[5,6,8,10,11,12,13,18,33,34]

A substantial force is required to disrupt normal articular cartilage with a single impact. A study of human articular cartilage subjected to blunt trauma showed that articular cartilage could withstand impact loads of up to 25 newtons per square millimeter (25 MPa) without apparent damage. Impact loads exceeding this level caused chondrocyte death and matrix fissures.[21] The authors suggested that the stress level required to cause cartilage damage was greater than that necessary to fracture the femur. Another study[35] measured the pressure on human patellofemoral articular cartilage during impact loading and found that impact loads less than the level necessary to fracture bone caused stresses greater than 25 MPa in some regions of the articular surface. With the knee flexed 90 degrees, 50% of the load necessary to cause a bone fracture produced joint pressures greater than 25 MPa for nearly 20% of the patellofemoral joint. At 70% of this load nearly 35% of the contact area pressures exceeded 25 MPa. At 100% of this load, 60% of the patellofemoral joint pressure exceeded 25 MPa. These results suggest that impact loads can disrupt cartilage without fracturing bone.

Repetitive impact loading splits articular cartilage matrix and initiates progressive cartilage degeneration.[19,36] In vitro cyclic loading of human cartilage caused surface fibrillation,[36] and periodic impact loading of bovine metacarpophalangeal joints combined with joint motion caused degeneration of articular cartilage.[26] Repeated overuse of rabbit joints in vivo combined with peak overloading caused articular cartilage damage including formation of chondrocyte clusters, fibrillation of the matrix, thickening of subchondral bone and penetration of subchondral capillaries into the calcified zone of articular cartilage.[19] The extent of cartilage damage appeared to increase with longer periods of repetitive overloading, and deterioration of the cartilage continued following cessation of excessive loading. This latter finding suggests that some cartilage damage is not immediately visible.

An investigation of cartilage plugs also revealed that repetitive loading disrupted the tissue and the severity of the damage increased with increasing load and increasing

number of loading cycles.[22] Two hundred and fifty cycles of a 1000 pound per square inch compression load caused surface abrasions. Five hundred cycles produced primary fissures penetrating to calcified cartilage and 1000 cycles produced secondary fissures extending from the primary fissures. After 8000 cycles the fissures coalesced and undermined cartilage fragments. Higher loads caused similar changes with fewer cycles. The experiments suggested that repetitive loading can cause propagation of vertical cartilage fissures from the joint surface to calcified cartilage and extension of oblique fissures into areas of intact cartilage, creating cartilage flaps and free fragments.

The clinical diagnosis of chondral fractures is difficult.[12,13] Because cartilage lacks innervation, injuries limited to cartilage do not cause pain directly. The most common symptoms of chondral fractures include an effusion, locking, catching or giving way of the joint. Arthroscopy is the best current method of evaluating chondral injuries and can show chondral damage not detectable by other methods.[5,8,9,15,16,17,34,37,38] Important variables in the arthroscopic classification of acute and chronic chondral lesions include the condition of the articular surface and the depth, diameter and locations of the damage.[16]

Articular cartilage fissures, flaps and free fragments are seen clinically similar to those produced experimentally by single and repetitive impact loads.[5,6,7,8] In at least some patients, acute impact loading of the articular surface or twisting movements of the joint apparently caused these injuries.[5,6,10,12,13,34] In other patients, the cartilage damage may have resulted from repetitive loading. Frequently, other joint injuries, including rupture of the anterior cruciate ligament and meniscal tears in the knee occur in association with the cartilage damage.

The lack of blood vessels and cells that can repair significant tissue defects limits the response of cartilage to injury.[3,4,24,25,39,40,41] Because cartilage lacks blood vessels these injuries do not cause hemorrhage, fibrin clot formation or inflammation. The local response to injury depends entirely on chondrocytes. Undifferentiated mesenchymal cells cannot migrate to the site of injury, proliferate, differentiate, and synthesize a new matrix. Chondrocytes do not migrate through the matrix. They respond to tissue injury by proliferating and increasing the synthesis of matrix macromolecules near the injury.[3,4,24,25] Yet, the newly synthesized matrix and proliferating cells do not fill the tissue defect, and soon after injury the increased proliferative and synthetic activity ceases.

Current treatments of chondral fractures and flaps include debridement of the cartilage edges, debridement of the cartilage and abrasions of exposed subchondral bone and replacement of cartilage fragments with tissue adhesives.[12,13,18,38,42,43] It is not clear which treatment provides the best results or if any of these treatments produce better results than removing loose cartilage fragments and leaving the defect site untreated.

OSTEOCHONDRAL FRACTURES

Closed joint trauma can cause fractures that extend through cartilage into subchondral bone.[5,6,7,8,10,11,12,13,14,34] Chondral fractures and osteochondral fractures may result from similar impact and twisting joint injuries, but they tend to occur in different age groups.[5,6,8] Chondral fractures generally occur in the skeletally mature people, while osteochondral fractures typically occur in the skeletally immature or young adults. This difference may result from age related changes in the mechanical properties of the uncalcified cartilage, the calcified cartilage zone and the subchondral bone.

Large osteochondral fractures can be treated by reduction and mechanical fixation of the fragments. This must be done soon after the injury or the fragments will remodel making accurate reduction difficult. In addition, the articular surface may begin to deteriorate.[7] Experimental work shows that immediate anatomical reduction and rigid stabilization of osteochondral fractures produces the best results. Intra-articular fractures of the distal femur in rabbits were anatomically reduced and stabilized by compression fixation. The bone fracture healed with apparently normal articular cartilage;[44] but whether inadequately or adequately reduced, fractures that were not stabilized by compression fixation healed with fibrocartilage.

Unlike injuries limited to cartilage, fractures that extend into subchondral bone cause hemorrhage, fibrin clot formation and activate the inflammatory response. Experimental studies have clarified the complex sequence of vascular and cellular events that follow acute osteochondral defects.[3,24,25,39,40,41,45,46,47,48,49,50,51,52,53] Soon after injury, blood escaping from the damaged bone forms a hematoma that temporarily fills the injury site. Fibrin forms within the hematoma and platelets bind to fibrillar collagen to establish hemostasis. A continuous fibrin clot fills the bone defect and extends for a variable distance into the cartilage defect. Platelets within the clot release vasoactive mediators and growth factors or cytokines (small proteins that influence multiple cell functions including migration, proliferation, differentiation and matrix synthesis) including transforming growth factor beta and platelet derived growth factor.[4] Bone matrix also contains growth factors including transforming growth factor beta, bone morphogenic protein, platelet derived growth factor, insulin like growth factor I, insulin-like growth factor II and possibly others. Release of these growth factors may have an important role in the repair of osteochondral defects. In particular, they probably stimulate vascular invasion and migration of undifferentiated cells into the clot and influ-

ence the proliferative and synthetic activities of the cells.

Shortly after entering the tissue defect the undifferentiated mesenchymal cells proliferate and synthesize a new matrix. Within two weeks of injury, some mesenchymal cells assume the rounded form of chondrocytes and begin to synthesize a matrix that contains type II collagen and a relatively high concentration of proteoglycans.[3,4,24,25] These cells produce regions of hyaline-like cartilage in the chondral and bone portions of the defect. In many osteochondral defects the regions of hyaline-like cartilage first appear next to the exposed bone matrix leaving the central region of the defect filled with fibrous tissue.[45]

Six to eight weeks following injury the repair tissue within the chondral region contains many chondrocyte-like cells in a matrix of type II collagen, proteoglycans, some type I collagen and noncollagenous proteins.[45] Unlike the cells in the chondral portion of the defect, the cells in the bone portion produce immature bone, fibrous tissue and hyaline-like cartilage. They soon restore the original level of subchondral bone.

Six months after injury the mesenchymal cells have repaired the bone defect with mostly bone, but also containing some regions of fibrous tissue, small blood vessels and hyaline cartilage.[45] In contrast, the chondral portions of large osteochondral defects rarely fill completely with repair tissue (in animal experiments repair tissue filled about two thirds of the total volume of the chondral portion of large osteochondral defects and more than 95% of the total volume of the bone portion of the defects).[4,24] This tissue in the chondral portion of the defect differs significantly in composition from the tissue in the bone portion of the same defect.[45] The chondral repair tissue contains no bone or blood vessels and has a significantly higher proportion of hyaline-like cartilage. Usually, it has a composition and structure intermediate between hyaline cartilage and fibrocartilage and it rarely replicates the elaborate structure of normal articular cartilage. This differentiation of the repair tissue in the chondral and bony parts of the same defect shows that the environment in the two regions causes the same repair cells to produce different types of tissue. It is not clear whether the important differences in environment are mechanical, biological, electrical, or due to unknown factors.

Occasionally, the cartilage repair tissue persists unchanged or progressively remodels to form a functional joint surface. But in most large osteochondral injuries the chondral repair tissue undergoes depletion of matrix proteoglycans, fragmentation and fibrillation, increasing collagen content and loss of cells with the appearance of chondrocytes within a year or less.[3,4,24,25,51,53] The remaining cells often assume the appearance of fibroblasts as the surrounding matrix evolves into densely packed collagen fibrils. The fibrous tissue usually fragments and often disintegrates leaving areas of exposed bone.[3,24,25,53]

The inferior mechanical properties of cartilage repair tissue may be responsible for its frequent deterioration.[25] Repair tissue that successfully fills osteochondral defects lacks the stiffness of normal articular cartilage. Cartilage repair tissue formed in rabbit metatarsophalangeal joint arthroplasties deformed more easily and took longer to recover from deformation than normal articular cartilage.[54] Repair cartilage formed in pig joints swelled more in Ringer's solution and had greater permeability and less stiffness on compression than normal cartilage.[55] Detailed study of chondral repair cartilage in primate osteochondral defects showed the repair tissue was more permeable and less stiff on compression than normal articular cartilage.[56]

Differences in matrix composition and organization may explain the differences between the mechanical properties of repair cartilage and normal cartilage, and the frequent deterioration of repair cartilage.[25] The increased swelling of repair cartilage may result from a lack of organization or weakness of the collagen fibril meshwork. Microscopic studies of repair cartilage support this suggestion, showing the orientation of the collagen fibrils in even the most hyaline-like cartilage repair tissue does not follow the pattern seen in normal articular cartilage.[25,45] In addition, the cells may fail to establish the normal relationships between matrix macromolecules, and in particular, the relationship between cartilage proteoglycans and the collagen fibril network. This might occur because of lack of organization of the macromolecules or the presence of molecules like type I collagen that may interfere with the assembly of a normal cartilage matrix. The decreased stiffness and increased permeability of repair cartilage matrix may increase loading of the macromolecular framework during joint use, resulting in progressive structural damage to the matrix collagen and proteoglycans. This exposes the repair chondrocytes to excessive loads further compromising their ability to restore the matrix.

Clinical experience and experimental studies suggest that the success of chondral repair in osteochondral injuries may partially depend on the severity of the injury (as measured by the volume of tissue or surface area of cartilage injured), and the age of the individual.[4] Smaller osteochondral defects that do not alter joint function heal more predictably than larger defects that may change the loading of the articular surface.[4,24,45,49] Potential age related differences in healing of chondral and osteochondral injuries have not been thoroughly investigated. Bone heals more rapidly in children than in adults and the articular cartilage chondrocytes in skeletally immature animals show a better proliferative response to injury and synthesized larger proteoglycan molecules than those

from mature animals.[57,58,59,60] Furthermore, a growing synovial joint has the potential to remodel the articular surface to decrease the mechanical stresses created by a chondral or osteochondral defect.

SUMMARY

Blunt trauma, twisting or impact loading of a joint can damage articular cartilage without disrupting the surrounding soft tissues. At least some mechanical injuries of articular cartilage lead to progressive degeneration of the joint surface. Understanding the mechanisms, natural history and optimal treatment of these cartilage injuries is limited, partially because of the difficulty in defining the tissue damage. Arthroscopic examination of injured joints shows visible articular surface disruption occurs frequently in association with other joint injuries. Experimental studies show that excessive joint loading causes three types of articular cartilage damage: 1) loss of matrix macromolecules, alteration of the macromolecular framework or cell injury without visible tissue disruption, 2) disruption of articular cartilage alone (i.e., chondral fractures and flaps) and 3) disruption of articular cartilage and subchondral bone (i.e. osteochondral fractures).

Loss of matrix macromolecules or alteration of the organization of matrix macromolecules can alter cartilage mechanical properties and may make the tissue more vulnerable to further mechanical injury. If a cartilage injury decreases proteoglycan aggregation or proteoglycan concentration, but the fibrillar collagen meshwork of the matrix remains intact and enough cells remain viable, the chondrocytes can usually replace lost matrix macromolecules. Repetitive excessive loading weakens the cartilage macromolecular framework before visible matrix disruption occurs. These observations may help explain the frequently reported association between joint instability and deterioration of the articular cartilage. That is, joint instability causes repetitive excessive loading of some regions of the articular surface. This leads to decreased proteoglycan aggregation and concentration, increasing matrix permeability and decreasing matrix stiffness. These changes increase loading of the matrix collagen fibril meshwork which eventually fails as visible fissures appear.

Disruption of cartilage alone stimulates chondrocyte proliferation and matrix synthesis surrounding the injury, but this response does not persist and the cells do not migrate into the tissue defect or fill the defect with new matrix. The natural history of these injuries is not well understood, but clinical experience suggests that large lesions in load bearing areas of the articular surface cause symptoms and may progress. Current treatments of chondral fractures and flaps do no predictably restore the articular surface.

Injuries that extend through cartilage into the subchondral bone cause hemorrhage and inflammation, followed by repair that fills the bone defect and a variable portion of the cartilage defect with new cells and matrix. Six months after osteochondral injuries, mesenchymal cells have filled the bone defect with a tissue consisting primarily of bone and some fibrous tissue, small blood vessels and hyaline cartilage. In contrast, the chondral portions of large osteochondral defects rarely fill completely with repair tissue; and this tissue differs significantly in composition from the tissue in the bone portion of the same defect. It contains no bone or blood vessels and has a significantly higher proportion of hyaline-like cartilage. Usually, it has a composition and structure intermediate between hyaline cartilage and fibrocartilage.

Occasionally, the cartilage repair tissue persists unchanged or progressively remodels to form a functional joint surface. But in most large osteochondral injuries the chondral repair tissue loses matrix proteoglycans and cells within a year or less. The collagen content increases and the matrix fibrillates and fragments. The remaining cells assume the appearance of fibroblasts as the surrounding matrix becomes densely packed collagen fibrils. This fibrous tissue often disintegrates leaving areas of exposed bone.

Cartilage injuries caused by mechanical forces occur frequently and may have significant consequences, but remains less well understood than injuries to the other musculoskeletal tissues. Advances that will improve the diagnosis and treatment of these injuries include better understanding of the effects of acute repetitive loading of joints, development of clinically applicable methods of measuring cartilage mechanical properties and methods of stimulating regeneration of articular cartilage in chondral and osteochondral injuries.

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UPPER EXTREMITY LENGTH EQUALIZATION

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ABSTRACT

Significant upper extremity length inequality is uncommon but can cause major functional problems. The ability to position and use the hand may be impaired by shortness of any of the long bones of the upper extremity. In many respects upper and lower extremity length problems are similar. They most commonly occur after injury to a growing bone and the treatment modalities utilized in the lower extremity may be applied to the upper extremity. These treatment options include epiphysiodesis, shortening osteotomy, angulatory correction osteotomy and lengthening.

This report reviews the literature relative to upper extremity length inequality and equalization and presents an algorithm for evaluation and planning appropriate treatment for patients with this condition. This algorithm is illustrated by two clinical cases of posttraumatic shortness of the radius which were effectively treated.

INTRODUCTION

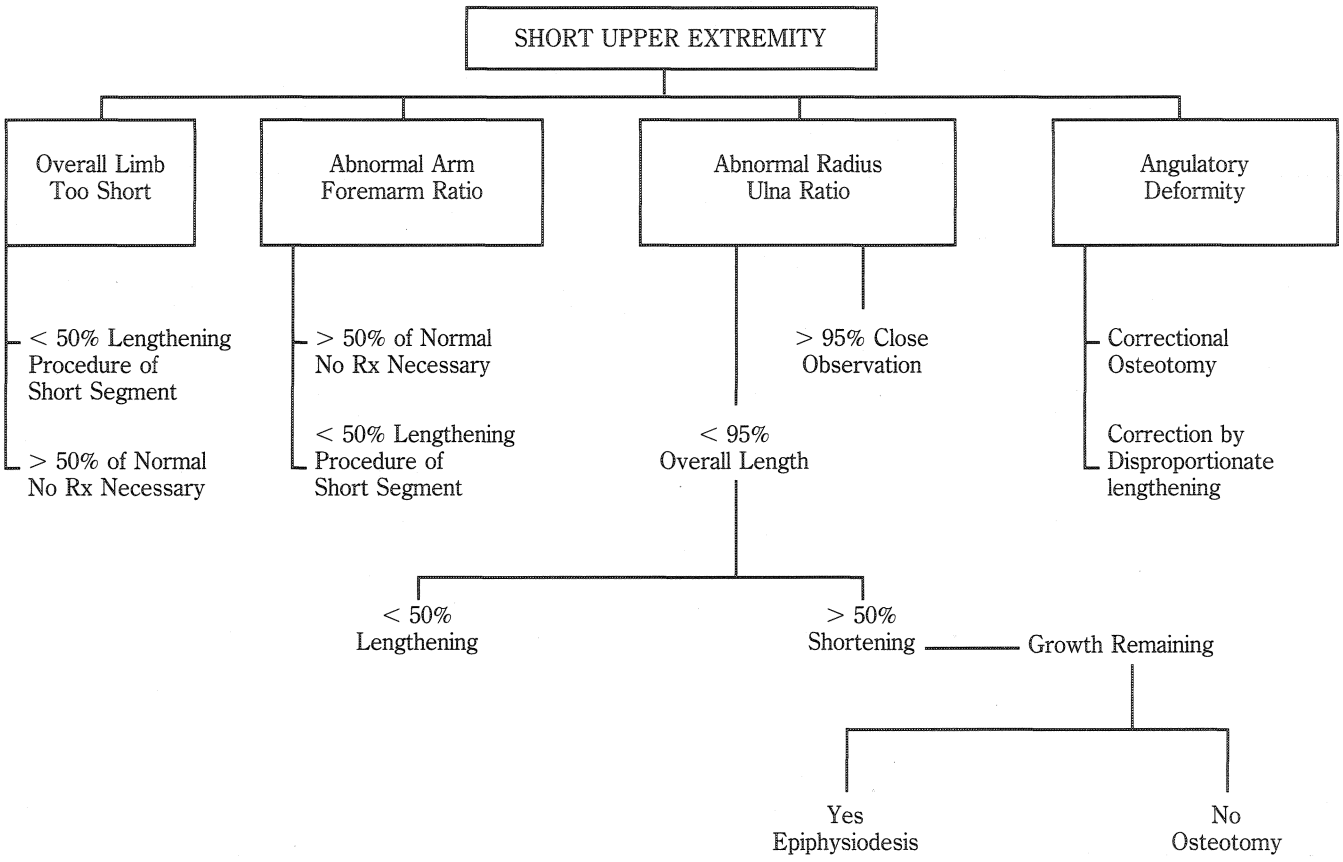
Significant upper extremity length inequality is a relatively uncommon condition and when it does occur it only rarely causes functional problems. Skeletal shortness can result from trauma to the physis of any of the bones of the upper extremity. Severe shortening can be a problem by interfering with muscle function or the ability to position the hand in space. Upper extremity length inequality is less commonly a cause of functional problems in patients than similar length differences in the lower extremity. Leg length inequality and the problems of limp and tilt are much more familiar to the practicing orthopaedist. Small differences in upper extremity length cause cosmetic problems and may require such treatment as special tailoring of cuff lengths.

A variety of functional problems can occur from length differences in the upper extremity including: a short upper extremity, a short ratio between the arm and forearm, abnormal proportion between the radius and ulna and angulatory deformities. Disproportionately of the radius and ulna and resultant loss of function from relative ulnar or radial overgrowth is the most common of the upper extremity length problems. The functional problems attributed to upper extremity length inequality include acquired scoliosis, inability to position and use the hand in space, muscle dysfunction, loss of range of motion, loss of hand function due to carpal motion loss or abnormal mechanical alignment, loss of pronation and supination, premature degeneration and cosmesis.

GENERAL ALGORITHM

In general the approach to a potential problem of growth inequality in the upper extremity is to determine the cause and the natural history of the condition. Specific attention should be paid to the exact anatomic abnormality and the functional impairment which is anticipated as a result. These growth disturbances can usually be divided into an overall shortness, angulation, or disproportionality between radius and ulna length. The anticipated problems can be categorized as cosmetic, motor weakness and

UPPER EXTREMITY LENGTH INEQUALITY ALGORITHM



dysfunction, abnormal arc or range of motion, inability to position and use the hand or premature degeneration of joint surfaces.

