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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 1990 journal is February 1, 1990.

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When you send an article it is *essential* that the following items be submitted:

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2. A **bibliography,** alphabetical and double-spaced, of 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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Preparation of manuscript: Manuscripts must be typewritten, double-spaced with wide marigns. Write out figures under 100 except percentages, degrees, or figures expressed in decimals. A direct quotation should include the exact page number on which it appeared in the book or article. All measurements should be given in SI metric units. In reporting results of surgery, only in rare instances can cases with less than two years' follow-up be accepted.

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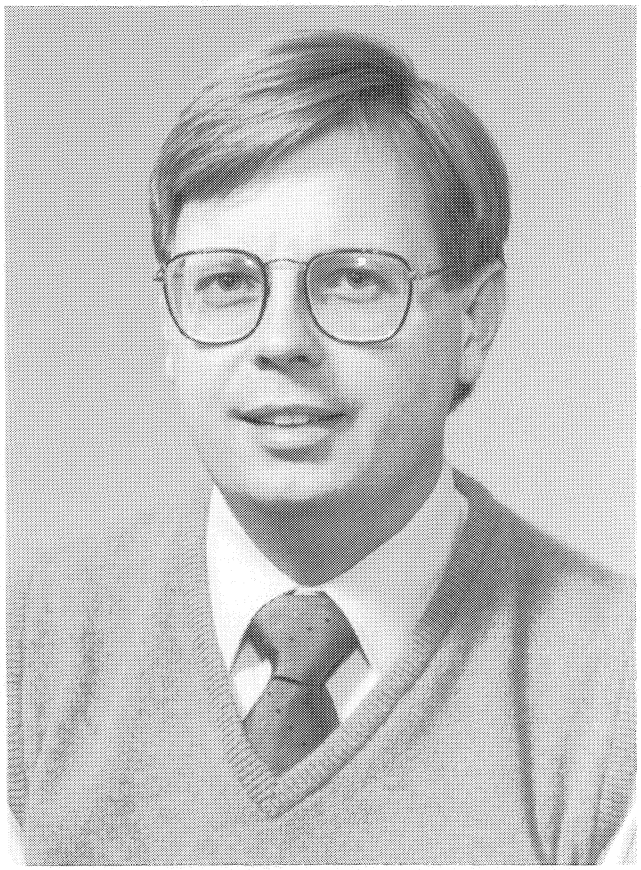
The Iowa Orthopaedic Journal



Richard A. Brand, M.D.

"It is what we think we already know that keeps us from learning."

Claude Bernard

EDITORS' NOTE

The Iowa Orthopaedic Journal has as its primary objective education and seeks to reflect the activity of the residents, fellows, faculty, alumni and visitors of the Department. We wish to thank Drs. Cooper, Clark and Found for their support and guidance; Mr. Paul Etre and Mrs. Kay Redlinger-Phillips for their administrative assistance and Lori Schneider for her superb secretarial help. Also, we extend special thanks to the Iowa Orthopaedic Society for their continued support.

The 9th Volume of the Orthopaedic Journal is dedicated to Dr. Richard A. Brand: researcher, teacher, surgeon and friend. His critical analysis of both new and accepted orthopaedic concepts and his fundamental approach to problem solving stimulate orthopaedic residents to think with perception and creativity. We appreciate his sharing of knowledge, experience and incite, and we thank him for accepting us as his colleagues.

James L. Guyton, M.D.

Peter M. Murray, M.D.

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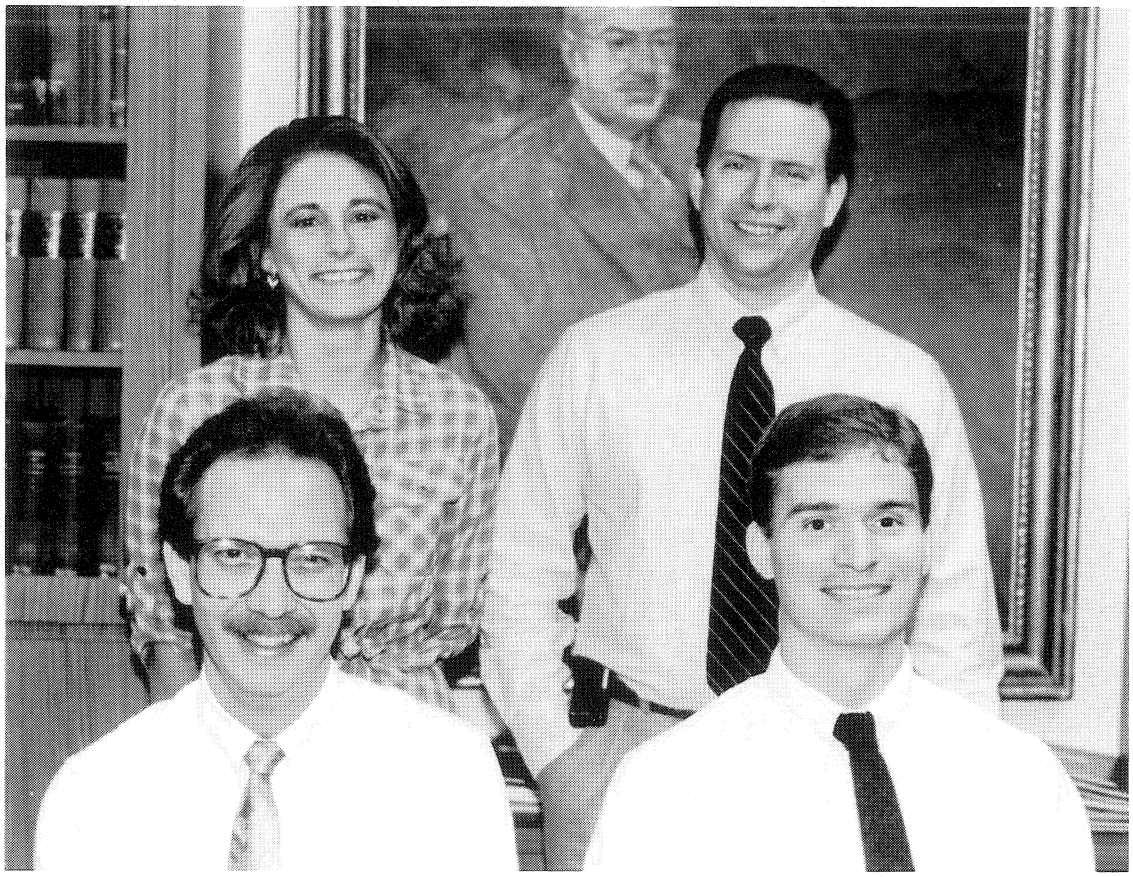

Standing left to right: Laura J. Trombino, M.D.; Robert F. McLain, M.D. Seated left to right: Richard A. Berger, M.D., Ph.D.; David A. Oster, M.D.

Richard A. Berger, M.D., Ph.D. was born September 21, 1954 in Austin, Minnesota. He attended Midland Lutheran College in Fremont, Nebraska where he received a B.A. degree in Biology and Natural Sciences. He received a Ph.D. in Anatomy in 1981 and his M.D. degree in 1984, both from the University of Iowa. Dick will move to Rochester, Minnesota to complete a hand surgery fellowship at the Mayo Clinic.

Robert F. McLain, M.D. was born in Agana, Guam in 1956 and raised in Southern California. He received both a B.S. degree in Biology and an M.D. degree from the University of California at Davis. Rob and his wife Becky plan to remain in Iowa City where he will complete a one year research fellowship in the Department of Orthopedics studying two topics: "Neuropeptides" and "The Neurosensory Elements of the Anterior Cruciate Ligament."

David A. Oster, M.D. was born on July 16, 1959 in Des Moines, Iowa and raised in Syracuse, New York. After receiving his B.S. degree from the University of Iowa, he obtained his M.D. degree from the University of Iowa College of Medicine in 1984. Dave and his wife Ann will be relocating to Cincinnati, Ohio where he will complete a sports medicine fellowship at the Cincinnati Sports Medicine Center.