After determination of the natural history of the disorder, calculation of the anticipated deformity at skeletal maturity should be undertaken. Calculations must be made with respect to overall forearm length as well as the proportional lengths of the radius and ulna in order to formulated a treatment plan with a final goal of obtaining a functional limb. This commonly is a limb with correct radius/ulna proportion and forearm length and overall upper extremity length being between 50 and 90 percent of normal with a near normal biomechanical alignment. The specific problems which can be anticipated should be listed and the available treatment options for each of the problems should be identified. If the ulna is going to be too long it can be: (1) shortened by epiphysiodesis, (2) shortened by osteotomy, or (3) relatively shortened by lengthening of the radius. A similar analysis can be made of the radial length. An angulatory osteotomy can be considered when there is abnormal mechanical alignment and small length difference. If the limb or a segment is going to be too short (< 50% of normal) then lengthening will be required as opposed to shortening of the other part. The simplest combination of operative treatments should be chosen for complex deformities.

A sequential approach to operative options ranging from simplest to most complex should be considered (Table 1). Epiphysiodesis is a very effective and minimally invasive way of obtaining or preventing disproportionate growth between the radius and the ulna, and is the preferred choice of treatment when it alone can achieve a goal of appropriate radio-ulnar proportion and acceptable overall length. The secondary line of treatment is angulatory osteotomy which can also combine an element of shortening or occasionally slight lengthening. If the goal cannot be achieved by epiphysiodesis and angulatory osteotomy, or shortening osteotomy then the fourth option is a lengthening procedure. Occasionally lengthening needs to be combined with another treatment.

Treatment Options in Upper Extremity Length Equalization
1) Epiphysiodesis
2) Angulatory Osteotomy
3) Length Adjusting Osteotomy
a. Shortening
b. Lengthening
4) Lengthening
5) Combinations

Table 1. Legend—A sequential approach to operative options ranging from simplest to most complex should be considered.

CASES

Case #1

This patient sustained a displaced distal radius fracture at age one with virtually no subsequent longitudinal growth of the distal radial physis. At age two years she had reduction and plating with repositioning of the carpus and the small distal radial epiphysis on the distal end of the radius. At 5 years 6 months she was evaluated in our clinic. The radius was short and the hand was dysfunctional. The natural history was determined by serial radiographs and plotted on growth curves. At skeletal maturity her radius would be only thirty percent of normal length with functional impairment of the hand from radial subluxation of the carpus, loss of wrist motion, disproportionality of the radius and ulna length and premature degeneration of carpal cartilage. Goals of sixty percent of normal forearm length, correct radial/ulnar proportion and correct mechanical axis were chosen as achievable with the expectation of reasonable function. Three procedures were planned: 1) lengthening, 2) lengthening plus angulatory correction, and 3) epiphysiodesis. At age six years she underwent radial lengthening with corticotomy and distraction osteogenesis using uniplanar external fixation (Fig. 1). The hand was centralized on the carpus as the radius was lengthened. She was over lengthened and the ulna and proximal radius were allowed to grow. The deformity partially recurred as expected as disproportionate ulna growth continued. At age ten the next operation included a second radial lengthening and correction of the flexion and radial deviation of the carpus using the Ilizarov technique (Fig. 2). At age thirteen an ulna epiphysiodesis is planned with an ultimate length of 65% of normal of the forearm with a correct radius/ulna proportion and mechanical axis. She remains at some risk for carpal degeneration but near normal function of the hand and acceptable range of motion is expected.

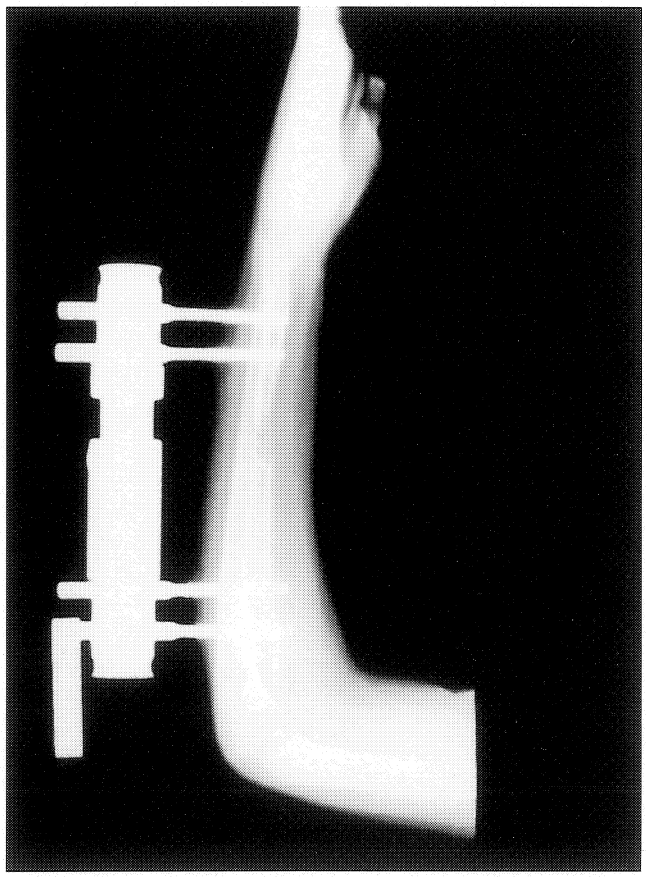


Figure 1
Radial lengthening utilizing an EBI distractor with half pins and a simple uniaxial after corticotomy. There is new bone formation in the distraction gap.

Case #2

This patient sustained a Salter II distal radius fracture at age twelve. He was treated with closed reduction and casting and presented to our clinic fourteen months post injury with pain from ulnar impingement. There had been minimal longitudinal growth of the distal radius after the fracture. Serial radiographs were obtained and the relative lengths at skeletal maturity calculated using Pritchett's modification of Hensinger's normal tables, the percentile for this patient's long bone length and bone age, and the percentage of growth retardation of the distal radius from this injury. The functional problems anticipated from a 1.2 cm shortness of the radius were predicted to be continuation or worsening of the ulnar wrist pain, loss of ulnar deviation and supination with resultant loss of hand function, and possible early degenerative changes of the carpus. No functional problem was anticipated from overall upper extremity shortness of eight percent. Sufficient growth remained to allow for current radius/ulna proportionality from the first line of surgical treatment options: epiphysiodesis. The timing was calculated to result in 92% of forearm length with radius/ulna proportionality equal to the uninjured upper extremity. Distal and proximal ulna epiphysiodeses (Fig. 3) were performed at age 13 years 6 months and he has demonstrated satisfactory fusion of the

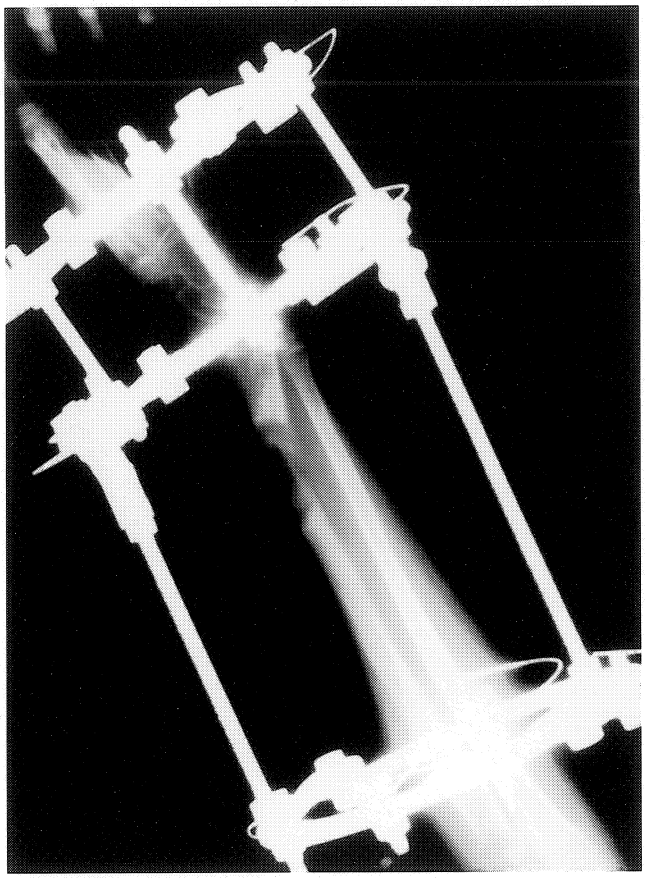


Figure 2
The ring fixator with small transfixing wires was utilized to obtain additional radial length as well as correction of angular deformity at the wrist.

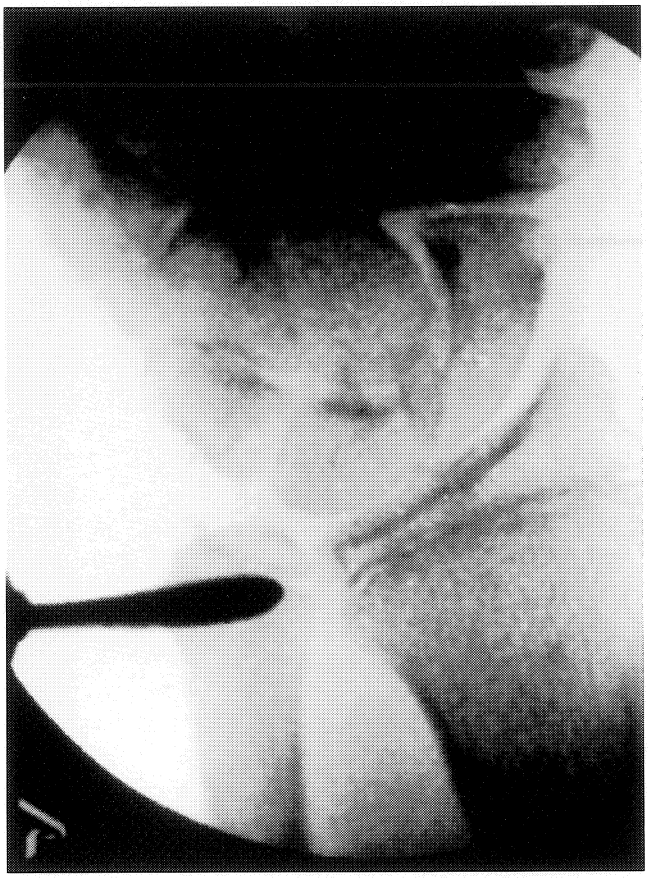


Figure 3
Distal ulna epiphysiodesis is achieved by obliteration of the physis with a burr under fluoroscopic control as illustrated in this figure.

ulna physes. At skeletal maturity his radius/ulna proportion is equal on the right and on the left and the left is 92% percent of the length of the right. At this time he has no functional problems with his wrist. The risk of early degenerative changes of the carpus is low.

DISCUSSION

Upper extremity length problems are less commonly encountered by the practicing orthopaedist than leg length discrepancies. Comparison reveals similarities in causes (physeal injury from fracture or infection, congenital, developmental) and treatments (epiphysiodesis, shortening osteotomy, angulatory osteotomy, lengthening). The upper extremity length problem differs from leg length inequality in the frequency of functional problems (less) and the need for operative treatment (less). The types of problems are generally similar (overall length, proximal/distal ratio, right/left ratio, muscle imbalance, mechanical axis). The upper extremity problems of hand positioning in space and forearm pronation and supination do not have direct counterparts in the lower extremity.

The natural history of upper extremity length has been reported by Hensinger and Pritchett.[1,3] Their studies showed that 80% of the growth of the humerus comes from the proximal growth plate. The radius and ulna on the other hand get the majority of their length, 80% and 85% respectively, from the distal growth plates. The functional problems from upper extremity length inequalities have been reported anecdotally in the literature. Upper extremity length differences may result from injury to a growing long bone physis. The initial injury may be an infection, torus fracture, or may involve the diaphysis.[4] The most typical initial injury is a fracture through a growth plate. Only rarely does acceleration of growth cause a significant length problem. If the entire upper extremity length is at least 50% of normal reasonably good overall function can be anticipated.[4] If the arm or forearm unit is at least 50% of normal, reasonably good overall function can be anticipated.[3] Any disproportionality of radius to ulna length may cause problems and 90% of patients with > 1 cm of radius-ulna length abnormality will have objective functional deficits.[3] Since the majority of radius and ulna

growth comes from the distal physis, this is the area where injury prior to skeletal maturity is most likely to create a length problem. Most growth injuries are partial rather than complete and growth retardation is seen more commonly than total growth arrest. Determination of the natural history requires sufficient time to calculate the remaining growth to accurately predict length at skeletal maturity.

Appropriate analysis includes an assessment of expected length at maturity including overall length of the upper extremity and forearm, relative lengths of the radius and ulna, and mechanical axis. The functional problems expected from any anticipated length problems should be identified.

The selection of appropriate treatment requires a complete knowledge of options available matched with the expected length abnormality. The algorithm presented helps the physician determine the indications for operative intervention.

The achievement of at least fifty percent of normal length while keeping the normal ratio between the radius and ulna usually will eliminate the expected functional problems from length inequalities. This goal should be achieved in the most expeditious and least invasive manner as identified in the algorithm.

CONCLUSIONS

A variety of operative treatments are available for patients with abnormal upper extremity bone length. An algorithm is presented and illustrated for identifying the most appropriate operative treatment which includes a determination of the natural history of the malady with predicted lengths and anticipated functional problems.

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THE PATHOGENESIS OF DIABETIC CHARCOT JOINTS

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Charcot, in 1868, provided an excellent description of the clinical features and bizarre bone and joint changes associated with tabes dorsalis. Since then numerous conditions characterized in part by extremity sensory deficits have been shown to lead to the typical bone and joint destruction of Charcot joints (see Table 1).[4,20,26,27,38] Charcot arthropathy has been described in almost every joint of the extremities and spine. In 1936, Jordan provided the first description of Charcot joints associated with diabetes mellitus.[14,24,35,38,39] Diabetes is now the leading cause of neuropathic joints.[6,7,10,16,38] This paper will specifically address the pathogenesis of Charcot joints in diabetes, with emphasis on the foot and ankle.

TABLE 1

ETIOLOGIC FACTORS IN CHARCOT JOINTS

Diabetes mellitus
Tabes dorsalis
Syringomyelia
Alcoholism
Amyloidosis
Amyotrophic lateral sclerosis
Arachnoiditis
Cerebral palsy
Cerebrovascular accident
Congenital insensitivity to pain
Cord trauma/tumors
Elephantiasis
Hereditary insensitivity to pain
Hereditary sensory radicular neuropathy
Lead poisoning
Leprosy
Meningomyelocele
Multiple sclerosis
Peripheral nerve injury
Pernicious anemia
Yaws

Various other terms have been used to describe Charcot bone and joint changes. These include neuroarthropathy, neuropathic arthropathy, osteoarthropathy, neurotrophic joint, and neuro-osteoarthropathy. These terms reflect the uncertainty of the pathogenesis of Charcot joints and they are used interchangeably with reference to the same clinical phenomena.

The common denominator for neuropathic joints is some decrement of protective sensation. This is true for diabetic neuroarthropathy as well. The incidence of diabetic neuroarthropathy has been reported as one in 680 diabetics[38] to one in 800 diabetics.[13] The diabetic patient with neuroarthropathy of the foot is typically middle-aged or older, has had diabetes for more than 10 years, and is insulin dependent.[6,39] There are case reports of younger patients with juvenile onset diabetes mellitus who have neuropathic joints.[16] Retinopathy, nephropathy, and peripheral vascular disease are often present.[6] The typical presenting complaints are painless swelling, deformity, increased warmth and ulcerations of the foot.[6,26] However, up to one third of patients present with a complaint of pain.[6,13,38] Although the precipitating event is often a minor traumatic episode the patient may be unaware of any recent trauma.

Patients with neuropathic joints are often reported to have bounding peripheral pulses.[4,10,14] However, in Cofield's series of patients with diabetic neuroarthropathy, 38% were reported to have no evidence of peripheral vascular disease, 32% had some evidence, and 30% had significant compromise of the blood supply to the foot.[6]

Most patients with diabetic neuroarthropathy have some evidence of peripheral neuropathy. Many patients complain of numbness of the extremity but this may be difficult to quantify by testing fine touch. The loss of vibratory sensation is the most consistent estimate of the amount of sensory deficit. Using a 128 cycle/sec tuning fork only 7% had normal sensation, 73% had diminished sensation and 20% had absent sensation. The gastrosoleus reflex is most commonly altered; absent in 67% and decreased in 20%.[6] The quadriceps reflex is absent in 30% and decreased in 25%.[6]

The lower extremity, especially the foot and ankle, is most frequently involved in the diabetic. This is in contrast with tabes dorsalis which primarily afflicts the larger joints of the lower extremity, and syringomyelia which most commonly affects the upper extremity.[4,27] In the diabetic the joints most frequently involved are the tarsometatarsal joints (60%), metatarsophalangeal joints (30%), and less frequently the ankle and subtalar joint (10%).[10,38] When the metatarsophalangeal joint is involved it most commonly presents with an associated ulceration. Tarsometatarsal and ankle joint involvement is infrequently present with associated ulceration.[6,38,39] Twenty percent develop bilateral neuropathic joints in the feet.[38]

Radiographically, two types of neuropathic joints are described in the literature - hypertrophic and atrophic.[4,26] The hypertrophic joint has massive juxta-articular new bone formation, osteophytes, osseous debris, and the disruption of the articular surfaces. Brower suggests that the hypertrophic type represents the late, or chronic,

presentation of a Charcot joint, and occurs primarily in weight bearing joints.[4] This may represent the "healing" attempts of a neuropathic joint. The atrophic form presents with osteopenia and osteolysis, the radiographic appearance of extensive bone resorption without evidence of accompanying bone repair. This resorptive pattern is most frequently seen in the upper extremities, and is most commonly associated with syringomyelia or peripheral nerve injuries.[4] However, atrophic Charcot joints have also been described in the feet of diabetics without evidence of infection.[32] Brower suggests that the term "atrophic" is a misnomer in that it does not appropriately describe the underlying mechanism for the radiographic appearance. The "atrophic" joint results from bone resorption in a hypervascular bed with increased osteoclastic activity.[4]

Fractures may precede the onset of a neuropathic joint.[27,29] It has been observed that the fractures often occur without significant trauma or even without a known traumatic event. Brower suggests that these fractures are pathologic because they result from an underlying bone abnormality.[4]

Cofield reported that swelling, diaphyseal osteoporosis, and diaphyseal thinning were seen more often in feet with ulcers. Cartilage loss, metaphyseal osteosclerosis and osteophytes, and juxta-articular bone or calcific debris were more common in feet without ulcers and infections.[6] Harris and Brand described five patterns of joint destruction in the anesthetic feet of patients afflicted with leprosy.[23] Cofield recognized three patterns of bone and joint destruction in the feet of diabetics with neuroarthropathy.[6] The most frequent pattern involved the metatarsophalangeal joints and phalanges and was usually associated with ulceration. The second pattern was that of tarsometatarsal joint destruction. The third pattern was similar to the anterior pillar-medial arch type, described by Harris and Brand[15], where the bones involved are the head and neck of the talus, the navicular, and cuneiforms.[6]

Complications of diabetic neuroarthropathy of the foot result from altered mechanics secondary to foot deformities. Untreated, or improperly treated, neuroarthropathy will lead to progressive deformity, callus formation over areas of excessive weight bearing, ulceration and possibly sepsis necessitating amputation.[27] Difficulties with gait may arise, especially in those with ankle involvement.[6,38] Proper fitting shoes may be difficult to obtain.

Pathologic descriptions of Charcot joints have concentrated primarily on the joint abnormalities. Little attention has been given to the bone itself.[4] The early changes may be similar to osteoarthritis with erosion and fibrillation of cartilage, subchondral trabecular thickening and osteophyte formation. The identification of large amounts of cartilaginous and osseous debris within the synovium has been described as the primary diagnostic determinant for neuroarthropathy. The process may rapidly progress to severe bone and joint destruction. Brower described the pathologic changes of the bone in an atrophic Charcot joint to include widening of the Haversian canals, replacement of the marrow spaces with a fibrovascular stroma, and numerous areas of osteoclastic bone resportion.[4] These findings indicate an increased blood supply to the bone leading to the resorptive appearance of an atrophic Charcot joint. Late findings include fibrous thickening of the capsule, synovial membrane induration with villous transformation, and replacement of articular cartilage by fibrous tissue and fibrocartilage. Callus and large osteophytes form; angular deformities and joint subluxations and dislocations occur.[27,34,38]

PATHOGENESIS OF DIABETIC NEUROARTHROPATHY

What are the pathogenetic mechanisms responsible for the clinical manifestations of diabetic neuroarthropathy? Certainly, they are not without controversy. Peripheral neuropathy with its consequent loss of sensation and proprioception plays a significant role in the pathogenesis of Charcot joints. Loss of pain sensation and proprioception in the foot of a diabetic allows repeated unrecognized trauma which results in rapid and progressive bone and joint destruction with proliferative bone formation. This results in the typical hypertrophic Charcot joint, described as "the worst degenerative arthritis you will ever see."[26]

The investigators of neuroarthropathy can be grouped into two different camps. There are those who believe that loss of sensation and repetitive trauma are the primary pathogenetic mechanisms for Charcot joint destruction.[15,19,23,38] This has been called the neurotraumatic theory. Other investigators believe that there is an underlying bone abnormality which makes the bone susceptible to destructive changes (i.e. fractures and dislocations) with repetitive unrecognized trauma. With respect to diabetes in particular, this latter group can be further divided into those who believe the metabolic effects of diabetes result in susceptible bone[25,30] and those who believe an underlying vascular abnormality weakens the bone.[4,13,28] This concept has been called the neurovascular theory. Charcot, in his original description, believed the joint changes resulted from damage to central nervous system "trophic" centers which controlled bone and joint nutrition. Volkman, and later Virchow, disagreed with Charcot and felt that the joint changes resulted from repeated trauma sustained by a joint unable to sense pain.[4,26]

PATHOGENESIS OF THE NEUROTRAUMATIC THEORY

The neurotraumatic theory is the most widely recognized explanation for the appearance of Charcot joints. The neurotraumatic theory suggests that unrecognized trauma results in occult bone and joint injuries. These go undetected in a patient with a sensory peripheral neuropathy because of diminished pain sensation. The patient continues to traumatize the bone and soft tissues which leads to hyperemia as part of the inflammatory response to injury. This leads to soft tissue swelling and bone deformities. Callous and osteophytes form in an attempt to "heal" the injuries. However, a vicious cycle is set up as the patient continues to traumatize the tissues and the repair responses are not allowed to complete their course resulting in further bony deformities. This results in the easily recognized, late stage of hypertrophic neuroarthropathy.