Laura S. Trombino, M.D. was born and raised in Waukegan, Illinois. She later graduated from Bowling Green University in Bowling Green, Ohio with a B.S. degree in Biology. In 1984, she graduated from Northwestern University Medical School. Laura plans to remain in Iowa City to complete a clinical fellowship in pediatric orthopedics.

DEPARTMENTAL EVENTS 1989-1990

June 7-9, 1989 — Senior Residents and Fellows Day

Guests: Thomas N. Lindenfeld, M.D.; Cincinnati, Ohio
Ronald L. Linscheid, M.D.; Rochester, Minnesota
Malcolm H. Pope, Ph.D.; Burlington, Vermont

September 28-30, 1989 — Iowa Orthopaedic Alumni Meeting

Guests: Lorraine J. Day, M.D.; San Francisco, California
James O. Johnston, M.D.; Oakland, California
Roger A. Mann, M.D.; San Leandro, California

October 6-7, 1989 — Camp Lectureship

Guest: Mark D. Brown, M.D.; Miami, Florida

May 10-11, 1990 — Hawkeye Sports Medicine Symposium

Guest: To be announced

THE MECHANICAL EFFECTS OF HIP OSTEOTOMY

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Are the beneficial effects of osteotomy mechanical in origin? This question is important because we can not substantially improve our results without knowing how osteotomies achieve their beneficial effects. Our view of how osteotomies work is hampered by our lack of understanding the process we are treating: osteoarthrosis (OA). Our relatively crude observations suggest primary and secondary types of OA implicating multiple etiologies. However, it is unclear whether there are inherent subtle structural and chemical abnormalities causing human "disease" or whether the "disease" is a response to the abnormalities. Without a clear framework in which to observe osteotomies, their interpretation will remain obscure.

Since their introduction for the treatment of osteoarthrosis, osteotomies have been believed to have either biological or mechanical effects or both. The purpose of this paper is to review whether and how osteotomies might create beneficial mechanical alterations. I will not discuss possible biological effects, although I will say that they are not necessarily exclusive or distinct from the mechanical effects. I will outline the possible mechanical effects of osteotomies, briefly describe how we have studied some of these effects and make arguments for or against each of these possibilities.

Historically, investigators have presumed that osteotomies reduce joint loads or joint stresses in some way; more recently it has been suggested that osteotomies result in a redistribution of bone stresses, leading to a remodeling of the bone (i.e., Wolff's Law). Several mechanisms have been suggested which might reduce joint forces (see Table 1). Cartilage and adjacent bone stresses might be reduced by either decreasing the joint load or increasing the contact area of the opposing joint surfaces. Therefore, bone remodeling might be initiated by either increases or decreases in bone stresses.

We have a limited number of ways to study these possibilities. Until recently, we could only study these possibilities with the simplest observations and experiments. It is important to note, however, that the observations we make regarding the mechanical effects of osteotomy are on a "macro" scale, rather than a "micro" scale. It is likely these latter effects, coupled with biological responses are more important than the former. I should also point out that given the apparent complexity of OA and its apparent complex response to osteotomies, it is likely that some of the simple studies might be misleading.

Table 1

Hypothesized Mechanisms By Which Osteotomies Might Beneficially Affect Osteoarthritis

Reduce joint force
Load sharing[11,18,23]
Relax tight capsule[9,10,17]
Relax tight muscles[24]
Reduce muscle forces[1,2,21,24]
Reduce cartilage stress
Reduce joint force
Increase contact area[21,24]
Stimulate bone remodeling[22]
Reduce bone stresses
Increase bone stresses

COMPUTER SIMULATIONS OF OSTEOTOMIES

Several years ago we decided to study some of the possible mechanical effects of osteotomies because we felt, based on our experience with computer simulated surgery, that the existing notions were misleading[15]. Our methods allowed us to mathematically estimate the resultant forces on the hip joint before and after osteotomy[4].

The muscle and joint force problem is formulated in two parts (Figure 1). The first part may be called the inverse dynamics problem in which body segment inertial properties, external forces and body segment accelerations form the input for a Newtonian formula of motion to yield the intersegmental resultant forces and moments. The second part may be called the distribution problem. Since there are many more unknown muscle forces than the maximum eighteen equations of motion (one force and one moment equilibrium equation at the hip, knee and ankle resolved into 3 orthoginal components yields 18 equations), the problem of calculating the muscle forces is indeterminate. However, a unique solution for this indeterminate problem may be provided by the mathematical technique of optimization. The intersegmental moments form constraint conditions for an optimization algorithm which yields the individual muscle forces. That is, any muscle and contact force solution must result in the experimentally observed intersegmental forces and moments. The muscle forces

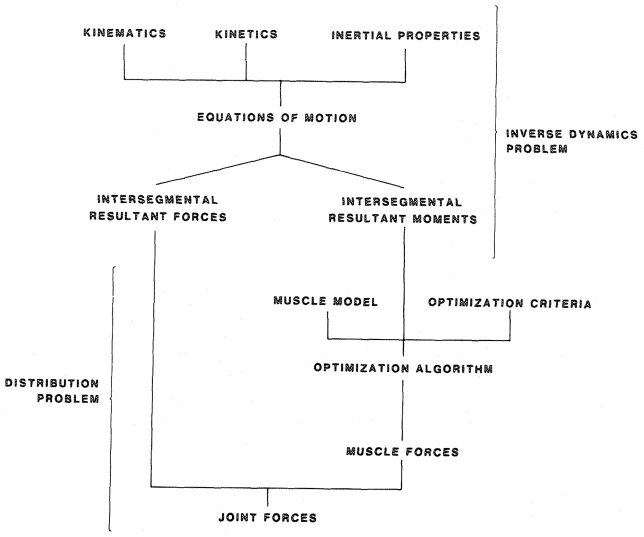

Figure 1:
Formulation of muscle and joint force calculation problem.

are than vectorally subtracted from the intersegmental resultant forces to yield the joint contact force.

Body segment accelerations are determined by three-dimensional photographic technique, and body segment inertial properties are estimated by making anthropometric measurements on the subjects. The body segment parameters are then calculated based on published regression equations[8]. External forces are recorded with a Kistler piezoelectric force plate. The equations of motion then yield the intersegmental resultant forces and moments. These resultant forces are not the actual forces on the joint surfaces but rather the vector sums of all the forces in muscles, capsule, and on the joint contact surface.

To solve this distribution problem, we formulated an optimization criteria based on the experimentally determined exponential relationship between muscle force and endurance time[7]. Using three as an exponent in the middle values reported for the exponential relationship between muscle force and endurance, and assuming that this relationship applies to muscle stresses the same as it does to muscle forces, we minimized the sum of the muscle stresses cubed thereby maximizing endurance.

A 47 element three-dimensional muscle model was developed from the dissection of six cadaver limbs. The locations of the muscle origins and insertions relative to the joints was studied in each specimen[3]. Thus, we determined the three-dimensional moment arms of each muscle element throughout the gait cycle.

Using a nonlinear optimization algorithm, we then calculated the forces in each of the 47 muscles. These force predictions were correlated with five to ten channels of simultaneously collected EMG signals on normal subjects. This provided us with a way to determine how good the predictions might be. Predictions were not used when they did not reasonably agree with the EMG. Finally, we calculated the hip contact force by vectorally subtracting all the muscle forces from the intersegmental resultant force (Figure 1). This does not yield a force acting on a point, but rather a force distributed over the articular surface.

Our three-dimensional model allowed us to alter the muscle origins and insertions in any manner. Therefore, to simulate intertrochanteric osteotomies, we rotated the proximal and distal fragments of the femur to create 10, 20, and 30 degree varus, valgus, anteversion, and retroversion osteotomies (Figure 2). This new muscle and joint geometry then formed the input for new optimization problems at each instant during the gait cycle.

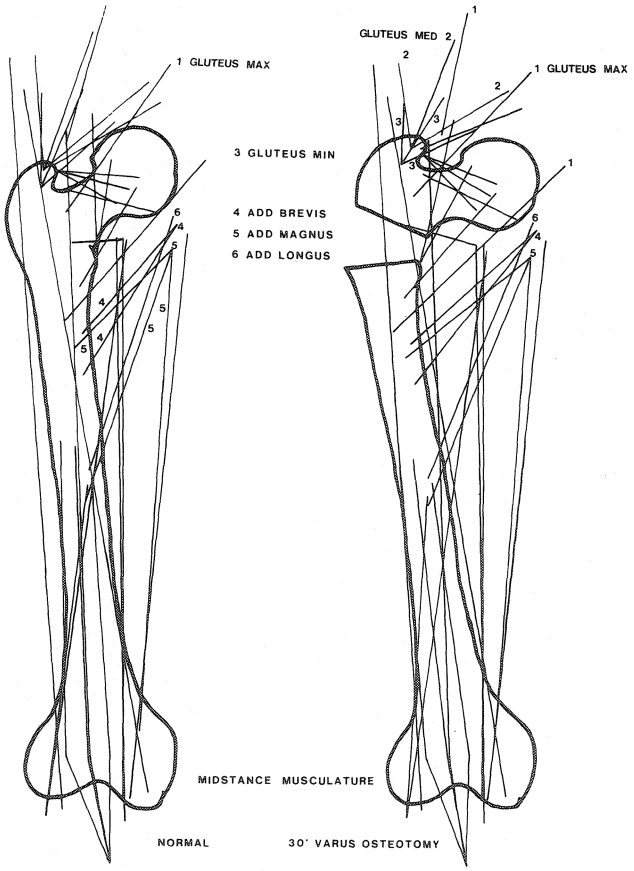

Figure 2:
Frontal plane projection of 3-D muscle model before (left) and after (right) varus osteotomy. (Illustration reproduced with permission of C.V. Mosby).

However, there are four major assumptions of this study. First, we assumed that biological effects of an osteotomy will not confound the mechanical effects. Second, we assumed that the osteotomy does not change the relative locations of the hip and knee "centers". We know that this assumption is only approximately correct. However, this assumption would be correct for a varus osteotomy with

interposition bone grafting and medial displacement of the distal fragment, and would also be correct for a valgus osteotomy with slight femoral shortening and lateral displacement of the distal fragment. Third, we assumed that gait is normal after osteotomy. This is also not true. Modeling is not yet sophisticated enough to input the biological, clinical and mechanical changes necessary to eliminate this limitation. Fourth, gait was assumed to occur on level surfaces. Differing forces would need to be predicted for other activities of daily living. Previous computer models have been subject to not only these but other limitations: single muscle modeling, planar anatomy and static equilibrium.

HYPOTHESIZED MECHANISMS OF HOW OSTEOTOMIES WORK

LOAD SHARING

The first is the load sharing concept proposed by Galland (unloading of the hip joint by articulation of the distal fragment with the inferior aspect of the acetabulum), and later supported by McMurray as well as by Osborne and Fahrni[11,18,23]. Given the amount of medial displacement recommended by these authors and considering their x-ray results, this mechanism is quite likely. However, this mechanism is clearly not consistent with current surgical practices where the distal fragment does not articulate with the inferior acetabulum and the transverse acetabular ligament.

RELAX TIGHT CAPSULE

Lloyd-Roberts and later Ferguson suggested that a primary effect of osteotomies was to relax a tight capsule[9,10,17]. This might certainly provide initial, if not lasting, pain relief; it could also lower the hip joint load. However, it is difficult to believe that the joint load could be substantially lowered without improving hip range of motion. If the latter could not be achieved (there is no reported evidence suggesting osteotomies improve hip range of motion), it is unlikely that relaxation of a tight capsule could alter the natural history of osteoarthrosis. Furthermore, this mechanism can not explain the good results of medial displacement osteotomies without significant angulation of the proximal fragment. It is even contradictory with Bombelli's notion that traction, that is increased strain, placed on the capsule results in the formation of beneficial osteophytes.

RELAX TIGHT MUSCLES

Pauwels suggested that osteotomies might lower the joint load by relaxing tight muscles[24]. For example, in a varus osteotomy a tight gluteus medius muscle would be relaxed. This notion was based on the change which occurs in a single muscle and in a single plane.

In our model, the lengths of various muscle elements changed from 0 to 20%; it must be recognized however, that if some muscles are being relaxed others are being stretched (Table 2). It would be difficult to selectively relax only those contracted muscles. Also, while osteotomies might selectively "relax" certain muscles in one position,

Table 2

Percent of Muscle Length Changes From Normal at Heel Strike

Muscle	Normal length (meters	30 Degrees valgus (%)	30 Degrees varus (%)	30 Degrees ante-version (%)	30 Degrees retro-version (%)
Add brevis (s)	0.012	−13	17	4	−5
Add brevis (1)	0.134	−9	10	3	−4
Add longus	0.192	−3	4	2	−2
Add magnus 1	0.100	−15	19	2	0
Add magnus 2	0.196	−2	3	0	0
Add magnus 3	0.359	1	−0	0	0
Gluteus max 1	0.118	14	−11	3	−3
Gluteus max 2	0.143	2	−6	−3	3
Gluteus max 3	0.165	−9	11	−0	1
Gluteus med 1	0.114	13	−11	16	17
Gluteus med 2	0.120	14	−17	5	−6
Gluteus med 3	0.105	13	−16	−7	5
Gluteus min 1	0.094	14	−13	20	−20
Gluteus min 2	0.087	16	−18	13	−10
Gluteus min 3	0.076	13	−17	1	−1

they may not have this same effect during activities of daily living. For example, the gluteus medius muscle changes 30% of its resting length during the gait cycle, and the most extreme osteotomies alter the muscle lengths somewhat less (Figure 3). Thus, the muscle lengths are changed less by osteotomy than by the normal gait cycle.

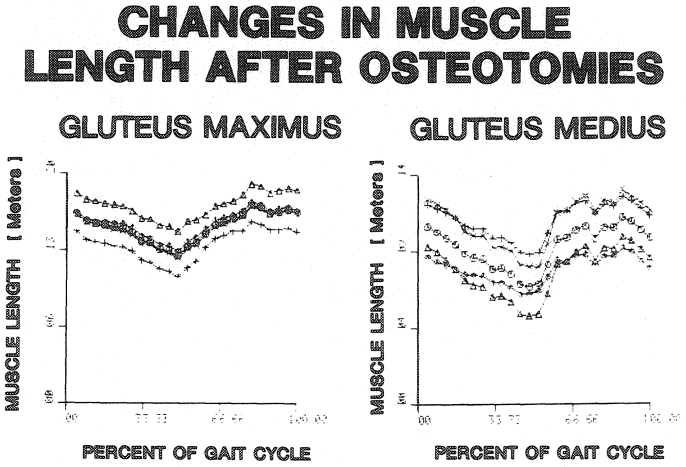

Figure 3:
Changes in muscle length after osteotomies. The 5 lines correspond to muscle length changes during a gait cycle for normal femur (circles) and the four 30 degree osteotomies: + = retroversion, △ = anteversion, x = varus; △ = valgus.

REDUCE MUSCLE FORCES

Pauwels and Bombelli have advocated the notion that muscle forces are altered by moment arm changes occurring with osteotomies[1,2,24]. The typical planar, one-muscle, static model of Pauwels suggests that a varus osteotomy reduces the abductor muscle force due to an increased moment arm and, therefore, reduces the joint force because most of the joint force is created by muscle tension. A valgus osteotomy, by the same reasoning, increases the abductor and joint forces.

Our data, suggests that usually the muscle forces are not significantly affected by osteotomies (Table 3). However, it is not individual muscle forces across the hip joint that are important, but rather their additive forces. This is perhaps the most serious (and nonphysiologic) limitation of simple muscle models.

Table 3

Peak Muscle Forces Before and After Osteotomies*

Muscle	Normal	30 Degrees varus	30 Degrees valgus	30 Degrees retro-version	30 Degrees ante-version
Gluteus min	409	408 (100)	415 (101)	334 (82)	346 (85)
Gluteus med	966	801 (83)	927 (96)	917 (95)	761 (79)
Gluteus max	652	568 (87)	558 (86)	637 (98)	386 (59)
Adductor mag	539	388 (72)	396 (73)	400 (74)	261 (48)

*Peak muscle force predictions are given in Newtons, with percent of normal in parentheses.