The classical model for the neurotraumatic theory was described in the 1917 study of Eloesser.[15] He sectioned the lumbar dorsal roots of cats and allowed them to walk after this procedure. This produced arthropathies and spontaneous fractures of the hips and knees. He accelerated the development of Charcot joints by electrocauterizing the articular cartilage of the femoral condyle after resecting the posterior roots of that joint. Through his measurements of the physical composition and tensile strength of the bones from both the normal and abnormal side, he concluded that there was no evidence of "atrophy" or the existence of trophic nerves as proposed by Charcot. He concluded that trauma and the lack of the warning sense of pain were the causes of the bone and joint changes.[15]

Johnson in 1967 reported on observations of neuropathic joints in 118 patients with various etiologies for their neuropathies.[27] Eighty-eight were tabetic in origin and only ten were in diabetics. Johnson's paper is important for the frequent observation of spontaneous fractures and joint injuries leading to Charcot joint changes. He could not identify an underlying bone abnormality predisposing to fracture or joint injury and reported that trauma must be the underlying mechanism leading to Charcot joint changes. He also postulated that the swelling and local heat were due to hyperemia in response to injury. He stated that the hyperemia combined with histiocytic and osteoclastic activity would lead to an early destructive, "atrophic", or invasive phase of repair as after all injures.[27] This phase would occur before any attempts at fracture healing or hypertrophic repair begins. The bone resorption that occurs from the hyperemia weakens the bone and makes it prone to further injury from minor forces. Further trauma leads to more hyperemia and bone resorption resulting in a vicious cycle. If proper protection is provided the cycle will stop and repair will begin. This results in massive soft tissue calcification, and massive callus and osteophytes.[37]

Johnson also performed experiments on dogs and cats similar to Eloesser's study. The right hind limb was made anesthetic by unilateral dorsal root rhizotomies in 18 cats and six dogs. This group of animals was allowed to walk on the denervated extremities. They developed pressure sores and fractures from repeated episodes of gross trauma in their large cages. Another group of animals (six cats and three dogs) were denervated and kept in small cages with narrow mesh wire and therefore were not able to walk freely. One cat and one dog developed ulcers from lying on the denervated extremity but no fractures or joint injuries resulted. Roentgenograms of the hind limbs were reported to demonstrate no changes in any of the animals protected from gross trauma and significant activity. He concluded, in agreement with Eloesser, that the basic causative factor was trauma and that there was no definite predisposing weakness of the bone. He did not perform any biochemical, histological or mechanical studies on the animals' bones.[27]

In the Joslin Clinic study of 101 diabetic patients with Charcot arthropathy, the authors supported the neurotraumatic theory.[38] They suggested that the absence of pain over a period of time may lead to an abnormal range of movement causing repeated "micro-trauma" to the affected joint. They point to the effect of non-weightbearing therapy in allowing ulcers and Charcot joint changes to heal as clinical evidence for the role of trauma. They reported that no experimental studies have proved an underlying bone abnormality. They refute the role of autonomic neuropathy in producing Charcot joints. Autonomic neuropathy is so common in diabetic peripheral neuropathy that one would expect a far higher incidence of Charcot joints.[38]

O'Connor, et al[33] developed another experimental model to evaluate the role of deafferentation and knee joint instability in the development of neuropathic arthropathy. Dogs that were subjected to unilateral dorsal root ganglionectomies failed to show biochemical, gross, or histological evidence of degenerative joint lesions in the femoral condyle after sixteen months when compared to normal controls. They presumed that an unknown neuromuscular mechanism must have been present to protect the canine knee joints and that this mechanism did not depend on ipsilateral sensory input. In the second part of the study dogs that underwent dorsal root ganglionectomy and ipsilateral transection of the anterior cruciate ligament showed gross and histological lesions of the femoral condyle three weeks after ACL transection. None of the control dogs developed any abnormalities other than a slight decrease in safarin-O staining of the superficial zone

cartilage. They concluded that the neuromuscular mechanisms that protect normal joints from damage are inadequate to protect unstable joints from becoming rapidly and severely damaged.[33] From their study they could not predict whether the pathological changes observed would have developed into classic Charcot arthropathy.[33] Joint instability led to their observed pathologic changes. However, it is not clear whether instability precedes articular lesions, or whether effusion and deterioration of the articular and periarticular structures cause instability of the joint.[33]

Finsterbush and Friedman performed a similar study in rabbits.[19] In their experiment the dorsal lumbar sixth, seventh and first sacral roots were resected. They then examined the femoral condyle articular cartilage in the knees immobilized in a plaster cast, and a group of knees that were not immobilized after dorsal root sectioning. Their results revealed progressive atrophy of cells in all structures of the knee joint, whether or not the knee was immobilized in the sensory denervated rabbits. Chondrocyte degeneration started in the middle layers and spread to superficial and deep layers. The number of cells in the synovium of the denervated animals decreased and the predominant cell type changed from type B to type A. They postulated that the effects on the cartilage were mediated through altered nutrition. It was their impression that trauma is not the primary factor in deterioration of anesthetic joints but it must be an important contributing factor.[19]

The proponents of the neurovascular theory point to several clinical, radiographic and pathologic observations which they believe do not support a neurotraumatic pathogenesis. They point out that one third of patients with neuroarthropathy have pain in the involved joint at the time of presentation, and argue that unsensed trauma could not be the initiating event. Neuropathic joints are also known to occur in paralyzed, bedridden and otherwise immobilized patients, who do not persistently subject their joints to repetitive trauma. They theorize that the underlying bone must be abnormal for their low level of activity to result in fractures or neuropathic joints. They also believe that the frequent rapid progression of Charcot joint changes cannot be explained adequately by trauma.

Proponents of the neurovascular theory criticize the study of Eloesser for not examining the bone in the early phase of neuropathic change. It may be that the atrophic stage is an earlier form of neuroarthropathy and the hypertrophic arthropathy is a late stage after repair has taken place. Eloesser also never examined the vascular changes in the bone, thus he could not rule out a role for vascular hyperemia of bone leading to an underlying bony abnormality.[4]

The neurotraumatic theory can adequately explain the appearance of hypertrophic neuroarthropathy. However, it does not adequately explain the atrophic form. The atrophic form gives the appearance of osteopenia and rapidly resorbing bone. The supporters of the neurotraumatic theory suggest that this radiographic appearance may be the result of hyperemia in response to occult trabecular fractures and ligamentous injuries. The supporters of the neurovascular theory suggest that the atrophic Charcot joint results from bone resorption due to an increased blood supply to the bone that results from a neurally mediated vascular reflex. The bone resorption occurs before any trabecular fractures or ligamentous injuries, but it may lead to fractures of the weakened bone.

PATHOGENESIS OF NEUROVASCULAR THEORY

The neurovascular theory suggests that an underlying primary bone abnormality results from a "neurally mediated vascular reflex".[4] This results in increased blood flow causing resorption of bone leading to osteopenia and osteolysis. The bone is then susceptible to pathologic fractures and dislocations, which in the setting of the loss of protective sensation, leads to a Charcot joint. The increased blood flow would explain the clinical observations of the Charcot foot which is hot and swollen at the time of its initial presentation. It would also explain the radiographic appearance of atrophic Charcot joints which have been described in the feet of diabetics.[32] The hypertrophic radiographic appearance is explained as a consequence of attempted repair of the bone and joint destruction and is considered a late sequela of the process.[4]

What is the nature of the "neurally mediated vascular reflex?" Blood flow in the neuropathic feet of diabetics has been noted to be increased. The uptake of technetium methylene diphosphonate is increased both in the early vascular phase and in the late osteoblastic phase in diabetics with Charcot joints.[12,17] The increased technetium uptake has also been noted in diabetic patients with peripheral neuropathy but with normal radiographs.[12,17] This would suggest that the abnormal blood flow occurs before any bone abnormalities which by themselves could lead to hyperemia from fractures and soft tissue injuries. In the study of Eymontt the nuclear scans had both diffuse and focal increased uptake. He postulated that the diffuse uptake could be explained by hyperemia secondary to vasodilatation. The focal increased uptake could be explained by reactive bone formation following repeated trauma.[17]

Doppler studies have also confirmed increased blood flow in the feet of diabetics with peripheral neuropathy.[11,36] Normal blood flow results in a triphasic Doppler pattern with forward flow in systole, followed by

a brief period of reversed flow and then a further short forward flow in diastole. In diabetics with neuropathy this flow pattern is altered with increased forward flow and the absence of reverse flow, and a prolonged diastolic flow. Archer, using mercury strain gauge plethysmography, found the blood flow in the feet of diabetics with peripheral neuropathy was increased on average five times above normal controls.[1] In the same study, the patients with peripheral neuropathy had a mean resting skin temperature of 33.5 degrees Centigrade compared to 25.8 degrees Centigrade in the normal controls. The increased skin temperatures were felt to reflect the high blood flow[1] and are consistent with the clinical observation of warm feet.[13,26,27,38] The increased arterial blood flow will lead to increased blood supply to the bones of the feet.

It is felt that the increased blood flow is a reflection of arteriovenous shunting in the neuropathic foot. Once again, the Doppler sonograms which show rapid forward flow of blood in neuropathic feet are highly suggestive of arteriovenous shunting which allows rapid blood flow to the venous side of the circulation. Boulton has shown that the venous Po_2 is raised and approaches the Po_2 of the arterial side, evidence for arteriovenous shunting.[2] Arteriovenous shunt volume can be measured using radiolabeled human microspheres with a diameter of 20-30 um. Normally these would be trapped in the capillaries. When arteriovenous shunting occurs the microspheres injected into the arterial circulation will go directly to the venous circulation through arteriovenous shunts. Eventually they are trapped in the capillaries of the lungs and measurement of the radioactivity over the lungs can give a measure of shunt volume. The mean shunt volume in diabetic patients with neuropathy was 8.45% compared with 5% in normal controls.[13]

The hemodynamic abnormalities in diabetics with peripheral neuropathy may also be due to changes in the vessel walls. Diffuse atherosclerosis may give rise to increased pulse wave velocity. However, medial wall calcification may also be responsible, leading to stiffening of the arterial wall. Recent studies have demonstrated an increase in pulse wave velocity and elastic modulus in the arteries of the diabetic neuropathic limb.[13] Edmonds demonstrated an association between medial wall calcification and diabetic neuropathy.[40] In lateral radiographs of the foot, 16 out of 20 patients between 22 and 50 years of age were found to have significant calcification of arteries.[40] All of these patients had a severe enough neuropathy to cause foot ulcerations and many had Charcot joints. In a large series of diabetic patients with Charcot joints, 90% were found to have vascular calcification.[38] Arterial calcifications have been described in diabetic and nondiabetic patients following sympathectomy.[20] Eleven out of thirteen patients who underwent unilateral sympathectomy developed Monckeberg's sclerosis on the operated side.[20] The sympathectomy results in atrophy of the arterial smooth muscle with the development of foci of necrosis and eventual medial wall calcification.[13]

The increased blood flow due to arteriovenous shunting and changes in the arterial wall can be explained by the sympathetic denervation, so common in diabetics with peripheral neuropathy.[40] Diabetes leads to both somatic and autonomic nerve damage. A defect of peripheral sympathetic nerves has been documented by direct measurement of sympathetic activity in postganglionic C fibers in the diabetic neuropathic limb.[18] The small arterioles in diabetics have either absent sympathetic nerve fibers or fibers at a significantly greater distance from effector sites compared with controls.[22] Arteriovenous shunts are controlled by sympathetic innervation. One would expect them to open up considerably after sympathetic denervation.[13] Thus small increases in arteriovenous shunt diameter would allow a marked increase in blood flow into the foot. Shim, et al found a five to 45 percent increase in blood flow to the bones of the hind limb of rabbits after division of the sciatic nerve. The sciatic nerve carries almost all of the sympathetic fibers below the knee.[37] Bone formation and structure are closely linked to vascular changes. Resorption of bone occurs as a result of increased blood flow in large venules.[31] Histological studies of Charcot joints have shown marked increase in vascularity with vessel dilatation, enlargement of Haversian canals and an increase in osteoclastic bone resorption.[4] Pathologic fractures are a common finding in the feet of diabetics with peripheral neuropathy.[6,9,13,24,26,27,29,38] These fractures would not occur in normal bones without a significant force. They could not be produced solely by insensitivity of the joint. This suggests a primary underlying bone defect which increases susceptibility to fracture in diabetic neuropathy. The initial bone abnormalities may lead to secondary destruction with subluxation of joints and callus and osteophytes.[28]

In summary, sympathetic denervation of arterioles and arteriovenous shunts leads to increased blood flow. This leads to resorption of bone which makes it prone to damage after minor trauma. Loss of protective pain sensation permits continued mechanical stress to occur and major destructive changes result.

OTHER PATHOGENETIC MECHANISMS

The metabolic effects of diabetes may lead to a loss of bone mass and osteopenia independent of peripheral neuropathy.[25,30] Osteoporosis has been demonstrated in the bones of diabetics using photon absorptiometry. Studies conflict with regard to an association between diabetic control and loss of bone mass. They also conflict as to whether or not the loss of bone mass remains stable or

whether it increases with duration of the disease.[25] The majority of studies suggest that the defect in bone mass occurs quite early in the course of the disease. While loss of bone mass leading to a primary bone abnormality may make it susceptible to pathological fractures and the development of Charcot joints this issue was not addressed by these authors.[25] Further investigations are needed to determine whether loss of bone mass in diabetics can lead to Charcot joints.

Ischemia does not appear to contribute to the development of neuropathic feet in diabetics. As previously mentioned, the neuropathic foot usually presents as a warm, dry, numb and painless foot in which the pulses are palpable. In contrast the ischemic foot is cold, the pulses are absent and it is complicated by rest pain, ulceration and gangrene. Some authors stress that normal pedal pulses are characteristic of feet with neuropathic arthropathy,[4,13,14,38] and there have been reports of neuroarthropathy developing only after successful revascularization procedures.[10]

TREATMENT

The treatment of diabetic neuroarthropathy begins with the recognition of the potential for the problem to exist.[4,10,27,38] In a diabetic with peripheral neuropathy the presence of unilateral warmth and swelling after an episode of minor trauma is suggestive of a developing Charcot joint. Prevention of trauma, daily foot inspection and appropriate adaptive shoes are essential for proper diabetic foot care.[26] Once a neuropathic joint has been diagnosed, further destruction of the joint can be prevented by limiting weight bearing and applying appropriate casts or orthoses. If the Charcot joint is diagnosed early and managed appropriately, the progressive joint destruction can be halted and the prognosis for functional ambulation remains relatively good.[5,10,13,21,18,38,39] The hallmarks of treatment include elevation, non-weight bearing and immobilization until there are radiographic signs of healing. This may require 2-3 months. Special shoes will be required to accommodate deformity and prevent ulceration once the patient begins to bear weight. Clohisy and Thompson suggest the prophylactic orthotic immobilization of the uninvolved extremity in order to protect it from the altered mechanics of the foot during immobilization and crutch walking.[5] The treatment of a fracture in a neuropathic foot involves adequate immobilization and total non-weight bearing on the affected extremity. These fractures can heal when given appropriate treatment.[27]

The role of arthrodesis has been infrequently mentioned in the orthopaedic literature for the treatment of diabetic neuroarthropathy. Arthrodesis has been frequently unsuccessful because of problems with nonunions and infections. Johnson stated that some of the poor results with arthrodesis are caused by superimposing surgical trauma on an actively destructive process.[27] Better results may be obtained by waiting until the extremity is no longer swollen or warm to the touch and roentgenograms indicate repair rather than resorption. He also emphasized the importance of resection of sclerotic bone to good bleeding bone, good apposition, prolonged immobilization, cancellous bone grafting and firm fixation. Harris and Brand felt arthrodesis could be successful in neuropathic joints secondary to leprosy if the operation is done early.[23] In late cases, arthrodesis is successful if the joints were excised back to normal appearing bone, if light compression was utilized and if the extremity is immobilized for a long period of time. Brooks and Saunders performed arthrodesis in the ankles of children with myelomeningocele and found a successful fusion was possible if the extremity was immobilized for a prolonged period of time.[3] In 1966 Heiple reported a successful triple arthrodesis in a diabetic patient with Charcot joints.[24] Connolly reported a successful midfoot osteotomy and tarsometatarsal joint arthrodesis in a diabetic with Charcot joints.[7] He felt that correction of the foot deformity solved his patient's problems with neuropathic ulceration and avoided the need for amputation. The goals of arthrodesis are to obtain a stable plantigrade foot in order to resolve progressive deformity and ulceration and prevent the possible need for amputation.

CONCLUSION

The diabetic with Charcot joint presents with a warm, swollen, deformed foot, usually associated with palpable pedal pulses, and pain may be present in up to one third of cases. The underlying bone and joint destruction is out of proportion to the patients level of pain secondary to a loss of pain perception. The pathogenesis of Charcot joints is controversial. It is probably a multifactorial problem with contributions from both an altered blood supply and the loss of protective sensation. The altered blood supply probably initially arises from sympathetic denervation. This increases the blood flow into the foot by removing the sympathetic vasoconstrictive effect on arterioles and allowing opening of arteriovenous shunts which are also controlled by sympathetic innervation. The bone is made osteopenic by the increased flow of blood through large dilated venules. This leads to the atrophic Charcot joint. The loss of proprioception from the peripheral neuropathy allows relaxation and hypotonia of supporting structures. The leads to occult trabecular fractures and ligamentous injuries. The inflammatory response to injury further increases the local blood supply and bone resorption. Pathologic fractures may occur in the weakened bone and lead to the rapid production of a Charcot joint. A vicious cycle is set up because the loss of protective sensation allows further trauma to mechanically and biologically deformed bones and joints. The process of attempted

repair forms bone callus and osteophytes leading to the hypertrophic Charcot joints. Further experimental and clinical investigations are needed to better delineate the pathogenic mechanisms and treatment modalities.

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HIGH HIP CENTER IN REVISION ARTHROPLASTY

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ABSTRACT

We conducted a retrospective review of seventeen patients with a revision of a failed cemented acetabulum with a minimum two year follow-up. The criterion for inclusion was a superior bony deficiency resulting in superior placement of the hip center in relation to a line drawn across the inferior margin of the tear drop. Minimum distance for inclusion was 30 mm superior to this line or three times the opposite normal hip. Surgical technique involved the use of a porous ingrowth prosthesis without the use of structural bone grafts for bony deficiency. There was no radiographic evidence of loosening or migration of the acetabular prosthesis in the thirteen patients in whom radiographs were obtained. There were no revisions, and all patients were independent ambulators, although one used a walker and nine used a cane (six part time, three full time).

INTRODUCTION

Inferior, medial and anterior placement of the acetabulum in hip arthroplasty is considered ideal.[3,9] This placement may not be possible when the dome of the acetabulum has been compromised by bone deficiency. There are many options for reconstruction in such cases including: 1) custom prosthesis, 2) oversized prosthesis (ingrowth or bipolar), 3) standard sized components using an anatomic position with a superior filler (cement vs. bone graft) or 4) high hip center (see figure 1).

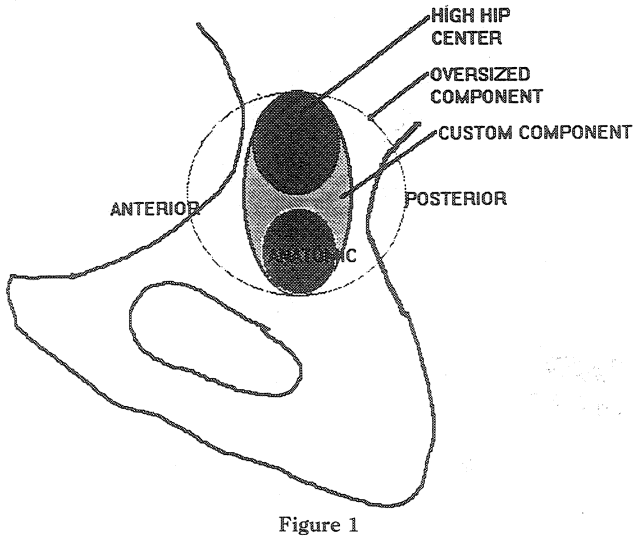

Figure 1

Many potential problems with these options exist. An oversized component may further compromise the anterior and/or posterior columns. Fillers may potentially fail. With bone graft fillers this may occur when the structural bone graft is not incorporated to the exact dimensions to which it was initially crafted. Late loosening of cemented acetabulae is a problem for cups placed with bulk cement filling, just as in primary total hips.