REDUCE JOINT FORCES

As a result of reduced muscle forces, Pauwels concluded that the hip joint force would be altered[24]. In the case of a 30 degree varus osteotomy, he suggested that the hip joint load would be reduced by 25%. He also estimated that a 30 degree valgus osteotomy would increase the hip joint load by about the same amount. If in fact the joint loads are increased in valgus osteotomy, and if joint loads alone are responsible for beneficial effects, it is difficult to imagine that increasing the load on diseased cartilage would actually help.

Our data suggests a different conclusion: the joint force is not significantly affected by osteotomy (Figure 4). There are a number of differences between the modeling process of Pauwels and our modeling process, partially explaining the discrepancy in results. One of these differences concerns moment arms. With angulation of the proximal fragment, some moment arms are lengthened while others are shortened. Unless one takes into account all of the changes, the effect is probably not correctly deduced. I would conclude that the mechanical effects of the osteotomy are complex, and that the joint force is not substantially altered. The mechanisms discussed so far do not

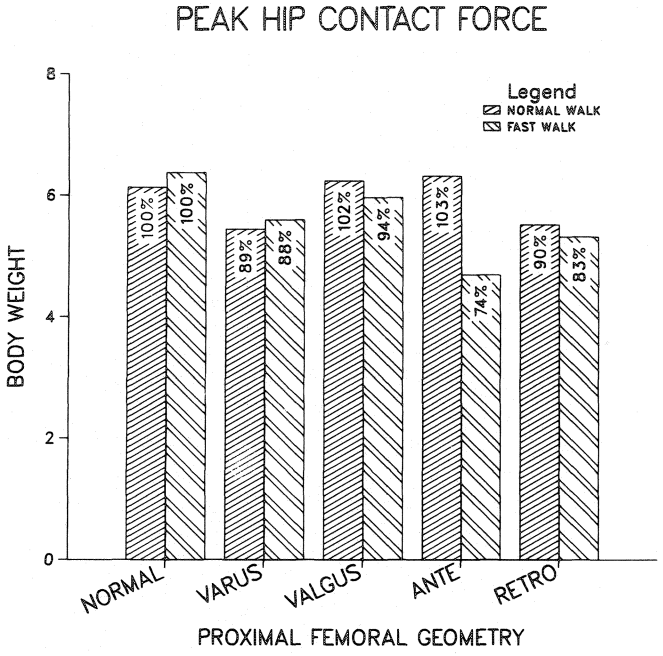

Figure 4:
Peak hip contact forces predicted for the normal femur and the various osteotomies for the two walking speeds. (Illustration reproduced with permission of C.V. Mosby).

account for the beneficial effects of osteotomy.

Collert and Gillstrom share this same opinion:

> "Although the present study was not intended to elucidate the mechanism underlying the effect of the operation, it clearly shows that biomechanical factors are not of crucial importance. Neither medial displacement of the shaft fragment, varus angulation, or tenotomy of the iliopsoas muscle are of any consequence for the long term results. Similarly, none of the other factors analyzed in relation to the effect of pain, i.e., roentgenological type and stage of osteoarthrosis, age, sex, preoperative range of flexion and preoperative contractures made any difference to the results[6]."

REDUCE CARTILAGE STRESS

The next consideration is whether osteotomies decrease the stress in the cartilage by increasing the contact area, ideas held by Pauwels, Bombelli and many other authors[1,2,24]. Studies of hip contact pressure by Rushfeldt et al. as well as Brown and Shaw show somewhat irregular distribution patterns with presumably normal hip joints, and peak pressures ranging from 3-11 megapascals (MPA) and average stresses of 1-4 MPA[5,27,28]. Rushfeldt et al. demonstrated that the interface of calcified cartilage has distinct but subtle asperities (in the range of 0.5 millimeters) which would affect local contact pressures but would not be discernible by radiographs. Studies of contact pressures in degenerated joints have not been attempted, but

it is likely that the irregularities would be greater. It is not likely that hip positions causing beneficial reductions in local contact pressures can be predicted, even if we estimate which positions produce greater "congruence" on radiographs. It seems that osteotomy is as likely to create elevated local contact stresses as it is to reduce them. We have little understanding of the stresses osteotomies usually experience.

STIMULATE BONE REMODELING

Finally, given the new relationship between the proximal and distal osteotomy fragments, the stresses throughout the bone are altered. I am not aware of any theoretical or experimental attempts to define these relationships. However, bone is sensitive to strain, and it is likely that changes in stress-strain patterns will result in bone remodeling. Cases reported by Pauwels dramatically illustrate the beneficial bone remodeling which may occur with osteotomy, as well as restoration of the radiographic joint shadow.[24]

Nissen may have been correct when he said, "The factor responsible would seem to be a mechanical one: Malalignment sufficient to cause a change of stress in the trabeculae of both the head of the femur and the acetabulum and so to stimulate remodeling of the disordered bone towards a normal pattern[22]." Nissen's conclusions have not yet been contradicted. The fact that the results are approximately the same no matter what type of osteotomy is performed tends to support his idea (Table 4). There is little doubt that within a given series factors can be identified which lead to poor results. However, it is likely that for each of those factors, a companion study can be found where the factor did not affect the results.

Table 4

Clinical Results of Proximal Femoral Osteotomy

Principal Author	Numbers of Hips	Follow-up Time (Years)	Satisfactory Results Five Years	Ten Years	Type
Harris[14]	71	4	92%	—	Med
Gudmundsson[13]	92	3-10	71%	—	Med
Goldie[12]	110	9	—	77%	Med
Langlais[16]	150	6	77%	—	Valg
Mogensen[20]	50	10	—	86%	Med
Santore[29]	50	11	—	78%	Valg
Miegel[19]	73	12-15	—	49%	Med
	30	12-15	—	85%	Med
Reigstad[26]	103	10	51%	30%	Med
Zaoussi[30]	70	8	—	70%	Med
Bombelli[2]	212	3-11	90%	—	Varus
Bombelli[2]	471	3-11	88%	—	Valg-Ext

Poss has noted, "A global interpretation of published results is impossible[25]." For the purpose of determining which osteotomies work in certain situations and for certain paitents, this view may apply. However, from Nissen's perspective, the data may show some consistency, at least within our ability to make accurate observations[22]. From his perspective, the results are similar (excepting the study of Reigstad and Gronmark) and independent of the type of osteotomy[26].

Nissen's idea, in my opinion, remains as viable an explanation as any other mechanical hypothesis:

> "The essential stimulus that switches the cellular activity from anarchy over to order would appear to consist of two factors: (1) Complete division of the upper end of the femur, (2) Some change of alignment between the two fragments. These are the factors common to every type of osteotomy in daily use, no matter what master is followed[22]."

It is clear that we have not yet identified the mechanism by which osteotomies work. Several of the hypothesized mechanisms seem unlikely given current data. Since our hypotheses are in part based on our ability to make biological and mechanical observations, and since many of these observations are only on a macroscopic scale, it is entirely possible that we are not observing the relevant behavior. It is important, however, to continue to develop and explore new ideas about how osteotomies function, and then to test those ideas with the means at our disposal.

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ANGULAR DEFORMITIES OF THE LOWER LIMBS IN CHILDREN

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INTRODUCTION

Angular malalignments, bow legs and knock knees, are a common concern in the early years of life. For the majority of children, the problem represents a normal physiologic variation, and spontaneously corrects[12,27,32]. A few will have pathologic malalignment leading to cosmetic or functional problems requiring bracing or surgery. Familiarity with the natural history of angular deformities and familiarity with normal growth patterns are necessary to evaluate malalignment.

PHYSIOLOGIC BOW LEGS AND KNOCK KNEES

Clinical evaluation of angular deformities should include family history, any description of onset and information about progression of the deformity. A child who has assymptomatic findings or a brief history of rapid progression is particularly suspect because these suggest a serious condition such as a neurologic disorder, congenital anomaly, tumor or infection.

The normal infant usually stands with the legs apart, and the subcutaneous fat may mask early physiologic varus angulation. Internal tibial torsion often accompanies physiologic genu varum, and accentuates the bow-leg appearance while standing or walking. Pes planus and external tibial torsion may accompany genu valgum and similarly accentuate the appearance of knock knees.