Recently, superior positioning of the acetabulum has been revisited as an alternative to the above techniques. It had been assumed that with superior placement of the cup there is a concomitant lateral position. However, Russotti and Harris[6] and Schutzer and Harris[7] have shown this is not the case.

Our paper differs from these two recent papers in that we used porous coated prostheses[6] and our definition of proximal displacement was more extreme.[7]

MATERIALS AND METHODS

We performed 139 total hip revisions for aseptic loosening from March 1986 to August 1989. Seventeen of these patients had significant superior acetabular bony deficiency, in which we elected to place the acetabulum in a superior position. We defined superior displacement of the hip center as greater than three cm from a line drawn through the most inferior portion of the teardrops or as three times the distance of the opposite side (if there was a normal opposite hip).

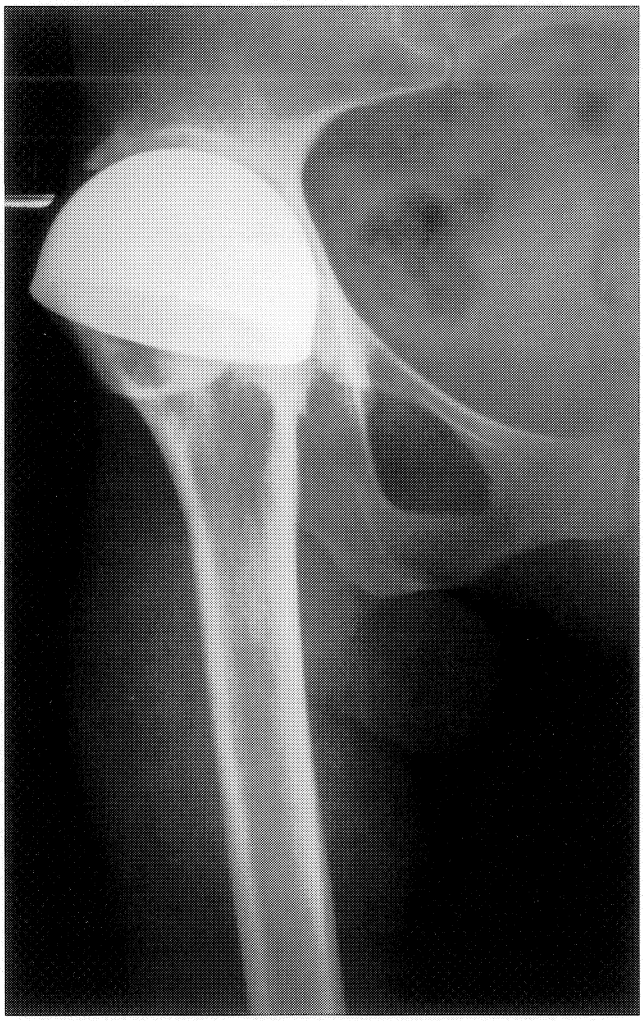

Figure 2A
Patient is a 51 y/o female whose past history is significant for septic right hip at age 1. She underwent a subtrochanteric osteotomy at age 8, followed by a cup arthroplasty at age 15. She did well until age 40 when she presented with progressive severe hip pain.

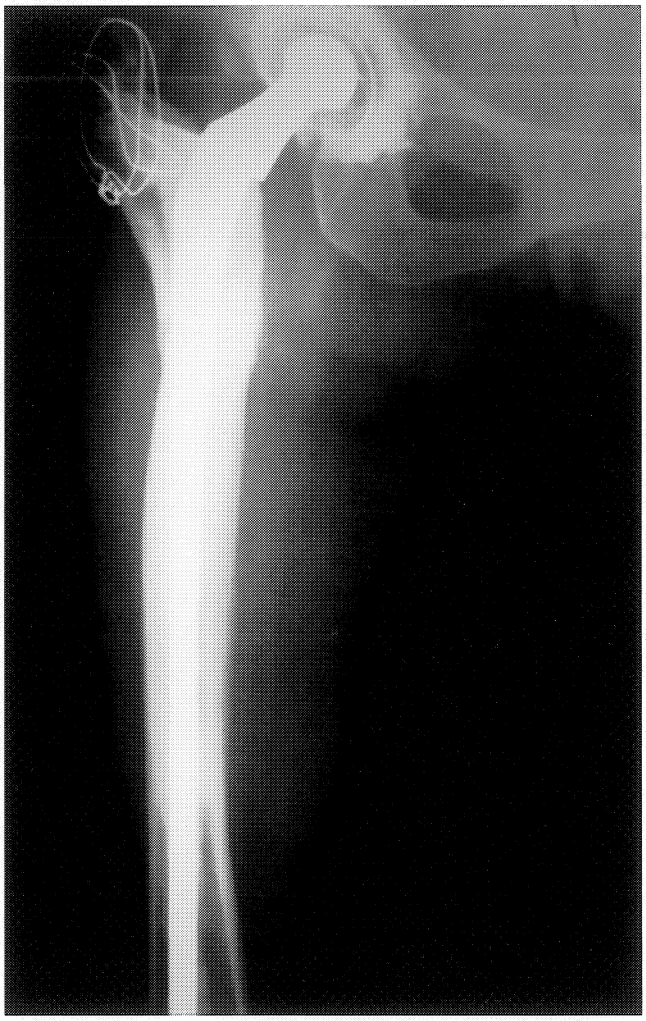

Figure 2B
At age 40 patient underwent total hip arthroplasty with osteotomy and placement of long stem prosthesis.

Surgery was performed by one of three surgeons, all via the transtrochanteric approach. A minimum follow-up of two years was obtained. Average length of follow-up was 36.4 months. Average age at time of revision surgery was 65 years.

Eight patients had two or more previous surgeries including four patients with three or more (see figure 2).

Two of the initial seventeen patients were deceased for reasons unrelated to surgery. Fifteen patients were available for follow-up; ten patients were able to return to our office for reevaluation and x-ray, three patients sent x-rays to our office and were contacted by telephone. One patient was unable to obtain x-rays, but was interviewed by phone. One patient was lost to follow-up.

All femoral components were revised with a longer femoral neck to regain abductor length, leg length and increase hip stability. With the exception of one patient in whom a long stem ingrowth femoral component was used, the femoral components were revised with a cemented prosthesis (ten standard stem length Iowa prostheses [140 mm], six with long stem lengths [200-250 mm]) . Only one of the sixteen cemented femoral components became loose and the one ingrowth component had early migration and instability, but has since stabilized.

RESULTS

Clinical

All fourteen patients who were interviewed were improved from their preoperative status and eleven had minimal or no pain at most recent follow-up. All were independent ambulators; four without ambulatory aids, nine used a cane (six part time users, three full time), and one required a walker.

Physical examination of ten patients revealed significant abductor weakness (less than 4+/5) in four patients.

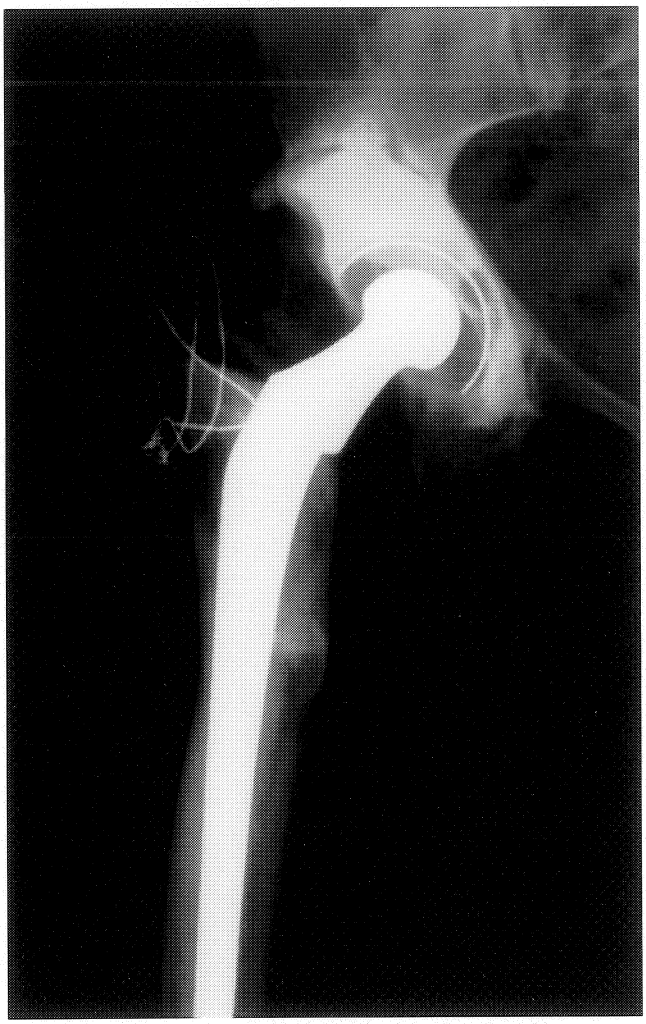

Figure 2C
She did well initially, but by age 47 had developed a loose acetabulum.

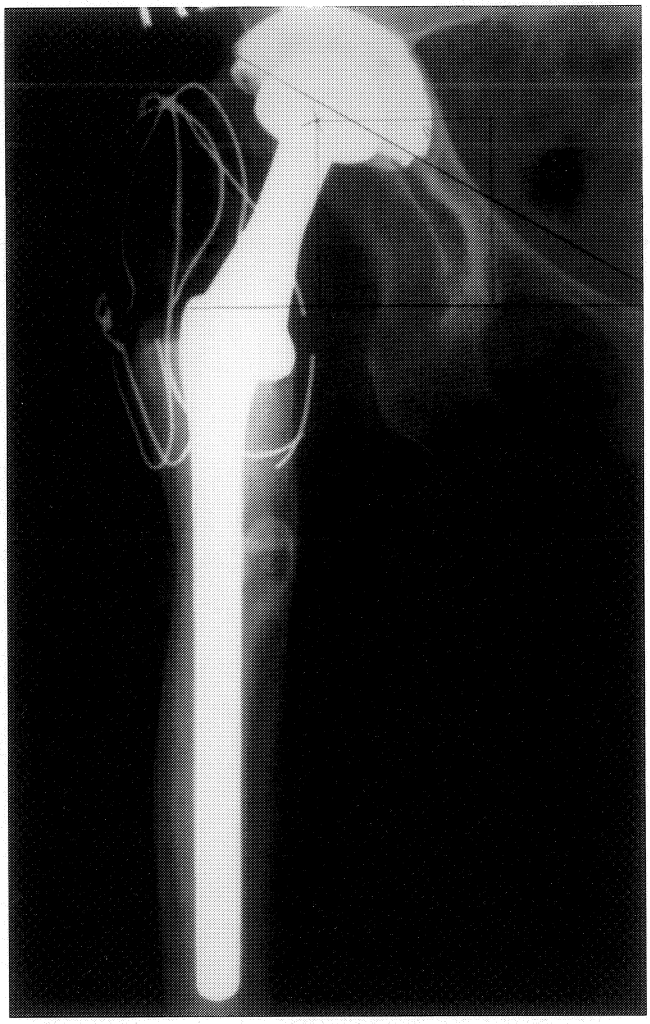

Figure 2D
Patient underwent revision to an ingrowth acetabulum with a high hip center. Femoral component was revised to a short stemmed prosthesis with a long neck length. At two year follow-up patient was ambulating without pain or assistive devices, with slight limp.

Abductor strength was normal in two patients, good in four, fair in three patients and poor in one patient. A moderate to severe limp was noted in seven of ten patients when ambulating without assistive devices.

Roentgenographic Measurements

Roentgenographic measurements were performed on fourteen of the seventeen hips in the study. Proximal location of the hip center averaged 36.9 mm (range 27.3-46.4 mm) above the inferior teardrop line for the involved hip. In the six patients with a normal opposite hip, the normal hip center averaged 12.8 mm superior to this line (range 6.4-16.0). Horizontal location, measured from the teardrop, averaged 31.6 mm (range 21.5-42.4 mm) in the involved hip and 33.3 mm (range 27.2-40.8 mm) for the normal hip (see Table).

TABLE

SUPERIOR CUP POSITION	
INVOLVED HIP: 14 revisions, 13 patients	
	AVERAGE 36.9 mm
	RANGE 27.3 - 46.4 mm
OPPOSITE NORMAL HIP: 6 patients	
	AVERAGE 12.8 mm
	RANGE 6.4 - 16.0 mm
LATERAL CUP POSITION	
INVOLVED HIP: 14 revisions, 13 patients	
	AVERAGE 31.6 mm
	RANGE 21.5 - 42.4 mm
OPPOSITE NORMAL HIP: 6 patients	
	AVERAGE 33.3 mm
	RANGE 27.2 - 40.8 mm

Trochanteric nonunion with greater than two centimeters of migration occurred in four patients. Ten patients had either bone or fibrous union. Significant abductor weakness was present in three of the four nonunions with migration.

There was no evidence of acetabular loosening or migration in the fourteen revisions (thirteen patients) evaluated radiographically. The one ingrowth femoral prosthesis in the study had early migration/instability, but eventually stabilized. Of the thirteen revisions in which the femoral component was cemented, one developed aseptic loosening.

DISCUSSION

The ideal cup position of inferior, medial and anterior should be achieved whenever possible. This is almost always possible in primary hip arthroplasty, including high congenital hip dislocations.[4]

Superior bone deficiency from a loose and migrating acetabular component has traditionally been reconstructed by one of four options: custom implant, oversized component, bulk cement and structural bone graft. Late cemented acetabular loosening in the primary setting[8] and early cemented acetabular loosening in revision arthroplasty[1] make bulk filling with cement an unattractive alternative. Structural allografts do not fair well long term, with a 47% loosening rate at eleven years.[5]

Recently, compromise of the ideal cup position has been revisted.[6,7] It has been assumed that superior placement of the cup is concomitant with a lateral position.[1,9] However studies[6,7] have shown this is not the case. Our findings are consistent with these reports.

Using a high hip center, Russotti and Harris found a 16% aseptic loosening rate of cemented acetabulae at an average of eleven years follow-up; only one of which required a revision. Using an ingrowth cup with an mean follow-up of 36 months, Schutzer and Harris found no adverse effects on fixation. Our study further supports these previous findings in a setting of more extreme proximal placement.

There is no significant difference in abductor strength when superior placement of the acetabulum is compensated for by increase in femoral component neck length.[2] When trochanteric union is achieved, our results supports this.

CONCLUSION

Although our study is relatively small with short term follow-up, we found that superior cup position did not result in concomitant lateral position. Our definition of superior placement was more extreme than previous studies. During the follow-up period we found no adverse effect associated with compromise of one parameter of ideal cup position.

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HEMATOGENOUS EPIPHYSEAL OSTEOMYELITIS: THREE CASE REPORTS AND LITERATURE REVIEW

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INTRODUCTION

Hematogenous osteomyelitis has classically been identified in the metaphyseal and diaphyseal regions of long bones in children. Epiphyseal involvement has been described, although it occurs rarely.[8,12,13,15,20,23] Green et al[8] suggested epiphyseal osteomyelitis presents with a nonspecific "subacute" clinical picture. Clinical and radiographic similarities to chondroblastoma, osteoid osteoma, aneurysmal bone cyst, clear cell chondrosarcoma, chondromyxoid fibroma and septic arthritis further complicate the diagnosis. We report on three cases of hematogenous epiphyseal osteomyelitis and review the literature illustrating the clinical presentation, the absence of sequelae and an approach to treatment.

METHODS

Between 1984 and 1988 three patients with hematogenous epiphyseal osteomyelitis of the distal femur were diagnosed and treated at the University of Iowa Hospitals and Clinics. Upon admission each patient had a history, physical examination, CBC and Sedimentation rate, and AP and lateral radiographs of the affected knee. All patients underwent surgical incision and drainage of the affected knee joint soon after admission. Intraoperative aerobic and anaerobic cultures were obtained as well as a cell count, protein and glucose analysis of the joint fluid. Each patient received parenteral antibiotic therapy for varying durations. Follow-up ranged from three to eight years.

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CASES

Case Report 1

A 23 month old female presented to a local hospital with a three week history of right knee pain, swelling, limp, fever and irritability. There was no prior history of trauma or infection. She was hospitalized by her local physician, treated with aspirin, and observed. Radiographs were negative. Upon presentation to our facility, the patient's right knee was warm, rested in 15 degrees of flexion, displayed an effusion, but had no erythema. There was marked tenderness over the lateral femoral condyle and joint motion was painful. Her rectal temperature was 38 degrees Centigrade. Her WBC was 9,000 with a left shift, hematocrit 33%, and ESR 55 mm/hr. Radiographs at this time showed a lytic lesion of the lateral femoral epiphysis (Fig. 1a and 1b). On the day of admission a right knee arthrotomy was performed and pus was drained. Granulation tissue was noted perforating the cortex of the lateral femoral epiphysis. The lateral femoral condyle was curetted carefully, avoiding the epiphyseal plate. The wound was closed over a Penrose drain. Joint fluid analysis showed 55,000 WBC per high power field with 95% PMN's and 5% lymphocytes. Glucose and protein were 20 mg/dl and 5.7 mg/dl. No organisms were identified on gram stain. Bacterial and fungal cultures were negative. Skin tests for TB, histoplasmosis, and coccidiomycosis were also negative. Post-operatively, parenteral ampicillin and methicillin were administered. The patient became afebrile 48 hours after surgery. She was discharged on the seventh post-operative day on oral ampicillin and oxicillin and was placed in a posterior splint. The splint was discontinued at three weeks and full activities were resumed at six weeks. The patient received six weeks of combined parenteral and oral antibiotic therapy. At six

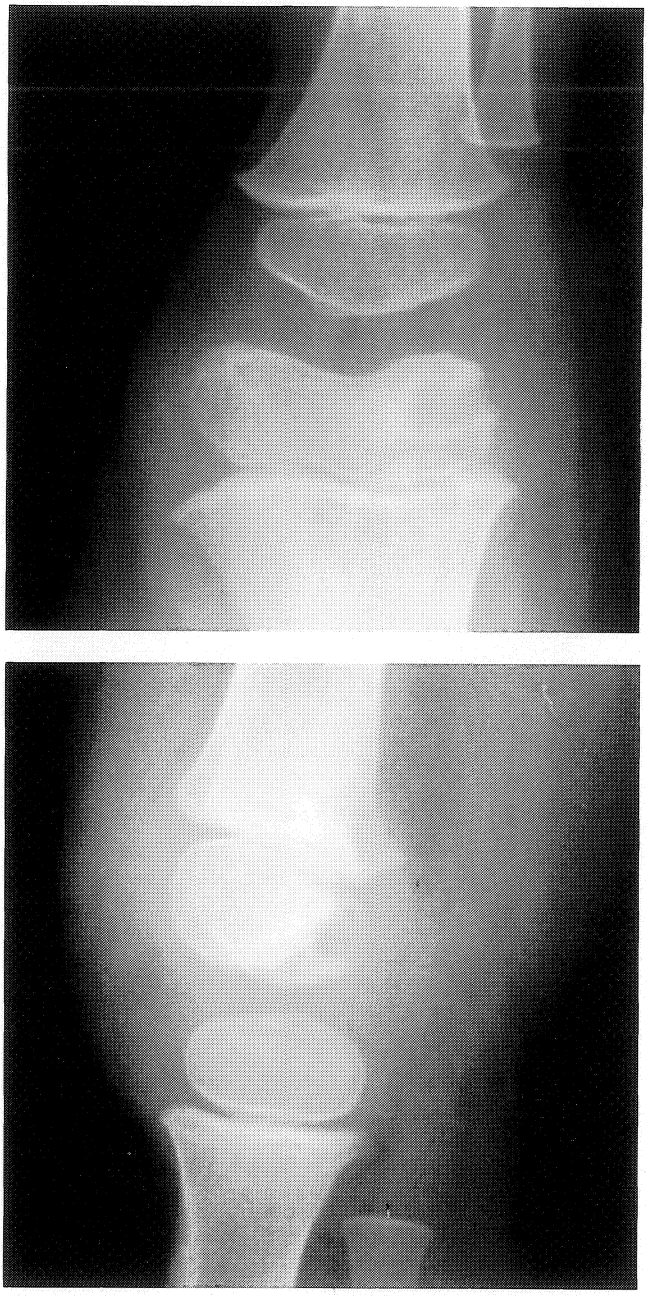

Figure 1a and 1b
Right knee AP and Lateral of a 23 month old female (case #1).

year follow-up no complications were noted and she had normal radiographs (Fig. 1c and 1d).