The child should be observed walking, with attention to the knees during the stance phase to determine if lateral thrust (genu varum) or medial thrust (genu valgum) occurs (Fig. 1). Children with physiologic varus or valgus angulation at the knees typically do not exhibit a thrust. However, in pathologic conditions a thrust is often present suggesting incompetence of the knee ligaments. Ligament incompetence increases the potential for continued progression of the deformity[37].

Figure 1

Child with unilateral bow leg, demonstrates the lateral thrust at stance phase. The weightbearing force is transmitted through the medial tibiofemoral compartment, slowing the growth of the medial tibial physis.

The natural history of physiologic genu varum and genu valgum has been defined by Salenius and Vankka[32]. They studied the development of the tibiofemoral angle in 1480 normal children (Fig. 2). The tibiofemoral angle within the first year is 15° of varus. As the child approaches 18 months, the angle gradually increases to neutral, and the lower extremities appear straight. During the second and third years, the tibiofemoral angle increases to an average of 12° of valgus. During the following years, the valgus alignment slowly decreases to that of the adult, 7° in male and 8° in females. When obtaining an AP roentgenogram of the knees to measure the tibiofemoral angle, the legs must be positioned in neutral rotation; external rotation will decrease valgus deformity and internal rotation will increase valgus angulation[18].

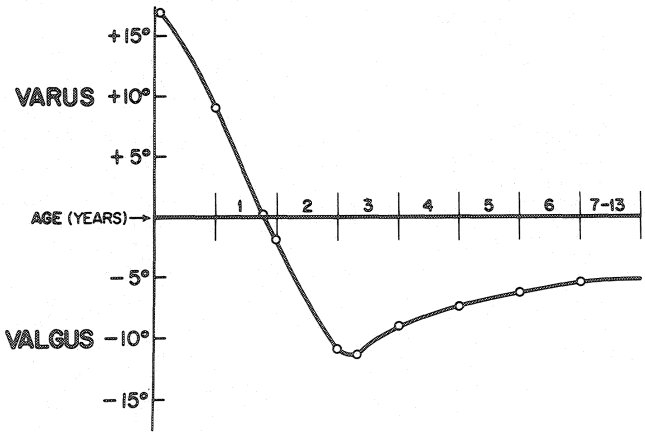


Figure 2

Development of the tibiofemoral angle during growth. Reproduced with permission from J. Bone and Joint Surg., 75A:259-261, 1975.

In the past, authors have recommended using the distance between the femoral condyles to measure genu varum and the distance between the malleoli of the ankles to measure genu valgum[27,30]. These measurements have never been shown as accurate or reproducible and do not allow for the normal variation in leg length.

DIFFERENTIAL DIAGNOSIS OF GENU VARUM

In the young child, Blount's disease is the most common cause of pathologic genu varum. However, the child should be evaluated to exclude other causes such as metaphyseal dysplasia (Fig. 3), osteochondromatosis, hemihypertrophy, fibular or tibial hemimelia, multiple epiphyseal dysplasia, osteochondrodystrophy, achondroplasia and fibrous dysplasia[16]. Trauma or infection involving the physis or epiphysis and fracture of the adjacent metaphysis can also result in varus deformity. Bone softening conditions such as rickets can cause varus or valgus knee deformities. Usually the direction of the deformity is dependent on the child's alignment at the onset of the condition. Thus, the various forms of rickets can be associated with either genu varum or genu valgum. Onset of vitamin D deficiency or vitamin D resistant rickets during the "bow-leg phase" will lead to more pathologic bowing. Genu valgum is typically associated with renal osteodystrophy because the onset of chronic renal disease generally occurs while children are in the valgus phase. Metabolic conditions such as rickets affect the entire epiphyseal plate, while Blount's disease involves only the medial aspect of the proximal tibia.

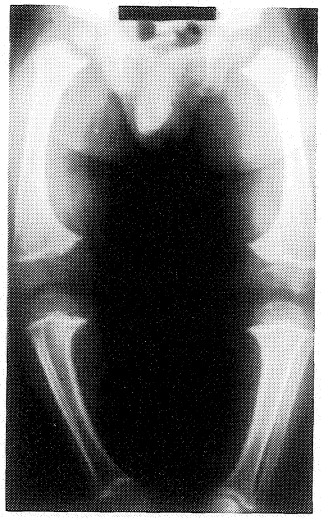


Figure 3
Three year old with metaphyseal dysplasia. Findings in the proximal tibia are similar to Blount's disease, however, similar changes are found in the other metaphyses of the lower extremity, suggesting a generalized condition.

Hypophosphatasia is an inborn error of metabolism characterized by low alkaline phosphatase activity. There are many forms of this condition, and the severely involved child is not likely to survive infancy. Less severely involved children have radiographic findings similar to vitamin D resistant rickets. Children with hypophosphatasia, renal rickets and nutritional rickets have osteopenic bone while patients with vitamin D resistant rickets are likely to have normal bone density.

The radiographic appearance of the epiphyseal plate in metaphyseal dysplasia is similar to rickets (Fig. 3). These children have varying degrees of involvement from markedly disturbed metaphyses, to mild involvement of the distal femur or proximal tibia.

BLOUNT'S DISEASE

Blount's disease, osteochondrosis deformans tibia, was initially described by Erlacher in 1922 and the first large series was reported by Blount in 1937[5,13]. Blount described both an infantile and an adolescent form. The infantile form is usually bilateral, progressive, associated with internal tibial torsion, and appears as a pathologic continuation of physiologic genu varum. The adolescent form is less common, occurring in older children (9-14 years of age), typically unilateral and less often associated with internal tibial torsion. Both forms are more common in blacks[2,3,10,15].

Infantile tibia vara is a developmental problem characterized as an abrupt angulation of the medial tibia, due to a disturbance in growth of the posterior medial portion of the proximal tibial epiphysis. In the advanced stages of this condition there is an associated depression in the posterior medial portion of the proximal tibia[36]. Pathologic reports are sparse but demonstrate irregular cartilage columns, scattered areas of hypertrophic chondrocytes, hypocellular fibrocartilage and delayed irregular ossification.

There is disagreement as to the etiology of the condition. Langenskiold and Riska, and Blount all believed that it is caused by disturbance in growth and ossification of the medial part of the proximal tibial epiphysis and metaphysis[5,6,24]. However, it is not due to avascular necrosis, infection or nutritional factors[6,14,21,24]. There are only scattered reports of familial bowing[33,34,41]. Others suggest that the problem develops as a result of early walking when physiologic genu varum is maximal. The weight bearing force is then transmitted across the medial tibiofemoral compartment slowing the growth of the medial tibial physis[1,2,3,4,9,16,19]. If a lateral thrust has developed with weight bearing (Fig. 1) in a child with genu varum, the prognosis for improvement is poor[37]. Burke developed a biomechanical model that demonstrates that in the 2 year old, 20 degrees of varus results in sufficient force to retard epiphyseal growth. In the 5 year old, only 10 degrees is needed to retard epiphyseal growth[7].

Langenskiold and Riska described six stages of roentgenographically visible progression based on epiphyseal depression and metaphyseal fragmentation of the proximal medial tibia[24]. The children were grouped by radiographic appearance (stage I through VI), depending on the degree of epiphyseal depression and metaphyseal fragmentation (Fig. 4). Medial fragmentation of the proximal tibial metaphysis is the minimal finding for stage I (Fig. 5A). Isolated tibial bowing or beaking of the metaphysis is not considered diagnostic[3,9]. As the condition progresses, an increased varus angulation is noted just distal to the proximal tibial epiphyseal plate (Fig. 5B). The epiphyseal line medially becomes irregular and the epiphysis slopes medially (Figs. 6A-B, 7A-B). As tensile forces increase laterally, there is often an accompanying widening of the epiphyseal plate and tilting of the epiphysis[7].