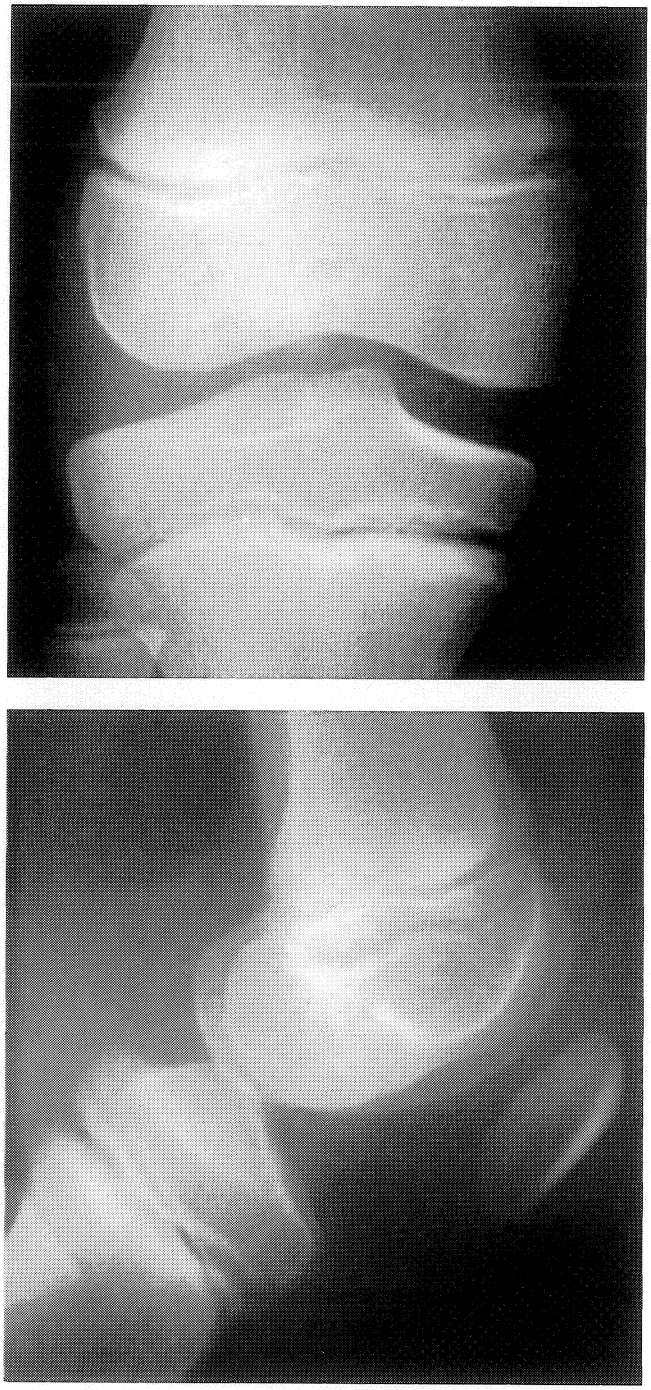

Figure 1c and 1d
Right knee AP and Lateral of patient in case #1 at 6 yr follow-up.

Case Report 2

A 4 year 9 month old male presented with a six week history of right knee pain, redness, and swelling in the absence of antecedent trauma or infection. Three days after the onset of symptoms, the boy developed a fever and was unable to bear weight on the right lower extremity. Radiographs taken by the local physician showed no abnormalities. Oral penicillin was administered and the fever subsided in a week, however, the pain, swelling, and loss of motion persisted. Six weeks after the onset of these symptoms, the patient was referred to our center. On admission, physical examination of the right knee was held in 45 degrees of flexion. There was warmth and swelling along the medial aspect of the knee as well as marked muscle atrophy. Significant tenderness was noted

over the medial femoral condyle. Pain was elicited with passive range of motion. Radiographs showed a destructive area (2 cm x 2 cm) in the medial distal femoral epiphysis. The epiphyseal plate and metaphysis were uninvolved. The WBC was 10,200 with a left shift and the hematocrit was 37.5%. The right knee was aspirated demonstrating pus. On the day following admission a right knee arthrotomy was performed and more pus was drained. Granulation tissue was observed overlying the medial femoral condyle. Upon penetrating this granulation tissue a 3 cm x 2 cm defect filled with yellow pus under pressure was discovered. The cavity was gently curetted without disturbing the epiphyseal plate and the wound closed over a Penrose drain. Cultures grew a penicillin-sensitive staphylococcus aureus. Parenteral penicillin was administered for seven days, and the patient was discharged in a right leg cylinder cast and given four weeks of oral penicillin and erythromycin. At four years radiographs showed arrested osteomyelitis of the distal femoral epiphysis. The patient was followed for eight years showing a full range of motion and no angular deformity. A 1/2 inch right leg length deficiency was present.

Case Report 3

An 11 year and 4 month old boy presented to our center with a two year history of right knee pain unresponsive to aspirin. Approximately six weeks prior to presentation the pain was accompanied by swelling of the knee. There was no history of trauma or prior infection. The mother did mention a history of intermittent limping since age three years and there was a history of a heart murmur. Physical exam showed mild swelling of the right knee without erythema. Range of motion lacked ten degrees of full flexion. Although no ligamentous laxity was noted, valgus stressing with external rotation was painful. The patient was afebrile. Radiographs showed a 1 cm x 1 cm lytic lesion in the lateral femoral epiphysis with sclerotic, geographic margins consistent with an indolent infection. A CT scan localized the lesion to the posterior medial portion of the lateral femoral condyle with extension through the posterior cortex into the joint. The WBC was 9,700, ESR was 20 mm/hr and hematocrit was 42%. On the day of admission the patient underwent a right knee incision, drainage, and debridement with joint fluid culture and biopsy of the lesion. The wound was closed over a Penrose drain. Cultures grew penicillinase-resistant staphylococcus aureus and the biopsy was consistent with acute and chronic osteomyelitis. Post-operatively the patient received intravenous nafcillin for three weeks followed by three weeks of clindamycin (changed due to drug fever). The boy was discharged at six weeks with a non-tender right knee and a painless range of motion from 0-100 degrees. At three years the patient had no complaints, displayed full motion and had resumed full activity. Radiographs showed a surgical defect with no active osteomyelitis.

DISCUSSION

Acute hematogenous epiphyseal osteomyelitis is uncommon. A review of the literature revealed 31 reported cases excluding our three cases. Between 1951 and 1960 Gilmour[6] identified four cases of distal femoral epiphyseal osteomyelitis from 328 cases of acute hematogenous osteomyelitis. The average age of these four patients was 5.5 years and they were treated successfully with surgery and antibiotic therapy. In 1967 Robertson[19] reported three cases of acute hematogenous osteomyelitis originating in the epiphysis of the distal posterior tibia, the distal fibula and the distal femur. Ages of these patients were four, eight, and nine years respectively. These children were successfully treated with curettage and antibiotic therapy. In 1969, King and Mayo[13] reported 67 patients with a "subacute" hematogenous osteomyelitis; two were located in the epiphysis. Both were originally misdiagnosed. Green et al[8] identified eight children between 1972-1978 with "primarily subacute" epiphyseal osteomyelitis. All presented with pain and a limp, two were located in the proximal tibia and six were located in the distal femur. In 1984 Lindenbaum and Alexander[15] described 15 cases of "subacute osteomyelitis" originally diagnosed as tumors; two were epiphyseal in location. In 1985 Rosembaum and Blumhagen[20] reported nine cases of epiphyseal osteomyelitis, all presenting with pain and elevated ESR's. Seven occurred in the distal femoral epiphysis, two in the proximal tibial epiphysis and one in the proximal humeral epiphysis. Sorenson[2] in 1988 described three cases, two involving the proximal tibial epiphysis, one involving the distal femoral epiphysis. The addition of our three cases brings the number of reported cases of primary epiphyseal osteomyelitis to 34.

The presentation of patients with epiphyseal osteomyelitis bears many similarities to that of the classic metaphyseal focus. Our three patients ranged in age from 23 months to 11 years and 4 months. All patients presented with pain, a limp, limited range of motion and an effusion. Two of the three had fevers. One patient had symptoms for two years. No patient in our series had leukocytosis. Each of the three patients displayed a well demarcated lytic lesion in the distal femoral epiphysis. However, two of three initially presented with negative radiographs. All patients responded promptly to surgical drainage and parentesal antibiotic therapy. Pus was found in all three knees.

The route of bacterial seeding in childhood epiphyseal osteomyelitis is not well understood. Vascular studies have shown that the growth plate cartilage is first apparent by age 18 months representing a mechanical barrier to the direct spread of infection from a metaphyseal focus.[17,25,27]

However, dynamic blood flow studies of immature bone have shown significant epiphyseal blood supply. Furthermore, quantitative hematogenous bacterial injection studies have demonstrated high bacterial counts in the epiphysis ten minutes following injection. This initial peak bacterial count was followed by a secondary peak of bacterial activity 24 hours later.[14,16] Trueta has shown a direct epiphyseal arterial supply which extends transversely through the epiphysis.[25,26] In our three cases no communication was found across the epiphyseal plate into the metaphysis. Likewise, Green et al[8] found no metaphyseal communication in their series of eight patients. This supports the premise that the epiphysis can be a primary location for hematogenous osteomyelitis.

A survey of other series and case reports of hematogenous epiphyseal osteomyelitis reveals the knee as the most common location.[6,8,12,13,15,19, 20,23] In fact, 29 of the 34 reported cases involved the knee, twenty-four involving the distal femoral epiphysis (Fig. 2). Other reported sites include the distal tibia (2), proximal femur (1), and distal fibula (1).

The differential diagnosis of hematogenous epiphyseal osteomyelitis should include chondroblastoma, osteoid osteoma, aneurysmal bone cyst, clear cell chondrosarcoma and chondromyxoid fibroma. Each of these lesions may occur in childhood and can involve the epiphysis of long bones.[1,2,3,4,22,24] Chondroblastoma is frequently a painful lesion found in the epiphyses about the knee. It is lytic with a sclerotic rim and generally presents during the latter years of growth.[4] The presence of punctate calcification helps distinguish chondroblastoma from osteomyelitis. Osteoid osteoma is a painful lesion which rarely exceeds one cm in diameter and more closely approximates the size of osteomyelitic lesions of the epiphysis.[4] Radiographically, osteoid osteoma is usually oval with a central radiolucent nidus surrounded by reactive bone.[1,2,3,4,24] Acute osteomyelitic lesions, in contrast, are usually round and have a smaller amount of reactive bone formation.[5,6,7,11,12,13,15,20,23] Although osteoid osteoma is more commonly found in the diaphysis or metaphysis of long bones, seven cases have been reported in the epiphysis with four of seven involving the distal femoral epiphysis.[1,2,3,4,22,24] Aneurysmal bone cysts commonly involve the knee and may extend from the metaphysis into the epiphysis.[4] Its expanded delicate shell of bone usually distinguishes it from osteomyelitis.[4] Clear cell chondrosarcoma is found in the epiphysis of long bones and is very similar radiographically to chondroblastoma.[4] It is most often found in the proximal femur and proximal humerus, sites uncommon for hematogenous epiphyseal osteomyelitis.[4,5,6,7,11,12,13,15,20,23] Chondromyxoidfibroma typically involves the tibia but rarely the epiphysis, and has distinct cortical thinning.[4] Lindenbaum and Alexander[15] reported 15 cases of subacute osteomyelitis in which the original diagnosis was bone tumor. Two of the 15 infections were epiphyseal in origin and were diagnosed as chondroblastoma and "tumor".

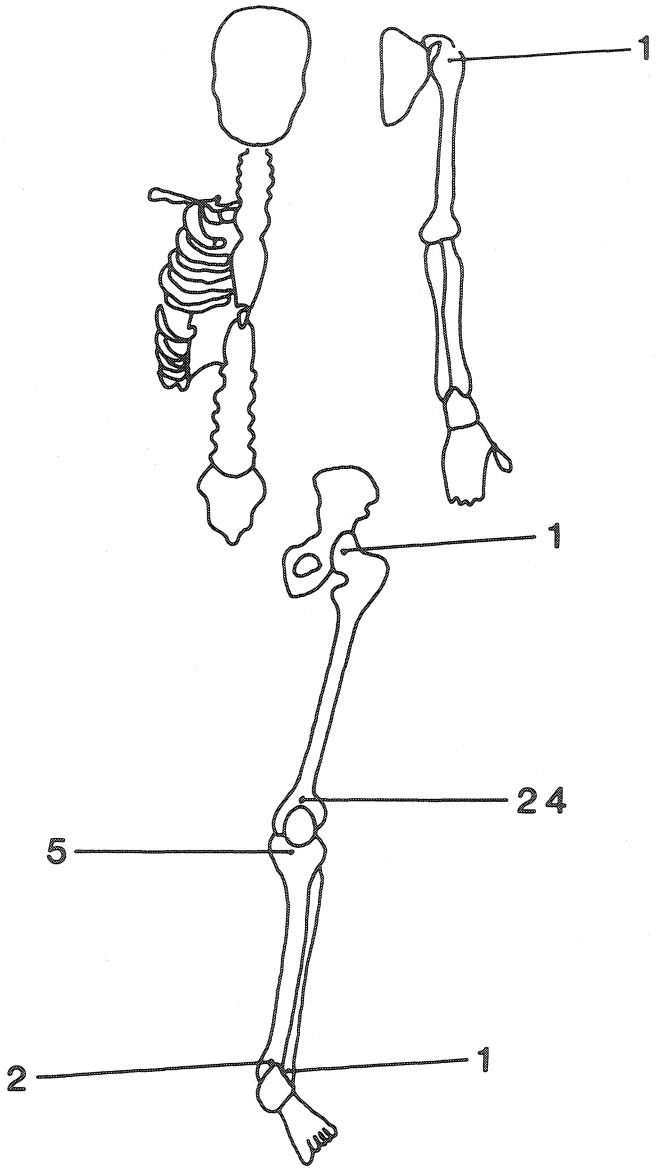

Figure 2
Skeletal distribution of the 34 reported cases of Hematogenous epiphyseal osteomyelitis.

At follow-up of six years, eight years, and three years the only sequelae noted in our three cases was 1/2 leg-length deficiency in one patient. Green et al[8] and Sorenson et al[23] have also provided follow-up on eight patients and three patients respectively. They reported no growth disturbance or associated joint sequelae. The initial lytic lesion seen in hematogenous epiphyseal osteomyelitis can be expected to heal in time. (Fig. 1a-1d).

In conclusion, hematogenous epiphyseal osteomyelitis is a distinct clinical entity which may present with acute or subacute symptoms. The clinical presentation may be identical to acute metaphyseal osteomyelitis or septic arthritis displaying pain, limp and an effusion. This condition may originally be misdiagnosed as chondroblastoma, osteoid osteoma or other bone tumors. The most common location of hematogenous epiphyseal osteomyelitis is the knee. We recommended aggressive surgical debridement followed by appropriate antibiotic therapy. With appropriate treatment, significant morbidity does not occur.

ACKNOWLEDGEMENT

Special thanks to Dr. George El-Khoury.

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NON-OPERATIVE TREATMENT OF A COMPLETELY DISPLACED AND SHORTENED PROXIMAL HUMERAL METAPHYSIS FRACTURE IN A CHILD

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Fractures of the proximal humerus represent a small percentage of pediatric long bone fractures.[4,8,10] Epiphyseal separations are more common than metaphyseal fractures.[5,6] Most reported cases of metaphyseal fractures have limited follow-up. A moderate degree of displacement and angulation may be accepted. However, there is controversy regarding the treatment of completely displaced proximal humeral metaphyseal fractures. This case is an example of a severely displaced proximal humeral metaphysis fracture treated nonoperatively, with remarkable remodeling.

CASE REPORT

An 11 year-old female was involved in a high speed motor vehicle accident and sustained multiple long bone fractures, including a closed right proximal humeral metaphyseal fracture without neurovascular compromise, a contralateral open humeral shaft fracture and a closed femoral shaft fracture. Initial radiographs of the right proximal humerus fracture (Fig. 1) demonstrated 100% lateral and posterior displacement, and proximal migration of the shaft with 45° angulation. Initial attempts at closed reduction were unsuccessful. Six days later closed manipulation was again attempted while the patient was under general anesthesia for a procedure in another extremity. Despite several attempts at closed reduction under fluoroscopy, contact between the shaft and proximal metaphysis could not be maintained. Biceps tendon interposition[11] was felt to be a possible cause. The clinically apparent deformity was accepted rather than proceeding to open reduction.

Radiographs two weeks after injury (Fig. 2) demonstrated medial callus formation with continued severe displacement. The posterior subdeltoid position of the shaft was readily apparent on examination. After six weeks, the shoulder immobilizer was discontinued and she progressed with active and passive range of motion. Since then, serial roentgenograms have shown progressive remodeling of the proximal humerus (Fig. 2-6).

At two year follow-up, the patient had .8 cm shortening compared with the contralateral upper extremity, which had a humeral shaft fracture healed in a bayonet position. Shoulder motions were symmetric with forward flexion and elevation to 180°, external rotation to 90° at 0° abduction, and internal rotation to permit the long finger to reach T7 (Fig. 7). Elbow range of motion was 0° extension to 145° flexion bilaterally. The patient noted no pain in either upper extremity and denied any sense of limb length discrepancy. She now plays school sports without restrictions.

DISCUSSION

Proximal humerus fractures occurred between the ages of six and fourteen in sixty-seven of eighty-seven pediatric patients in Kohler and Trillaud's series.[5] It has been

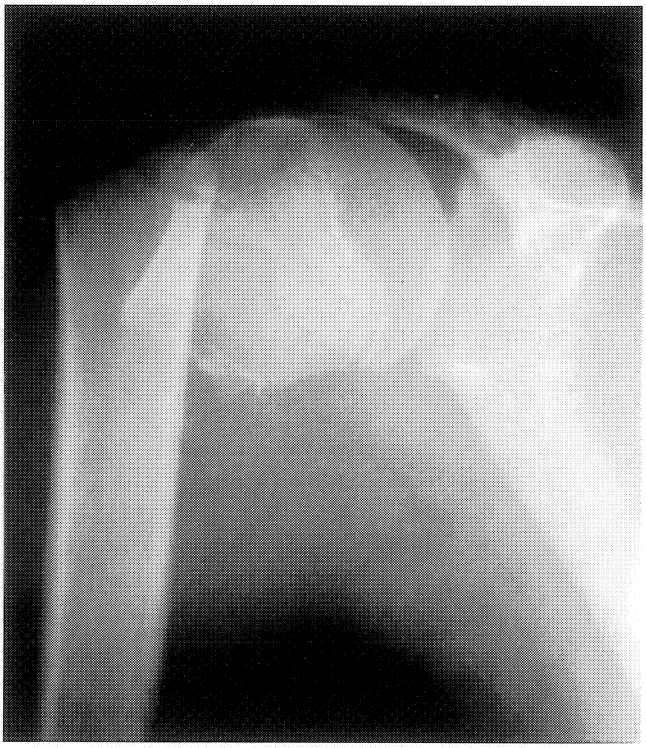

Fig. 1
Initial injury films demonstrating complete displacement of the proximal humerus fracture.

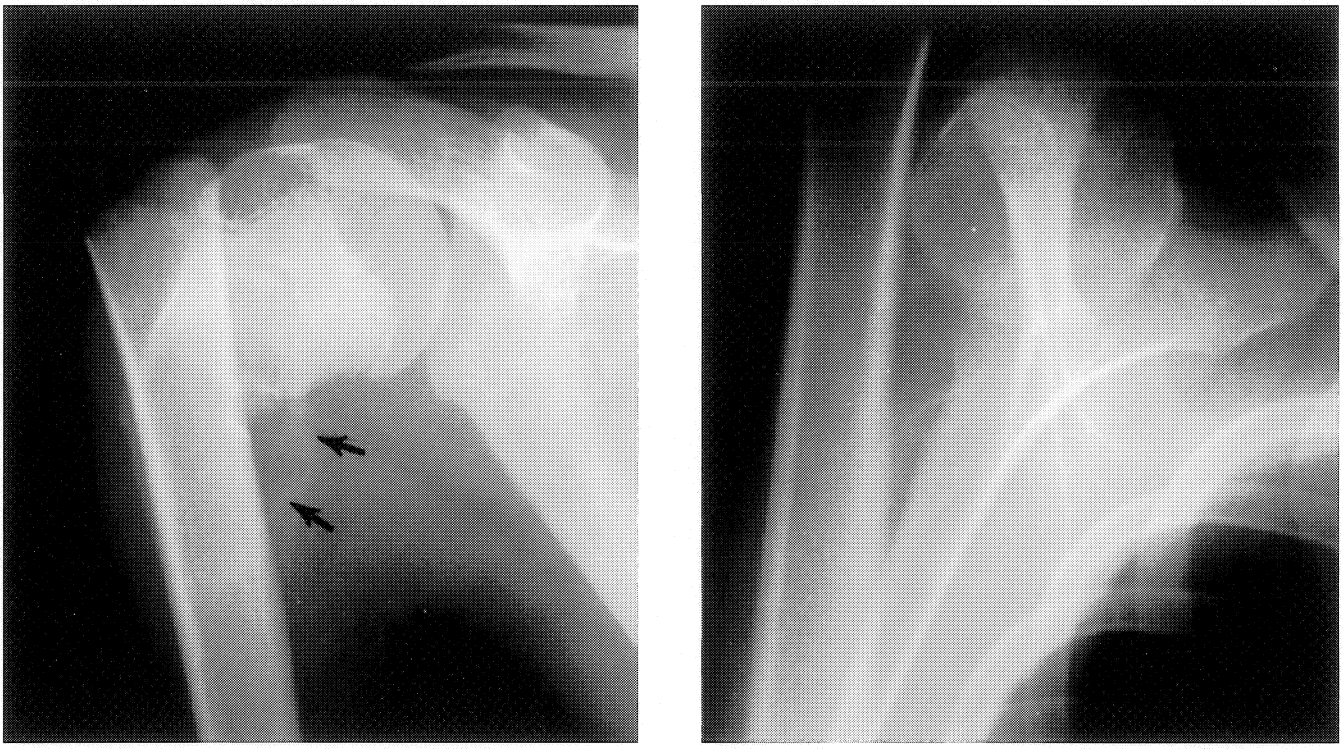

Fig. 2A-B
Already callus formation is seen two weeks after injury with the fracture fragments essentially unchanged in position from initial presentation. Black arrows indicate a calcific density which may be the intact periosteal sleeve.