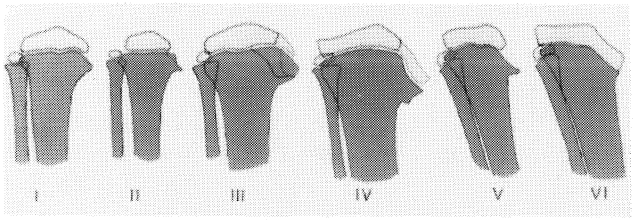


Figure 4
Schematic drawing of the Langenskiold classification, depicting the six stages of progressive radiologic change of tibia vara from mild to severe. The higher stages have more medial deformity and fragmentation.

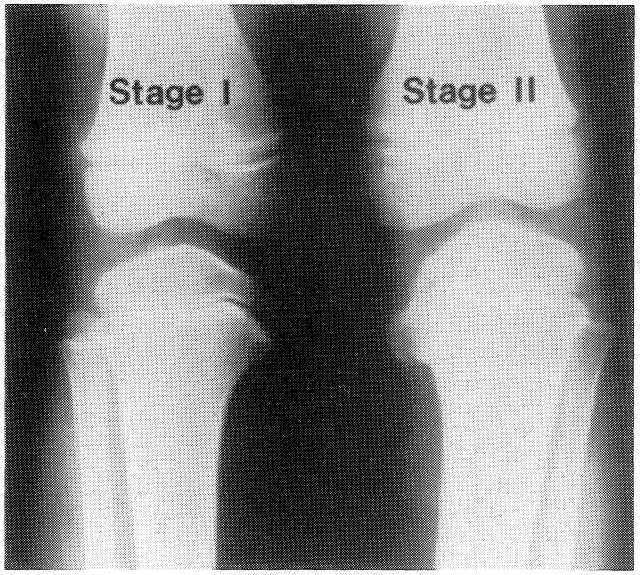


Figure 5A-B
(A) Radiograph of Langenskiold stage I demonstrating mild blunting of the medial tibial epiphysis and beaking of the proximal medial tibial metaphysis.
(B) Stage II demonstrates continuation of the process with radiographic fragmentation and radiolucencies in the medial proximal tibial metaphysis.

It is difficult to distinguish between the initial stage of infantile Blount's disease and the extreme physiologic genu varum[9]. Drenan suggested the use of the metaphyseal-diaphyseal angle to predict which children will progress from severe physiologic bow legs to Blount's disease[25]. On an anterior-posterior roentgenogram, a line is drawn perpendicular to the longitudinal axis of the tibia and another is drawn through the two beaks of the metaphysis to determine the transverse axis of the tibial metaphysis. The metaphyseal-diaphyseal angle is the angle bisected by these two lines. Angles less than 11° tend to resolve, while those greater than 11° progress to Blount's disease (Fig. 8A-B).

If Blount's disease progresses to stage VI, the medial epiphyseal plate will fuse (Fig. 7 B). It is extremely important to recognize this radiographic transition from stage V to stage VI. Once the patient progresses to stage

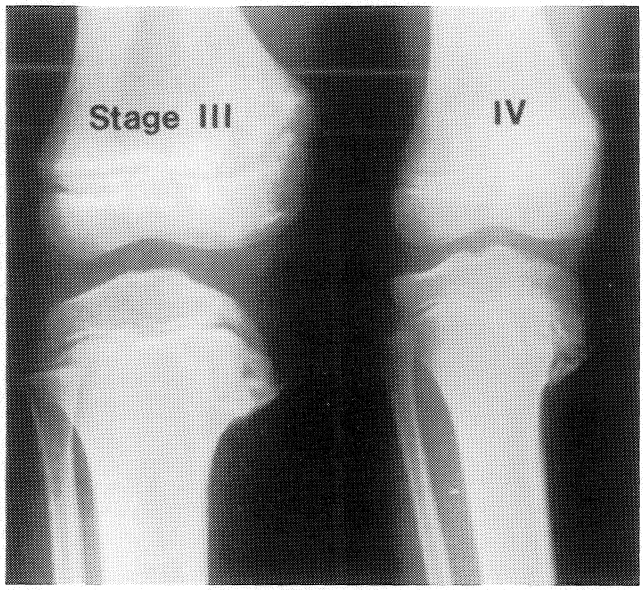


Figure 6A-B
(A) Langenskiold stage III. Note the sloping of the medial epiphysis. The metaphysis demonstrates further dissolution, fragmentation and collapse.
(B) Langenskiold stage IV. Sloping and further fragmentation of the proximal tibial metaphysis. Note the disappearance of the medial portion of the tibial epiphysis.

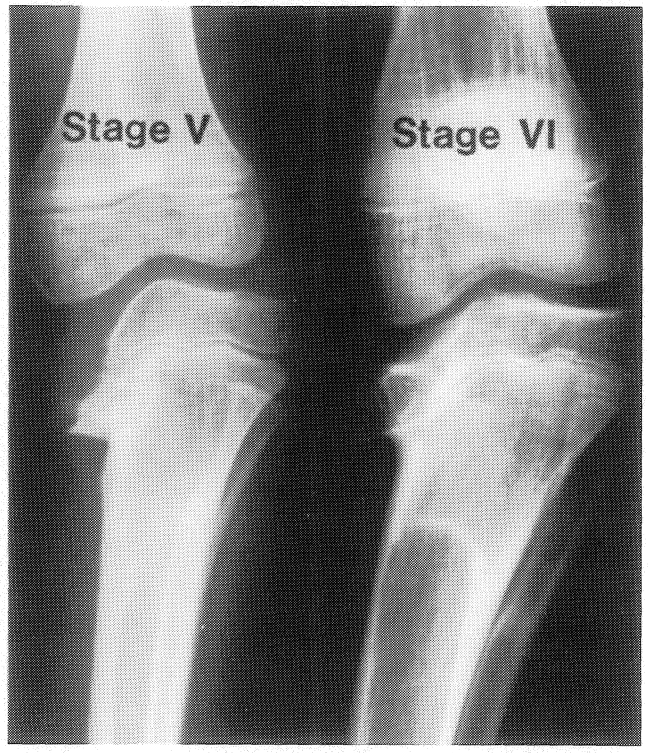


Figure 7A-B
(A) Langenskiold stage V. Complete absence of the medial tibial epiphysis. It is difficult to tell without laminagraphic view if the epiphyseal plate is open.
(B) Langenskiold stage VI. Closure of the medial part of the proximal tibial epiphyseal plate with a bone bridge evident radiographically between the two structures.

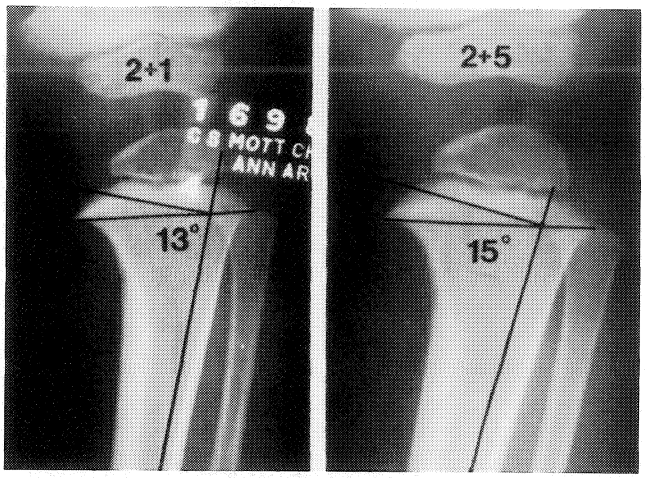


Figure 8A-B
The metaphyseal-diaphyseal angle.
(A) A 2 year 1 month old demonstrating a 13 degrees metaphyseal-diaphyseal angle suggestive of early Blount's disease.
(B) Same patient at 2 years 5 months, demonstrating continued progression to 15 degrees.

VI, surgical realignment will fail and recurrence of deformity is inevitable. As the disease advances through stages II-V, the normal epiphyseal enchondral ossification is inhibited[9,36].

TREATMENT OF BLOUNT'S DISEASE

Treatment of Blount's disease depends on the degree of deformity and the age of the patient. Infantile Blount's disease usually progresses rapidly during the first four years and then slowly during the remainder of growth[24]. Treatment is seldom required in patients younger than 18 months. If the varus deformity is not improving by 18-24 months of age it should be treated with a nighttime orthosis which is effective up to the age of three years[5,6,9,15]. These younger children are generally treated with a Blount's brace or A-frame (Fig. 9A-B). The Blount brace utilizes a Denis-Browne bar to correct internal tibial torsion and the knee straps to correct the bowing. The A-frame is similar but of more rigid construction. In the older children, a double upright brace may be worn throughout the day. With brace treatment, most children with stage I or II involvement can be expected to have satisfactory clinical alignment and radiographic resolution of their disease[28].

Children with advanced stage II disease and all children with stage III disease or greater should be considered for surgery. Once the deformity has progressed to stage III or the tibiofemoral angle is more than 15 degrees, further attempts at bracing are unwarranted, and surgery should be recommended[15] (Fig. 10). Similarly, operative correction is indicated in stage II if the deformity is persistent despite adequate brace treatment. The development of ligament laxity, depression of the tibial plateau, or the roentgenographic appearance of impending medial closure

Figure 9A
The Denis-Blount brace utilizes a Denis-Browne bar to correct initial tibial torsion and knee straps to correct bowing.

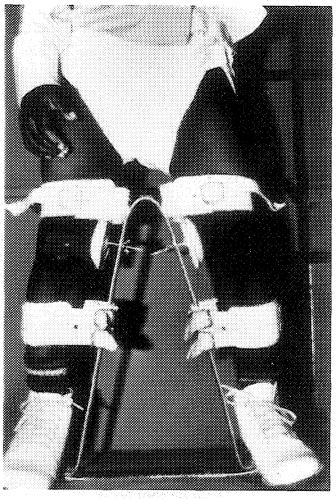


Figure 9B
A-frame design. A-frame is of rigid construction and maintains external rotation and valgus alignment with knee straps.

of the growth plate (stage VI) are definite surgical indications that occur in the later stages of the disease (stage IV-V)[24,27,36].

A valgus external rotation osteotomy performed at an early age will usually obtain complete and permanent correction of the deformity. There is greater chance for recurrence if the osteotomy is done in later years[24,27]. The surgical technique includes a tibial osteotomy correcting the internal tibial torsion, over-correcting the varus deformity, and a fibular osteotomy through a separate incision. If the epiphyseal plate is open, the osteotomy is performed below the insertion of the patellar tendon to avoid damage to the anterior apophyseal extension. The

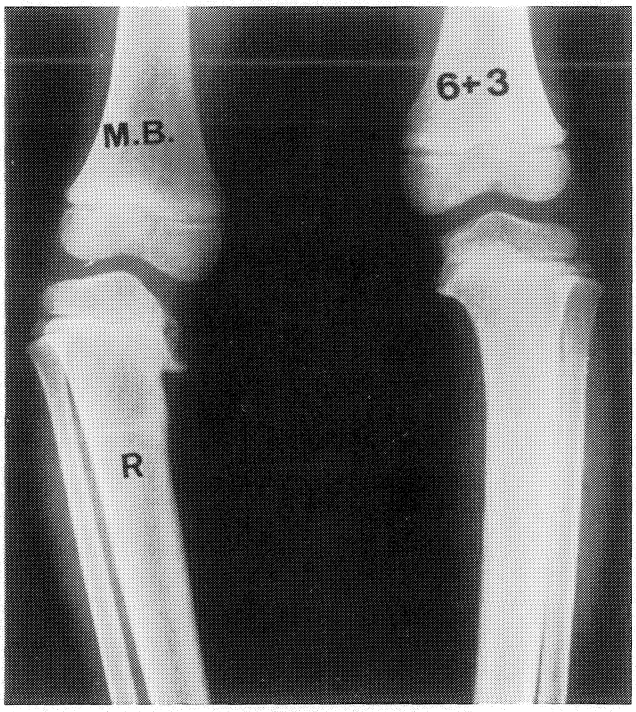
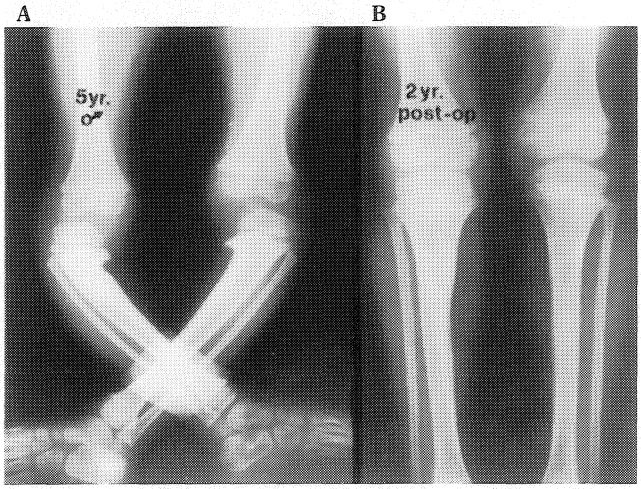


Figure 10
A child aged 6 years 3 months, with bilateral infantile Blount's disease. The involvement is asymmetric with a stage IV on the right and a stage II on the left. Both require surgical correction by osteotomy.

osteotomy may be performed at the level of the epiphyseal plate if the growth is complete or if an epiphysiodesis is necessary. To achieve normal development of the medial epiphysis, the valgus osteotomy must shift the weight bearing forces to the lateral plateau[37] (Fig. 11A-D). Ligament laxity and posterior medial depression of the plateau are frequent causes of inadequate correction and recurrence of deformity[36]. Over-correction is recommended to avoid this problem. Intraoperative roentgenograms or arthrograms are helpful in assuring satisfactory correction of the angular deformity[11]. The internal tibial torsion must also be corrected with the osteotomy. In Langenskiold's series, the osteotomy prevented further progression if done before eight years of age. In our series, we have had several children younger than eight years who required a second procedure and a few who presented with stage VI disease. However, we would agree that younger children with earlier stages of disease are more likely to have a satisfactory result[22,23,28].

We were uniformly unable to prevent stage IV from becoming stage VI with osteotomy alone. Previously, if medial fusion of the epiphyseal plate occurred a final realignment and epiphysiodesis was recommended. However, more recent attempts at epiphyseal bridge resection and interposition of material have met with encouraging results[28]. In children in whom early closure cannot be prevented, closure of the lateral epiphyseal plate and proximal fibula should be accomplished promptly to prevent further angular deformity. A timely epiphyseodesis of the opposite tibia should be considered to minimize leg length discrepancy[20,40,42].

A proximal tibial osteotomy in a child presents a poten-



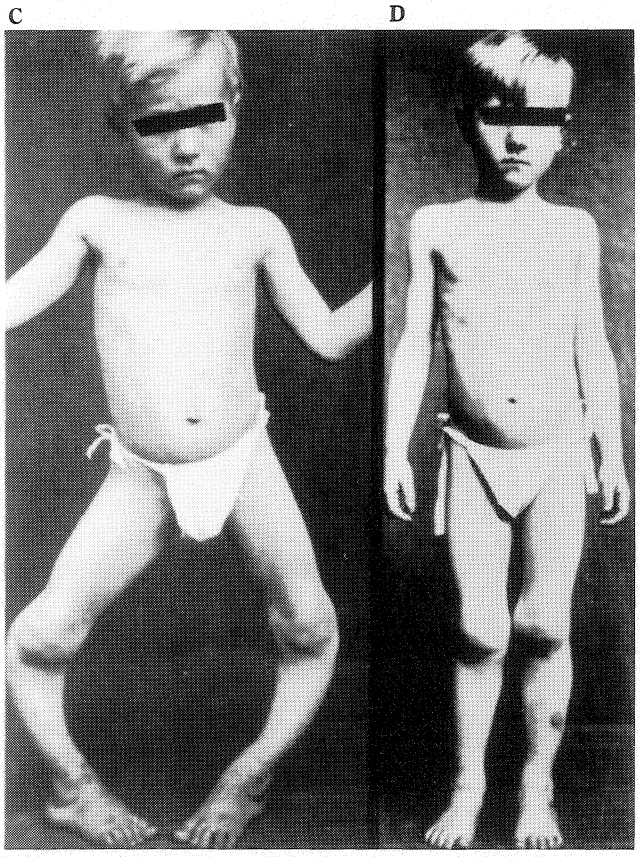


Figure 11A-D
(A)(C) Five year old with advanced Blount's disease (stage III-IV).
(B)(D) Satisfactory radiographic and clinical appearance two years following bilateral tibial osteotomies.

tial hazard and all should be observed carefully for compromise of the neurovascular status of the extremity in the postoperative period[38,42]. Steel and coworkers documented significant compromise of the neurovascular structures of the lower extremities in 20 percent of children undergoing proximal tibial osteotomy and suggested that the problem is due to entrapment of the anterior tibial artery following correction, not to injury of the peroneal nerve[38]. They recommended that if neurovascular compromise occurs following proximal tibial osteotomy, the limb should be immediately returned to the uncorrected position.