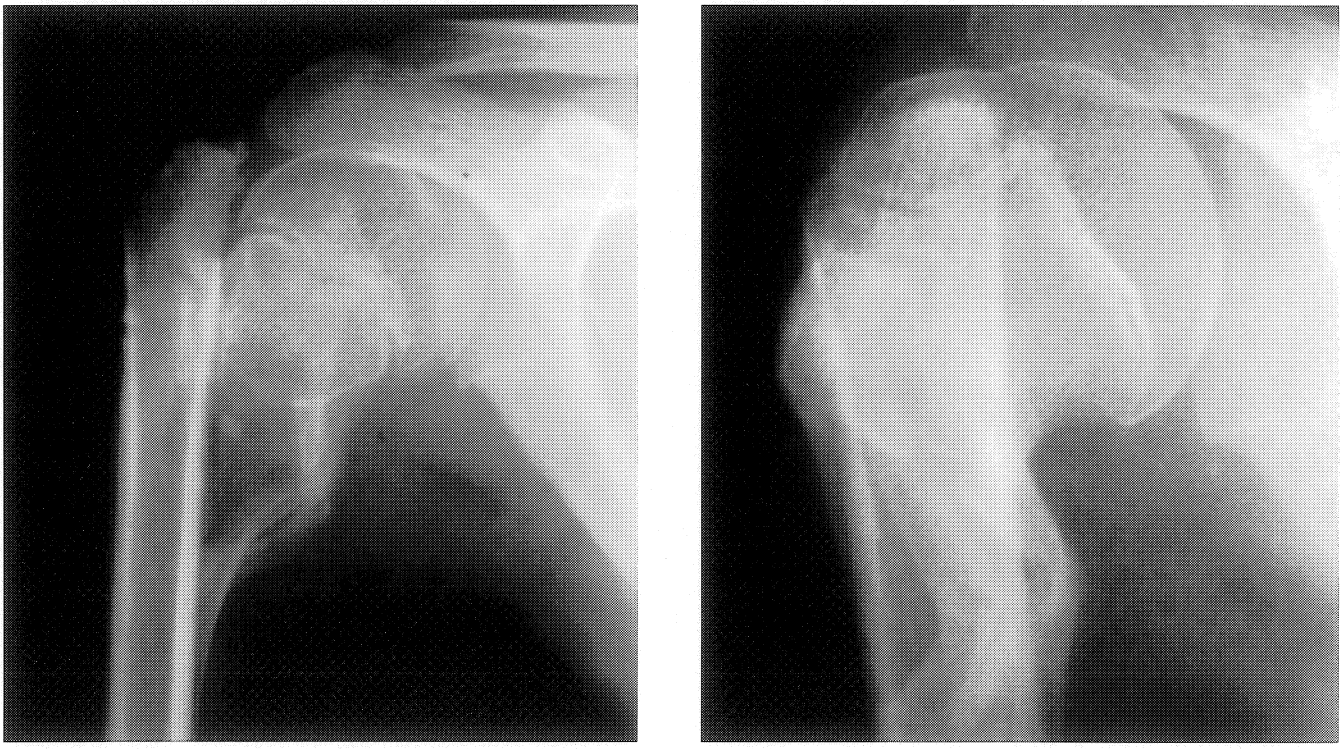

Fig. 3A-B
Six weeks post injury. By now abundant callus formation is seen.

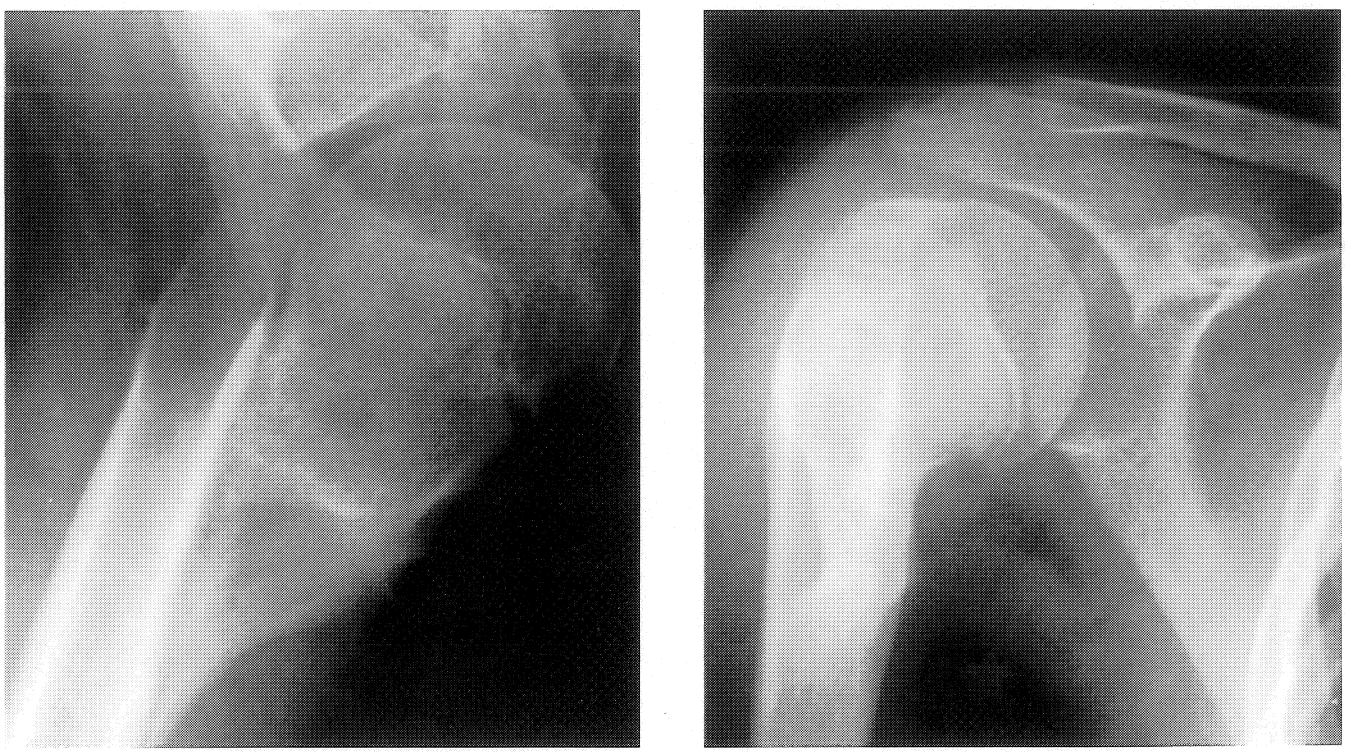

Fig. 4A-B
Thirteen weeks post injury. The proximal portion of the distal fragment has been resorbed and the humeral head is clearly becoming incorporated.

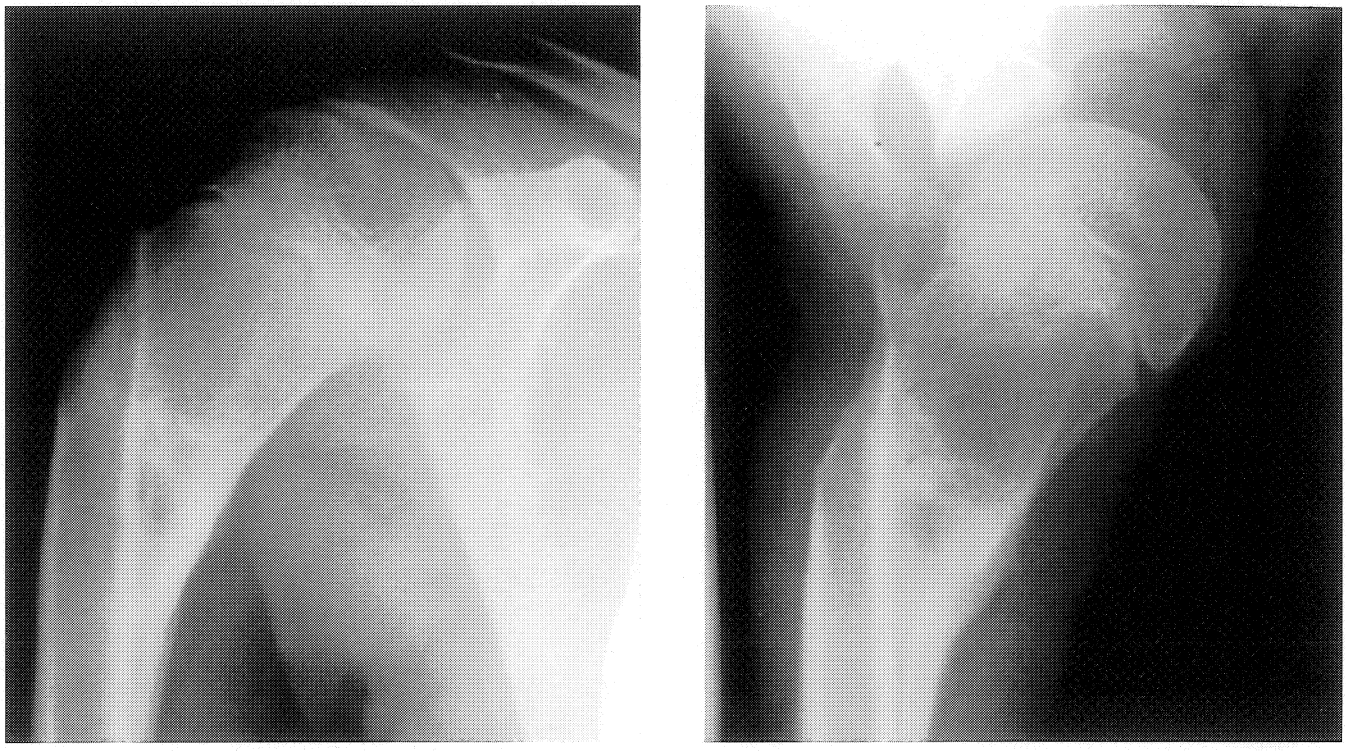

Fig. 5A-B
Seven months post injury. The posteromedial metaphyseal cortex is progressively resorbed as the humeral head is further incorporated.

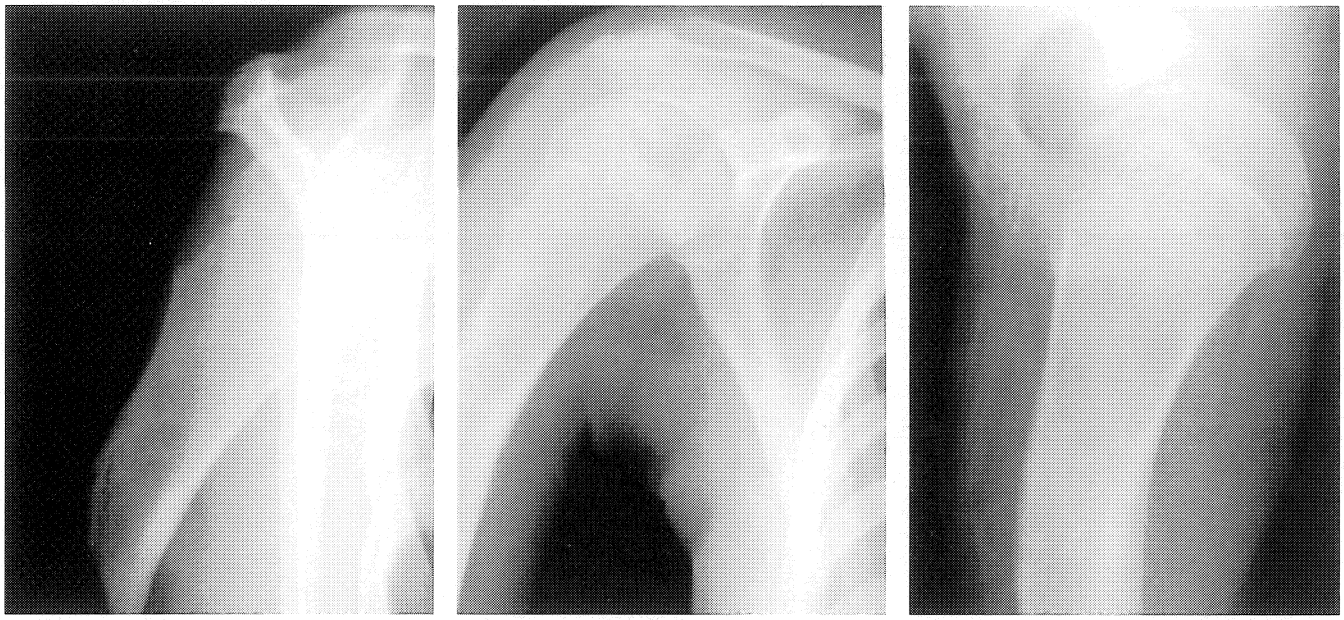

Fig. 6A-C
Two years post injury. The proximal humerus has reformed. A mild angulatory deformity remains.

Fig. 7
Photographs two years after injury demonstrate full range of motion in the injured right upper extremity.

suggested that this reflects accelerated growth and relative bone weakness at this age.[2,4,7] The metaphyseal subset has a predilection for pre-teen aged patients, with a median age of ten to twelve.[5,6]

The mechanism of epiphyseal separation is controversial,[2,4,8] but metaphyseal fractures generally result from direct trauma.[4] As in this case, the fracture line is usually transverse or slightly oblique.[5] In metaphyseal fractures, the presence of a thick periosteal sleeve and dense overlying muscle usually prevents significant displacement and angulation.[4] In epiphyseal separations, the anterior periosteum is weaker and anterior displacement is not infrequently noted.[3] In this particular case the displacement was complete with posterior and proximal migration of the shaft.

The proximal humeral physis provides up to 80% of the length of the humerus.[2] This, coupled with the likelihood that a periosteal sleeve (as shown in Fig. 2) remains intact between the fragments, endows the proximal humerus with extraordinary remodeling capabilities. This case demonstrates the remodeling potential of the proximal humerus more than any previously published case. This 11 year-old female rapidly remodeled an initial deformity that included 100% posterior displacement, 45° angulation, and 4 cm of shortening.

As her only residual from the fracture, this patient has a 0.8 cm of asymptomatic shortening measured from the acromion to radial styloid. This information is confounded, however, by her contralateral humeral shaft fracture which was in a bayonet position. According to Kohler and Trillaud,[5] shortening is a problem and is related to improper reduction. However, review of the literature revealed only one case of greater than 3.0 cm shortening after a proximal humerus fracture. This was in a twelve year-old boy with a Neer IV physeal injury.[8] Shortening has not been specifically documented for nonphyseal injuries. This case demonstrates significant potential for growth acceleration, even when initial shortening is accepted. Therefore, treatment should not be based on potential shortening alone.

The accepted treatment for proximal humeral fractures is based on a large number of reports with limited follow-up data. There is general consensus that most of these fractures can be treated non-operatively. Reported operative indications include skin tenting over the fragments,[1] open fractures, associated neurovascular compromise, and displacement of intra-articular fractures.[2,3] In addition, many authors support irreducibility of translations, angulation, or proximal migration as an indication for surgery.[2,9,12] Kohler and Trillad[5] support open reduction for "irreducible fractures, commonly associated with biceps tendon-periosteum interposition". Publications by Nilsson and Svartholm[9] and by Wahl[12] recommend open reduction for those patients with significant displacement which cannot be reduced closed. However, the rapid complete remodeling of this remarkably severe deformity makes it difficult to justify operative management of any proximal humeral metaphysis fracture, based on irreducibility alone when further skeletal growth is anticipated.

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HAND METASTASIS FROM RENAL CARCINOMA

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INTRODUCTION

Metastatic tumors of the hand are an uncommon but well-recognized entity. The majority occur in the distal phalanges. Soft tissue involvement, if present, typically results from tumor erosion through cortical bone or direct seeding. Isolated soft tissue metastases with secondary invasion of the adjacent bone have also been described.[1,2,3] A metastatis to the soft tissues of a digit without bony involvement is extremely rare and can easily be mistaken for other benign appearing lesions. We report a patient with metastatic renal adenocarcinoma isolated to the soft tissues overlying the distal phalanx. We also review reported tumors metastatic to the soft tissues of the hand.

CASE REPORT

A 77 year-old man first presented to his local physician with a chief complaint of intermittent fever. Extensive evaluation, including an intravenous pyelogram and chest radiograph, was inconclusive. The patient returned three months later with recurrence of his symptoms. A repeat chest radiograph demonstrated multiple nodules in the lung base consistent with metastatic disease. Computed tomography of the abdomen revealed enlarged, irregular kidneys and adrenal glands. A bone scan demonstrated two areas of increased uptake in the left anterior sixth and seventh ribs. Metastatic disease involving the lungs, kidneys, adrenals and musculoskeletal system was assumed, but the patient did not desire further evaluation or treatment.

Six months later he was referred to the hand service for treatment of a presumed pyogenic granuloma of the left long finger. Examination revealed a painless, firm, superficial, granulomatous nodule measuring 1.2 x 1.0 x 1.0 cm. A roentgenogram of the left hand demonstrated soft tissue swelling surrounding the distal phalanx of the long finger without bone destruction or periosteal reaction. An incisional biopsy caused brisk bleeding from the lesion that was stopped by prolonged firm pressure. Histologic examination established the presence of non-small cell carcinoma that was most likely metastatic renal adenocarcinoma. The patient underwent a PIP level disarticulation and further histopathologic examination of the finger confirmed both this diagnosis and lack of osseous involvement.

The patient died four months later, less than a year from the time he originally sought care. Autopsy established the left kidney as the primary site, with widespread metastases involving the liver, pancreas, spleen and soft tissues overlying the distal phalanx of the left ring finger and right fifth metatarsal.

PATHOLOGIC FINDINGS

The incisional biopsy demonstrated a highly vascular, large cell neoplasm occupying the entire dermis and attenuating the epidermis except for an area of ulceration (Figure 1). The neoplastic cells formed small clusters separated by delicate capillary vascular channels. Their abundant eosinophilic cytoplasm contained multiple vacuoles. The nuclei ranged from small, nearly round nuclei with small obscure nucleoli to large irregular vesicular nuclei with folded nuclear membranes often containing

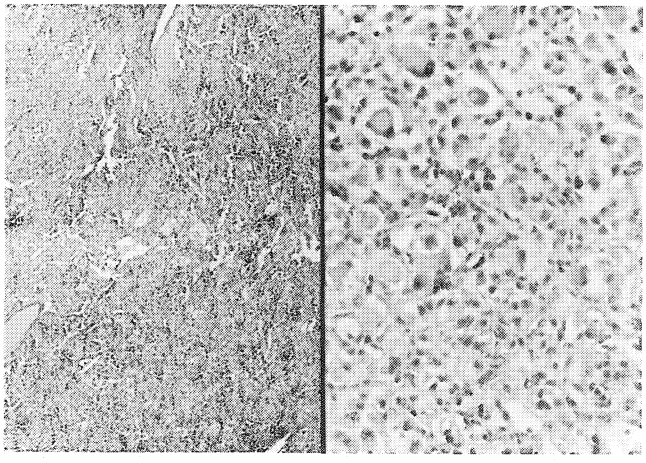

Figure 1
Highly vascular dermal tumor (1A, hematoxylin and eosin, original magnification 10X) with pleomorphic large cells with abundant cytoplasm, and occasional tumor giant cells, (1B, hematoxylin and eosin, original magnification 50X).

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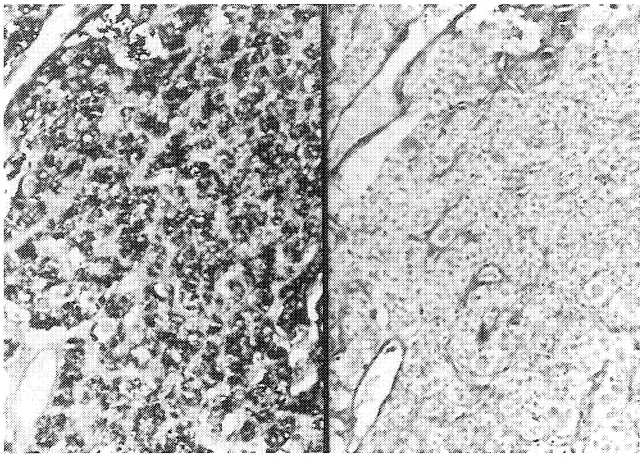

Figure 2
Copious quantity of periodic acid-Schiff positive granules (2A), extinguished by pre-treatment with diastase (2B), indicating glycogen (original magnification 25X).

large prominent nucleoli. Occasional multinucleate tumor giant cells were noted. Mucin was not demonstrated by the periodic acid-Schiff reaction with and without diastase (Figure 2). Stains for melanin were negative as was immunoperoxidase for Factor 8. The histology was felt to be most consistent with a metastatic renal carcinoma. Electron microscopy was performed on a portion of the paraffin embedded tissue but was technically inadequate for cytologic ultrastructural detail.

The amputated finger contained a 1.2 x 1.0 x 1.0 cm soft, tan-white mass at the tip which extended beneath the nail and abutted the bony phalanx without bone involvement. The histology was similar to the biopsy. Ultrastructural examination disclosed abundant lipid, glycogen and organelles, but other features suggestive of renal carcinoma were not identified.

DISCUSSION

Tumors metastatic to the hand are uncommon and represent less than 0.1% of all metastases.[2] The most frequent primary site is the lung which accounts for 47% of cases.[4] The kidney is responsible for 11% of hand metastases and 17% of pedal metastases.[4] The distal phalanges are the most frequent site of metastatic deposition, although involvement in all bones of the hand has been described.[5]

Metastases typically involve the osseous structures of the hand, although soft tissue lesions can occur as a result of tumor erosion or direct seeding. Isolated soft tissue metastates without associated bony involvement is extremely uncommon. The first reported case of metastasis to the soft tissues of the hand was described in 1907 and involved a kidney neoplasm overlying a phalanx with subsequent involvement of bone.[5] In 1978, Wu and Guise described three patients with "pure" soft tissue metastases from the breast, colon, and kidney.[6] The patient with renal cell carcinoma had a metastasis to the dorsum of the hand. A review of the literature by Kerin in 1983 revealed 156 cases of metastatic hand tumors, only 12 of which demonstrated soft tissue involvement (13%).[2] However, details of the tumor type and sites of involvement were not reported. A recent report by Amadio and Lombardi provided four additional cases of which two had renal primaries, both metastasizing to the palm of the hand.[7] To the best of our knowledge, renal adenocarcinoma metastatic to the subcutaneous tissue of a digit, without osseous involvement, has not been previously reported.

The mechanism by which tumors metastasize to the hand remains obscure. A variety of factors including trauma, tissue thermal differences, hormonal influence, hemodynamics and host immune responses have all been implicated.[4,9] Hematogenous dissemination of tumor emboli is the presumed mechanism in the majority of cases. It has been shown experimentally that the number of metastases occurring in a particular organ is proportional to the blood flow.[4] According to Mulvey, this would explain why metastases to the hand occur most commonly in the distal phalanx, where blood flow is greatest.[8]

Metastases to the soft tissues of the hand tend to be asymptomatic and can masquerade as such lesions as a lipoma, fibroma, pyogenic granuloma or pulp space infection.[6] Our patient was referred for treatment of a presumed pyogenic granuloma.

Radiographic findings are often nonspecific in isolated soft tissue metastasis and a periosteal reaction is uncommon.[10] Pain, swelling, tenderness and loss of function may be present. Thus, in those cases that do not demonstrate other features suggestive of malignancy, diagnosis requires a high index of suspicion and histologic examination of the lesion itself. Needle and excisional biopsy are the most commonly used techniques. However, if metastatic renal carcinoma is suspected, biopsy should be performed carefully because of the increased vascularity of these tumors and their propensity for significant blood loss and difficult hemostasis.

The majority of patients die within six months of diagnosis of hand metastases, although prolonged disease-free intervals have been reported in several patients with excised isolated lesions. Consequently, the goal of treatment is often palliation. Treatment options include surgical excision and/or local radiation therapy. If the lesion is located distally, amputation through the joint proximal to the level of involvement is recommended. When the metastasis is more proximal, local excision, alone or in combination with radiation therapy, is usually effective. Local radiation therapy can also be helpful in alleviating pain in those patients who are chronically debilitated or in those who do not desire surgical intervention.

CONCLUSION

Isolated metastatic lesions in the soft tissues of the hand are rare. They are believed to result from hematogenous seeding. The most common source is the lung followed by breast, kidney and colon. Radiographic and clinical findings are often nonspecific and the lesion may mimic more commonly occurring benign tumors. Diagnosing such lesions can be difficult and challenging and requires a high index of suspicion. Renal carcinoma is a highly vascular tumor and significant bleeding can occur at biopsy. The treatment of metastatic tumors to the hand is generally palliation since the prognosis in these unfortunate patients is poor.

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POSTERIOR TIBIAL TENDON TENOSYNOVITIS MASQUERADING AS A COMPLETE TENDON RUPTURE BASED ON CLINICAL AND MRI FINDINGS: A CASE REPORT

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PURPOSE

To present the clinical, MRI, intraoperative, and histological data in a case of stenosing tenosynovitis of a posterior tibial tendon.

CASE

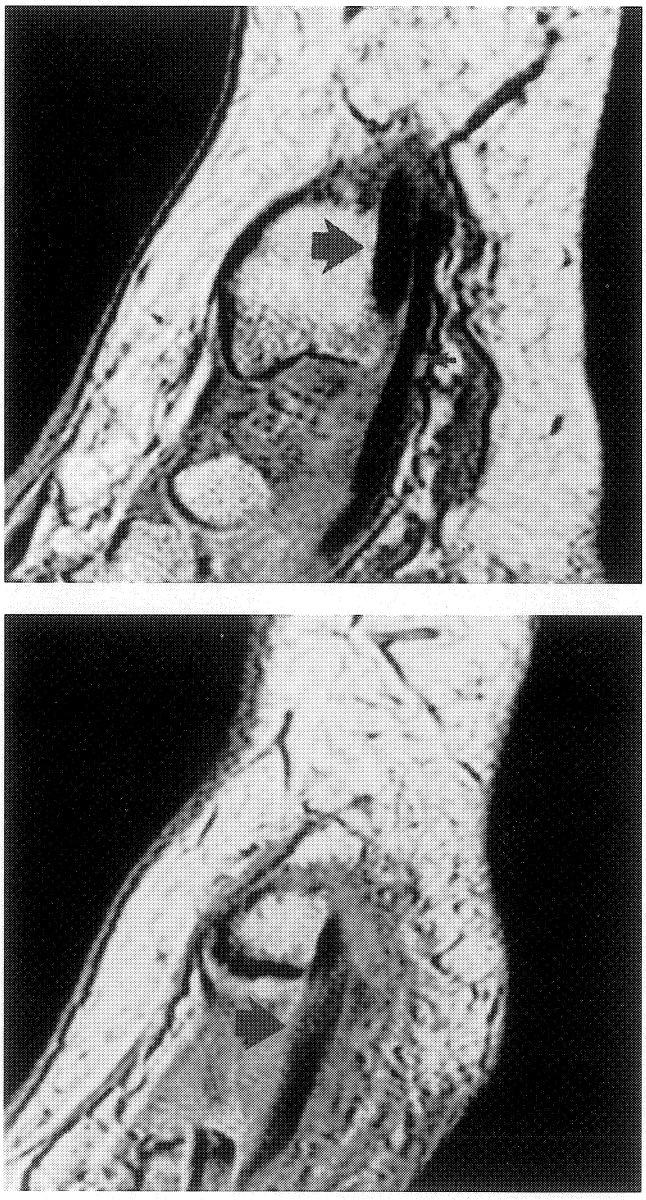
A 62-year-old woman presented with an 18 month history of progressive left medial ankle pain, unrelieved by conservative treatment. The patient also reported that her left foot had progressively drifted outward. Plain x-rays were significant for bilateral heel spurs, mild arthritic changes in the first metatarsal cuneiform joint, and bilateral accessory navicular bones.

Physical examination revealed a moderate splay foot deformity. Dorsalis pedis pulses were weak and light touch was intact throughout on both feet. There was full, painless, passive range of motion of all joints in both feet and ankles. The patient was tender to palpation at the insertion of the plantar fascia onto the calcaneus bilaterally, as well as along the distal course of the posterior tibial tendon posterior to the medial malleolus on the left. There was a moderate amount of swelling along the course of the tibialis posterior tendon from the medial malleolus to the tarsal navicular insertion. The patient was unable to actively invert her left foot, and was noted to exhibit a "too many toes" sign.[4]

Laboratory values, including an erythrocyte sedimentation rate, serum uric acid level, and rheumatoid factor were all normal.

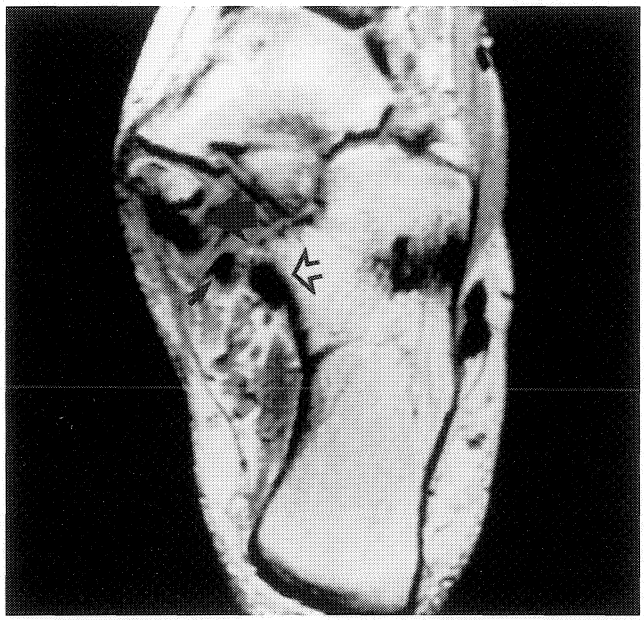
An MRI of the left foot and ankle was performed which was reported to reveal a complete or significant partial tear of the posterior tibial tendon (Figures 1-5).

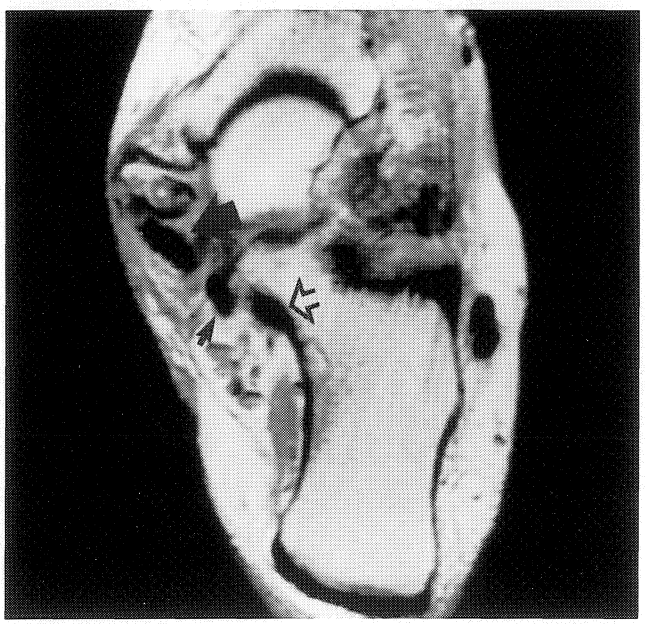
Surgical exploration was accomplished through a direct medial approach at the level of the left ankle. The posterior tibialis tendon was identified, and was completely intact. In addition to this unexpected finding, the tendon was hypertrophied three times normal just distal to the flexor retinaculum. The enlarged bulbous portion of the tendon measured 1.6 cm by 2.8 cm by 1.1 cm. The tendon inserted onto both the accessory and tarsal navic-



Figures 1 & 2
Sagittal MRI views of the posterior tibial tendon (large arrow) and the flexor digitorum longus tendon (small arrow) at the level of the ankle. Note the apparent disruption of the posterior tibial tendon (TR 35, TE 6, slice thickness 1 mm, slice spacing 1 mm).

ular bones in a fan-shaped manner. There was evidence of chronic tendinitis distal to the flexor retinaculum with adhesion of the tendon to its tendon sheath. An attempt at passive inversion and plantar flexion of the foot revealed that the bulbous portion of the posterior tibialis tendon was unable to pass beneath the flexor retinaculum. Release of the flexor retinaculum longitudinally restored the posterior tibialis tendon excursion, allowing passive plantar flexion, inversion, and forefoot adduction. Longitudinal debulking of the bulbous enlargement was performed revealing a homogeneous fibroconnective tissue with no evidence of purulence or necrosis. We observed a scant amount of clear synovial fluid and inflammation of the synovial membrane surrounding the tendon. The debulked tendon edges were reapproximated with a 4-0 Dexon suture. The tendon now appeared to glide normally with passive plantar flexion and forefoot inversion. The flexor retinaculum was reapproximated and the wound was closed without difficulty.

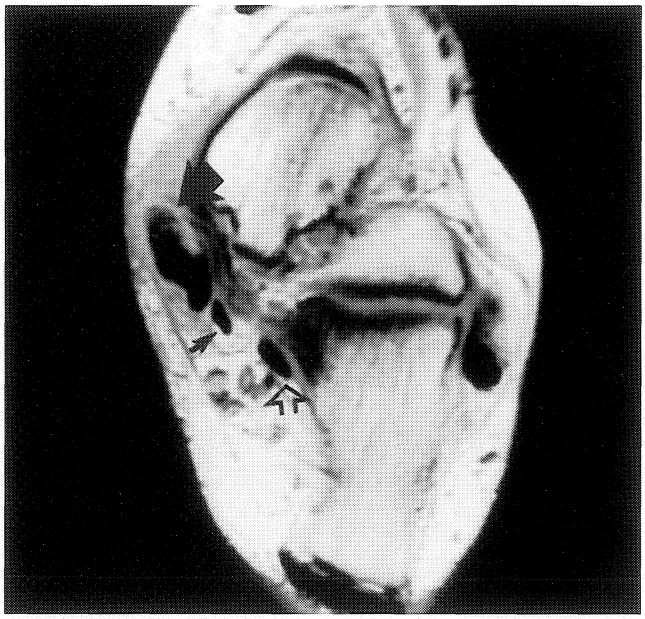




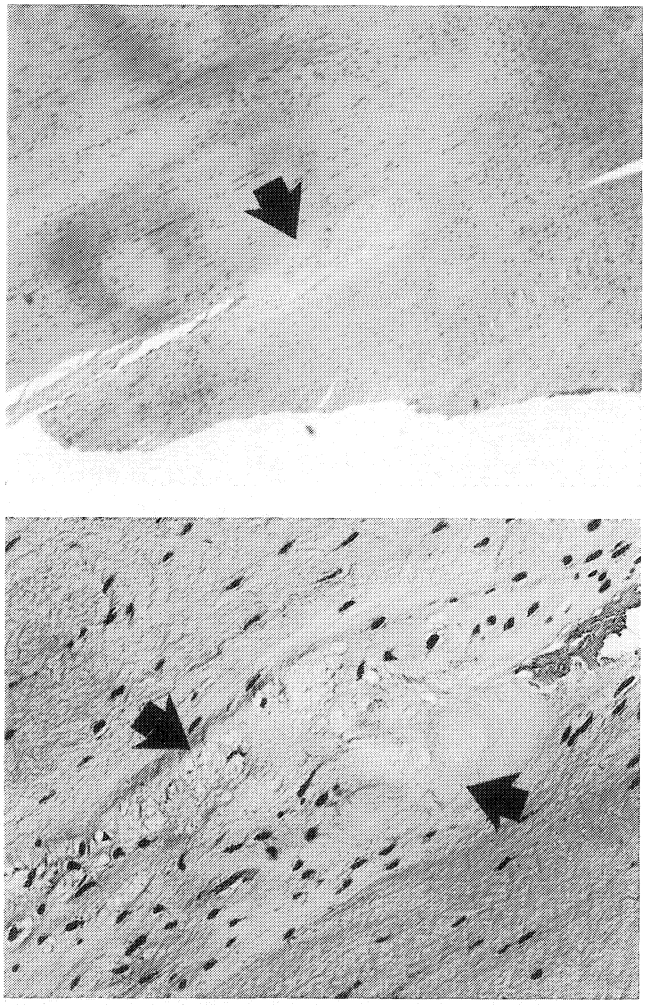
Histological analysis of the debulked specimen revealed dense fibroconnective tissue with focal areas of myxoid degeneration. Small areas of calcification and inflammation were noted. The histological findings supported a diagnosis of tendinitis associated with chronic degeneration (Figure 6-7). Follow-up examination two months after surgical exploration and debulking of the posterior tibial tendon revealed complete resolution of the patient's symptoms and the wound had healed uneventfully. Active ankle inversion, forefoot adduction and plantar flexion was restored without evidence of impingement or tenderness at the medial aspect of the ankle.

DISCUSSION

Non-infectious tenosynovitis was first described by Velpeau in 1818, and again in 1825.[1] Kulowski was the first to specifically describe tenosynovitis of the posterior tibial tendon in 1936. Lipscomb, in 1950, reported an estimated 16% of all cases of tenosynovitis affected the feet and ankles.[1] Several case reports have eloquently described the clinical and operative findings of tenosynovitis affecting the posterior tibial tendon.[2] Our case report is unique because it also includes MRI findings which were



Figures 3, 4, & 5
Coronal MRI views of the posterior tibial tendon (large arrow), flexor digitorum longus (small arrow), and flexor hallucis longus (empty arrow). Note the increased T2 weighted signal and abnormal thickening of the posterior tibial tendon. (TR 1800, TE 14/80, slice thickness 3 mm, slice spacing 1.5 mm).



Figures 6 & 7
High and low power views of the histological specimens demonstrating the myxoid degeneration (large arrow) within the posterior tibial tendon.

interpreted as a complete or significant partial tear of the posterior tibial tendon.

Since the recent advent of magnetic resonance imaging, many diagnostic conundrums have been illucidated.[3] MRI has been extremely useful in diagnosing a wide variety of musculoskeletal problems. MRI can provide high resolution images of tendinous structures. However, a paucity of information is available regarding the posterior tibial tendon. We attempted to utilize the high resolution capabilities of MRI in diagnosing a problem that was clinically a complete or partial rupture of the posterior tibial tendon. The report by two staff radiologists, with expertise in the interpretation of magnetic resonance images, concluded that the patient had a complete or partial rupture of the posterior tibial tendon. Operative exploration, however, revealed an intact tendon with gross and histological evidence of stenosing tenosynovitis.

CONCLUSION

The utilization of innovative technologies has traditionally been attractive, especially within the field of orthopaedics. Unfortunately, sophisticated tests do not always yield clear answers, but may muddy already murky waters. The MRI interpretation of posterior tibial tendon pathology may be inaccurate, as it was in this case. Further investigation of the tendinous anatomy of the foot and ankle by magnetic resonance imaging may improve the ability to accurately interpret pathological entities in this region.

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LATE ELBOW INSTABILITY: CASE REPORT AND LITERATURE REVIEW

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INTRODUCTION

Recurrent dislocations of the elbow are rare.[2,3,5,7] At an average of 24 years after injury, Josefsson et al found no recurrent dislocations in their series of 52 first time dislocations.[6] Lindscheid and Wheeler reported only two recurrences out of 110 initial dislocations.[7] Recurrent elbow instability without dislocation is more commonly recognized.[1,3,5,6,7,8,11] Some have claimed that elbow instability is typically asymptomatic.[1,6] This is a report of the late complication of elbow instability presenting as an incarcerated subluxation of the elbow, many years after elbow dislocation.

CASE REPORT

A 48 year old female homemaker presented to our emergency room complaining of the inability to move her left arm. While turning a vacuum around a piece of furniture, she extended her left elbow, felt sudden pain and was unable to flex or extend the elbow. She denied any trauma to her left arm or loss of consciousness. She did relate dislocating her left elbow at age 12, requiring a reduction maneuver and casting. During the intervening years, she had experienced occasional popping in the elbow but no pain.

The last episode of popping was nearly three years prior to this presentation. Inspection of the left arm showed the elbow locked in 45 degrees of flexion. The patient experienced pain with the examiners' attempts to move it. No contusions or swelling were noted. The neurologic and vascular exams of the left arm were entirely normal. X-rays showed a posterior subluxation of the left elbow (fig. 1). After intravenous sedation, longitudinal traction was applied to the left arm for approximately three minutes before reduction of the elbow occurred (fig. 2a and b). We demonstrated a stable range of elbow motion from 15 degrees to 120 degrees with minimal discomfort.

DISCUSSION

Traumatic elbow dislocations are generally caused by a hyperextension injury with the olecranon process impaling the olecranon fossa, causing the anterior portion of the joint to hinge open. The posterolateral dislocation is most common due to the lateral slope of the trochlea. With the posterolateral dislocation, significant valgus forces cause failure of the medial collateral ligament.[10] The medial collateral ligament must be ruptured, avulsed, or attenuated for an elbow dislocation to occur.[11] After surgical exploration of 31 consecutive elbow dislocations, Tullos et al has reported complete tears of the medial collateral ligament in all 31 patients.[10]

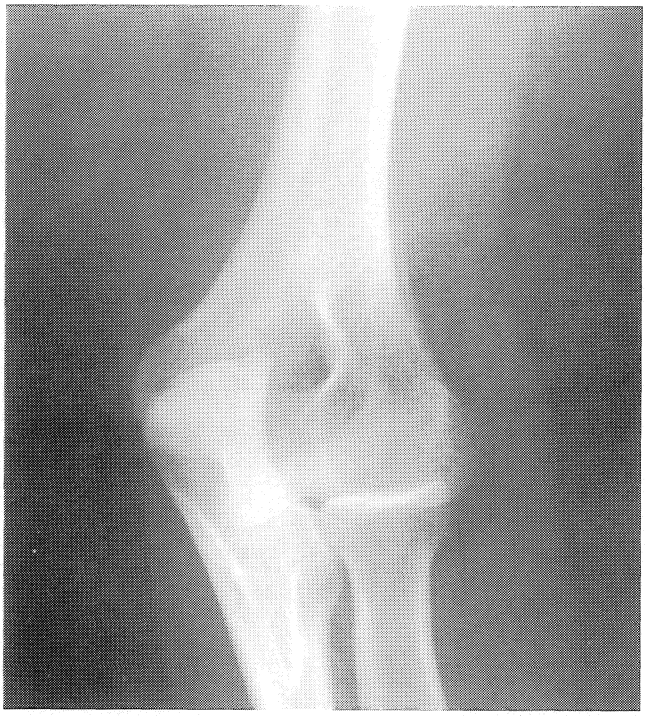

Figure 1

The anterior oblique portion of the medial collateral ligament is the primary stabilizer of the elbow to valgus stress, with negligible contribution from the posterior oblique portion.[4] Hotchkiss and Weiland have also shown the importance of the radial head to valgus stability of the elbow, reporting a 30% decrease in valgus load resistance after removal of the radial head in cadaveric specimens.[4] The extremely small redislocation rate after primary elbow dislocation (0-3%)[5,7] is significantly increased in patients with elbow dislocation and radial head fractures.[10]

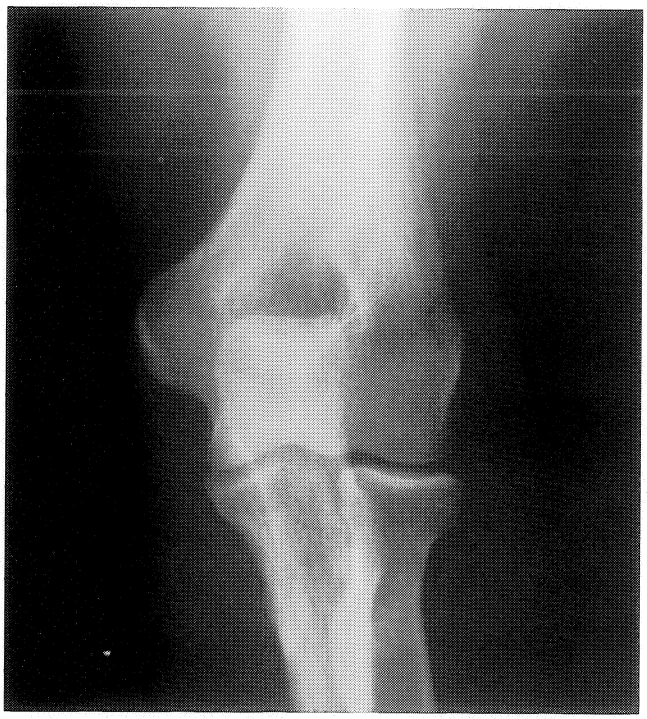

Figure 2A

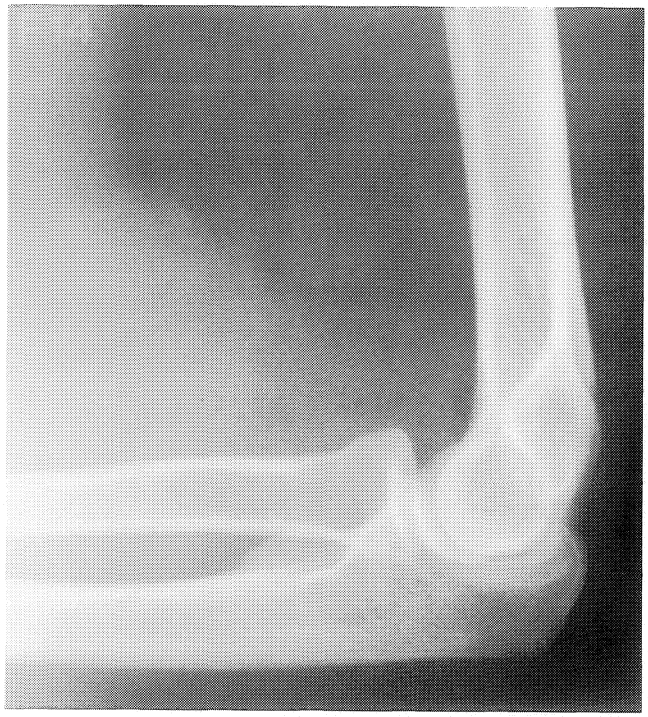

Figure 2B

The structures on the lateral side of the elbow seem to offer little stability to the elbow. Josefsson et al has shown in cadaveric specimens that sectioning the lateral collateral ligament and the lateral extensor musculature origin produces no significant elbow instability, as long as the medial structures are intact.[6] The articular surfaces and not the lateral soft tissue structures are the primary varus stabilizers of the elbow.[6]

More common than redislocation, but typically regarded as asymptomatic, is valgus instability of the elbow. Josefsson et al reported a series of 52 patients with elbow dislocations in which eight displayed clinical evidence of valgus instability. All were asymptomatic.[6] Borris et al reported on 41 adult patients two to 19 years after elbow dislocation, finding eight unstable elbows-seven in valgus and one in varus.[1] They also followed 43 children two to 16 years after an original elbow dislocation, finding 11 unstable joints. Eight were unstable in valgus, two in varus, and one in both varus and valgus.[1] None of the eight adults or 11 children were symptomatic.[1]

Recently, another form of elbow instability has been described—posterolateral rotatory instability.[8] According to O'Driscoll et al, this condition is caused by laxity of the ulnar portion of the lateral collateral ligament, allowing rotatory subluxation of the ulnohumeral joint and subsequent subluxation or dislocation of the radiohumeral joint.[8] Patients with this form of instability complain of locking, snapping, and giving way of the affected elbow and may experience redislocation by such trivial movements as extending the arm to turn on a light switch.[8]

Various surgical procedures have been described to correct chronic elbow instability, all performed on a small number of patients with limited follow-up. Zeier described using dual fascia lata slings to stabilize the lax medial and lateral collateral ligaments.[12] Direct repair of the medial collateral ligament to bone using a Bunnell suture and coronoid bone block transfers have been described.[11] Advancement of an osteotomized medial epicondyle with the attached medial collateral ligament also may restore valgus stability.[9] Dryer et al described an anterior approach to repair the avulsed anterior capsule and brachialis muscle, which they found in their patients with chronic elbow instability.[3] O'Driscoll et al have described direct repair of the failed ulnar collateral ligament in their series of patients with posterolateral rotatory instability.[8]

CONCLUSION

This is a report of late elbow instability with minimal provocation, causing significant morbidity many years after the original injury. Surgical exploration of acute posterior elbow dislocations has shown that the primary valgus stabilizer of the elbow, the medial collateral ligament, is commonly disrupted. This implies that the potential for chronic elbow instability exists in every patient suffering a posterior elbow dislocation. Therefore, the patient with even the most subtle elbow complaints following a reduced elbow dislocation should be carefully

examined for chronic elbow instability, irrespective of the length of the follow-up.

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RICHARD III—A VILLAIN FOR THE AGES AN ORTHOPEDIC DILEMMA

Kathi F. Teixeira, M.D.
Richard L. Jacobs, M.D.

Many orthopaedic surgeons, not including us, are egoists. This, when combined with a soupcon of exhibitionism can be a germinal event.

With this in mind, let us delve into history and center on William Shakespeare the master playwright.

First, his play *Richard III*. Consider the famous soliloquy:[3]

> But I, that am not shap'd for sportive tricks,
> nor made to court an amorous looking-glass
> I, that am rudely stamp'd, . . .
> Cheated of feature by dissembling nature,
> Deform'd, unfinish'd, sent before my time
> Into this breathing world, scarce half made up, . . .
> And therefore, since I cannot prove a lover,
> To entertain these fair well-spoken days,
> I am determined to prove a villain,
> And hate the idle pleasures of these days.
> (Shakespeare, Richard III, I., i)

How many actors of varying rectitude have declaimed these famous lines? Ah, the pathos, the chest-beating!

PURPOSE

Orthopedic Rumination About Richard!

MEDITATION

There are really two schools of thought about Richard. The one described him as a notorious villain replete with multiple deformity. The earliest description of Richard's deformities is dated at 1491, six years after his death. He was described by John Payntour as being a "crook back". Sir Thomas Moore later reported him to be "little of stature, ill-featured of limbs, crook-backed, his left shoulder much higher than his right. . . ." These, and later decidedly partisan observations were described by participants in the War of the Roses. Some of these reports were contemporary, but many, such as Shakespeare's, came in later years. The Bard really did a job on him! Try, for example, "Unlick'd bear-whelp' lump of foul deformity; bloody dog". It could be argued that most of these reports had been cooked! This school agrees on Richard's affects: if no one will accept such a deformed, ill-favored person as he, they can expect to be paid in the extreme.[2]

Another school, members of the Richardian Society, state that various people describing Richard's deformity were politically motivated. They argued that he was not deformed at all and no absolute contemporary reported a deformity! Contemporary head and shoulders portraits do not demonstrate deformity. They freely admit that he did many things we presently would look askance at. He may have caused the little princes to be murdered in the Tower of London (but no one is perfect!). He also may have been responsible for the murders of the Duke of Somerset, Prince of Wales, and King Henry VI. Perhaps he mandated the beheading of the Duke of Buckingham and the Lords Rivers and Hastings but *surely* he was a victim of circumstance. It was only by coincidence that he married the widow of the murdered Prince of Wales and subsequently poisoned her. And yes, he probably did lure his brother Clarence to be drowned in the head of Malmsey wine, but this was not without provocation. Admitting that all of

Figure 1
Richard III. Thus ends the Plantagenet Line!

these things may be true, the Richardians feel this was the standard art of kings in that era. They *do* vehemently deny that Richard had any physical deformity.[4]

When such contradictory testimony is offered, turn to Occam's Razor (the philosophical concept that when there are competing theories, the simplest is the truth). We choose to believe that Shakespeare *was* right, and Richard was a notorious villain because of reaction formation to his physical deformities and handicaps. This would be a simple explanation.

Daydream and consider the possibilities. If we had a time warp, we could transport a spine surgeon back in time—one morning in an operating room to perform a spinal fusion with instrumentation —a lessening of deformity. Perhaps Richard's true, sweet nature would emerge. Perhaps he *would* prove a lover! Possibly, there would be no War of the Roses. Perhaps his royal line would have continued. The Plantagenets might still be enthroned. He was placed in a coffin which was later used as a horse-trough and then broken up and used as the steps for the cellar of the White Horse Inn. Instead, he might have eventually made Westminster Abbey![4] History would have changed.

But what would Junius Booth have done? Would his son, John Wilkes have turned to acting? What would Laurence Olivier have done? They might have been actors, but not of so great a repute. No pathos, no chest beating, nor flaring of nostrils. Some of the flavor of our life would be lost. Would this have changed history? If the opening quotation represents Richard III's true self appraisal—it most certainly would have.

On the one hand, villainous, infamous exploits would have been aborted. On the other hand, one of the greatest plays in English literature would be forever lost. Therefore, *die* Richard III must! As his illustrious forebear Richard I (Richard, Coeur deLion; the Lion-Hearted) once said of the Plantagenet line, "From the Devil we sprang and to the Devil we shall go."[3] We therefore believe that we have described a new contraindication to spinal instrumentation!

CONCLUSION

A spine is only today, or a lifetime. Shakespeare is forever!

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LETTER TO THE EDITOR:

MORE ON "ETYMOLOGY AND THE ORTHOPAEDIC SURGEON"

Dear Sir,

Recently, one of our Professors passed around to us an article from your Iowa Orthopaedic Journal 1991 by Drs. S.F. Dye, B.E. van Dam and C.W. Westin titled, "Etymology and the Orthopaedic Surgeon: Onomasticon (Vocabulary)." He felt that it would be a valuable addition to the Residents' files. And indeed it is, for which I would like to thank the authors and your Journal for bringing it to the attention of your readers. I would like, if I may, to make a few corrections and comments which I think will be equally valuable to your readers.

The word tibia is not used merely because of a resemblance of that bone to a flute, but rather because the first such musical instruments were actually made from tibiae. The Greek word *omos* refers to the shoulder and upper arm together. The root of this word is *omer-*: this is how we arrive at humerus, which is the Latin derivative. Clavicle is not diminutive for *clavis* ("key"), but for *clavus*, meaning "nail". One can see how the Romans made use of such bones as nails to secure, for example, their tents in the field of battle. Hamate does come from the Latin *hamatus*. However, this does not mean "hook-shaped" (for the bone is not) but "furnished with a hook", which in my opinion more accurately describes the anatomical configuration. The root *agra* in the word podagra does not mean "seizure or attack". Rather, it comes from the Greek verb *agreo,* meaning "to pursue eagerly, to hunt after." Aristotle used this term to describe the patient's behavior as he "eagerly" reached for his foot in pain during a gouty attack. Skeleton does not derive from the Greek for "dried up". This root is *skell-*. The correct derivation is *skelos* (with a single 'l'), meaning "leg". Skeleton would thus be a logical consequence.

Synovium does come from the Greek *syn-* and the Latin *ovum*. However *syn-* does not mean "of" or "like" but "with" or "together." It is worth being careful about the meaning here because this word occurs so frequently in medical terminology. Hence, syndrome means a "running" (Greek verb *dramein*) "together" [of symptoms] and synergism means "working" (Greek verb *ergo*) "together".

Cuneiform precedes both the Greeks and the Romans. The root *cuneus* dates back to the Sumerians, who are considered to be the first civilization to adopt a written language. They wrote on clay tablets in symbols etched out by wedge-shaped instruments. This, which we now take for granted, was revolutionary. Indeed the Greeks, centuries later, continued to emphasis the spoken form of language, writing in metre. They insisted that to read in silence was to miss much of the subtlety and depth of language. This was the driving force behind Greek theatre and oratory.

The words astragalus and talus are very interesting. The Greeks did indeed use these bones as dice. They played a game utilizing dice with four sides and rounded edges (properties of these bones) and numbered 1,3,6 and 4. They also played another game utilizing dice with six sides numbered 1 through 6 and referred to as *kuboi.* Which bone did they use for this game? The cuboid, of course. Finally, the root of talus is not *taxillus-* (this was simply an alternate acceptable name for this bone), but *TEC-,* from which stems the verb *tego* (texi, tectus, tegere), "to cover", and in turn the noun *tectum,* or "roof". The talus was considered to be the roof of the foot.

The relationship between fascia and Fascist is also very interesting. Mussolini's Fascist comes from *fasces* (pleural of *fascis*), which the Romans used to describe a bundle of rods with an axe carried before the highest magistrates as an emblem of authority. There are many references to this in Latin, such as Cicero's *imperi populi Romani fasces,* or the "'fasces' of the imperial Roman people". Mussolini was not alone in drawing on the Roman Empire for his symbolism. Hitler's Swastika can be found in the decorative mosaics of Pompei.

Just as the sartorius muscle was so named to describe its function in helping the tailor to assume his typical sewing position, the *gracilis* muscle was often described as the "custodian of virginal Humility". Its function in adducting the thigh can readily be deduced from this description!

To say "Ibn Avicenna" is redundant. Avicenna alone is sufficient, as it is the Anglicized rendition of the Arabic "Ibn Sina" or the son of Sinai. It is tantalizing to note that many scientific historians are now attributing to Avicenna the first description of the circulation of the blood. This would precede the work of Harvey by half a millennium, an astounding accomplishment when one considers the state of medical knowledge and science at that time.

That the Greek noun *cheir,* meaning "hand", forms the root of the word "surgeon" is reflected to this day in the British abbreviation M.Ch., which stands for *Magister Chirurgiae,* or Master of Surgery. Now we can all understand why British Surgeons have these letters after their names.

Finally, Galen was not a Roman but a Greek. He came from Pergamum, a city of Mysia which was then a Hellenistic Kingdom in Asia Minor and would today constitute a part of Turkey. Having received his education in the great medical centre of Alexandria, he began as a

gladiator-physician in Pergamum. Later he journeyed to Rome, where he became court physician to the most scholarly of all Roman emperors, Marcus Aurelius. Galen was not the only Greek to find employment as a physician in Rome. Indeed, the Roman felt that it was below his dignity to practice medicine, leaving that profession to slaves and foreigners (the *barbari,* from which we obtain the word "barbarians" and which gives us some idea of the prejudice with which Western tradition has historically regarded things non-Western) while he pursued the more noble professions of politics and law!

As an undergraduate, I studied Greek and Latin. I have always considered the long hours spent pouring over old and dusty classical texts worth every last minute. Because of this, I must disagree with the conclusion of Drs. Dye, van Dam and Westin, that an understanding of the background to medical etymology is valuable not so much for its aesthetic qualities but for the help it will offer the Orthopaedic Surgeon in "learning new and foreign-sounding words". Why place all the emphasis on the practical? We as a society are quickly rendering extinct our aesthetic sense. In a cold and mechanical world, we glorify efficiency, productivity, and results. We stress vital signs, laboratory values and test results. We practically ignore the physical examination, mock the psychosocial aspects of illness and look upon the patient as a jumble of molecules and integrated systems. How are we then different from a chemist or an engineer or a mechanic? What has happened to the **colour** of Medicine? Hippocrates wrote *Longa ars, vita brevis*: "long is the Art, but life is short". Are we determined to stamp out the *Ars* in Medicine altogether? What I have come to value most about my studies in Greek and Latin has not been the practical tools with which I can now dissect medical terminology. To the contrary, I cherish most the colour which they have enabled me to infuse into what can only be described as a rapidly paling profession.

Yours sincerely,

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LETTER TO THE EDITOR

Dear Editors:

This is in response to the comments of Dr. Mohammad Diab. I appreciate very much the interest on the part of Dr. Diab in the topic of medical etymology as expressed in his letter.

As Dr. Diab recognizes, there are indeed subtleties in language and especially in the origins of current terminology. These subtleties can be and often are the source of controversy among recognized scholars in the field of linguistics. Drs. van Dam, Westin and myself are well aware that controversies exist and have not represented our work as the only interpretations extant. We did research many sources that are available on this topic and checked several references for each of the words presented in our Onomasticon. Where there were areas of controversy, we decided to rely on what is considered by many medical librarians as the "classic" of medical etymology, *The Origin of Medical Terms* by Henry Allen Skinner, M.B, FRCS. (C) published by Williams and Wilkins in 1961.

I would direct Dr. Diab and other interested individuals to this excellent source for a more extensive discussion of the terms we chose to present in our article.

For example, regarding the etymology of clavicle, Dr. Diab states that it comes from "clavus," meaning "nail." I would like, if I might, to have reprinted the section of the clavicle from the Skinner text.

CLAVICLE

Latin-claviculum, diminutive of clavis, a key.

Probably related to claudere, to shut or close. Note also the Greek κλείς, literally that which serves for closing or locking up, thus a key or a bolt. The Greek combining form "cleido" is thus used sometimes for clavicle as in sterno-cleidomastoid. Aristotle said clavis is an instrument for closing anything and signifies the bone which closes the thorax. Aristotle's father was physician to Philip of Macedon who suffered a broken clavicle. When it was nearly healed he replied to the request of his physician "Occipe quantum volueris, Claves tenes" (take as much as you wish, you hold the key). Both the Latin clavis and the Greek κλείς were terms used for key or bolt in the sense of a door-opener or a door-fastener. They were also used for the catch that fastens a window. This latter gives us the correct clue since a Roman key was shaped much like one of our old-fashioned large keys and this is not the shape of the clavicle. A curved window fastener is, however, a familiar object, and particularly in the more ornamental forms is exactly the shape of the clavicle. Barclay Smith also notes the kind of stick used by boys in trundling a hoop, a stick with a double curve and known to the Romans as "clavis trochi," which is also the shape of a clavicle (pictured in Anthony Rich's "Römischen Alterthümern," p. 161). Whether it was a window fastener or a hoopstick that first suggested the name clavicle is, however, unknown. Celsus used the word jugulum for this bone and the term clavicula occurs first in translation of the Canon of Avicenna. Vesalius adopted clavicula but also used clavis (under hinge joints Vesalius has an illustration (see accompanying figure) of the two metal plates of a door hinge secured together by a nail or rod which he calls clavis). The word has been used in English since 1600.

Regarding Galen, Dr. Diab is quite right in indicating that he was of Greek origin. His father, Niko, was an Asiatic Greek. Galen was born at Pergamos and is sometimes referred to as the Pergamite. However, recognizing his Greek origins, Skinner still refers to him as Claudius Galen, a Roman physician.

I would disagree with Dr. Diab's characterization of our concept of the value of knowledge in this area. We feel that the primary benefit to those who would take the time to look into the origins of our medical terms is that of aesthetic satisfaction. However, we feel, in addition to the aesthetics, knowing the derivations is of assistance in learning the terminology, especially for the young physician.

Sincerely,

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