ADOLESCENT BLOUNT'S DISEASE

Adolescent tibial Blount's disease is a separate disease from the infantile form[5,6,23]. It is usually unilateral, occurring in an older age group averaging 12.5 years (range 10-14 yrs.), and typically occurs without previous history of bowing. Radiographically, the epiphyseal height is normal and the physis is widened medially and not depressed[21,24,43] (Fig. 12). There is a strong relationship with obesity[40,43]. Also, there is an association of adolescent Blount's disease with slipped capital femoral epiphysis and tarsal coalition suggesting mechanical factors as an etiology[26]. From review of the literature and our own experience, bracing is ineffective in controlling the progression of adolescent Blount's disease[40,43]. Once initiated, the deformity progresses until growth is complete[40,41,43].

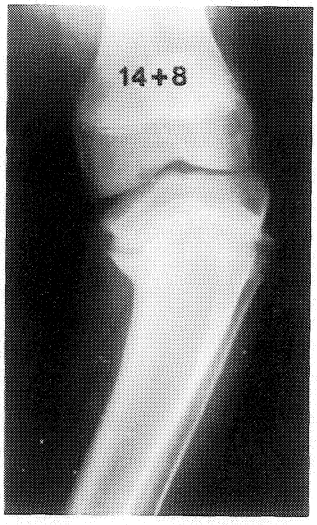


Figure 12
Adolescent Blount's disease in a 14 years 8 months old with typical unilateral evolvement. In addition to bowing other radiographic features include widening of the epiphyseal line medially and the absence of a depressed tibial epiphysis.

Complete resolution of the condition can be expected if the deformity is sufficiently corrected at the first surgery[21,24,40,43] (Fig. 13A-B). If the epiphyseal plate is closed, a proximal tibial osteotomy similar to that described by Coventry can be done through the old epiphyseal scar. If the epiphyseal plate is open, osteotomy below the insertion of the patellar tendon is indicated. This preserves further epiphyseal growth and avoids disturbing the tibial

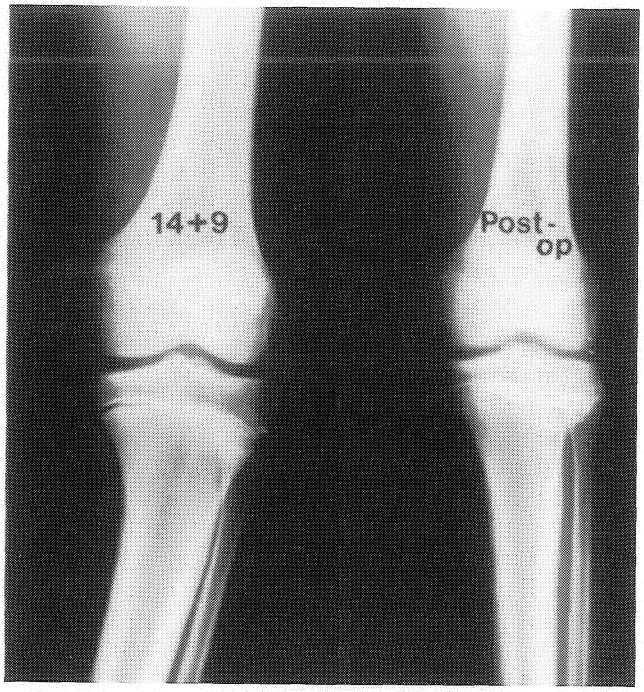


Figure 13-A-B
(A) This 14 years 9 month old has achieved skeletal maturation and no further growth of the proximal tibial epiphysis is anticipated.
(B) The osteotomy was performed at the epiphyseal plate.

apophysis. An epiphyseodesis of the contralateral tibia may be required to prevent leg length discrepancy. Occasionally, as in the infantile form, medial fusion of the epiphyseal plate occurs prior to lateral fusion leading to recurrence of the deformity[20,23]. Neurovascular compromise or fracture of the osteotomy into the joint are possible complications of any proximal tibial osteotomy.

DIFFERENTIAL DIAGNOSIS OF GENU VALGUM

Renal osteodystrophy secondary to chronic renal insufficiency (renal rickets) is now the most frequent cause of pathologic genu valgum (Fig. 14). Improved medical management, general availability of renal dialysis, and kidney transplantation have significantly increased longevity in these children. Thus, correction of these deformities by osteotomy is reasonable and should be performed if severe. Less commonly, obese children can develop idiopathic genu valgum. Additionally, osteochondroma about the distal femur or proximal tibia may cause differential growth leading to a valgus deformity or, less often, varus (Fig. 15).

Direct injury to the epiphyseal plate of the proximal tibia or distal femur (such as a Salter IV or V injury) may result in a late angular deformity. In the younger child, injury to the tibial metaphysis can also cause progressive valgus or late angulation. The unilateral valgus deformity following

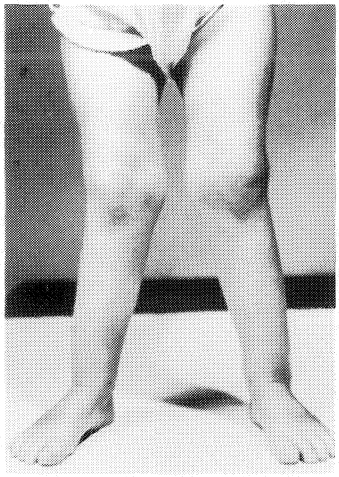


Figure 14
Four year old with genu valgum secondary to renal osteodystrophy.

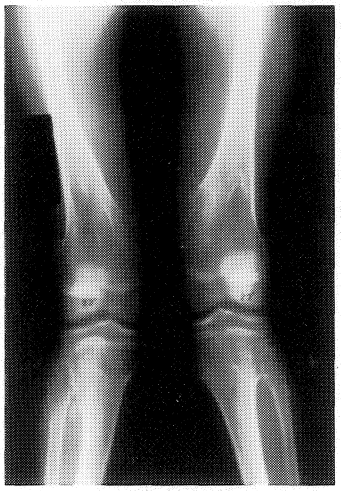


Figure 15
Multiple osteochondromatosis causing bilateral genu valgum.

an undisplaced or minimally displaced fracture of the medial side of the proximal tibial metaphysis desires special emphasis. Jackson and Cozen reported that genu valgum is a potential sequelae of this fracture when sustained in children three to nine years of age[18]. The resultant valgus may be over 15° and is usually recognized after the fracture heals. The development of genu valgum has been attributed to a discrepancy in growth between the tibia and fibula with the fibula exerting a tethering effect[39]. The parents of these children must be informed of this potential complication.

As with genu varum, infections, tumors, congenital anomalies and hereditary conditions such as metaphyseal dysplasia can cause angular deformity. These can generally be identified on initial radiographs (Fig. 16). Paralytic disorders such as cerebral palsy and polio can cause both rotational and valgus deformity due to a tight iliotibial band. This leads to valgus at the knees and external rotation of the tibia.

TREATMENT OF GENU VALGUM

Genu valgum in children younger than seven years of age may be safely ignored unless the tibiofemoral angle is greater than 15°, the valgus is symptomatic or the condition is accompanied by shortened stature[42]. Another exception is the young child with unilateral valgus following proximal tibial fracture. In this case, treatment using a genu valgum brace worn for about one year is usually successful[18]. Howorth believed that by the age of ten a genu valgum deformity greater than three inches (measured between the medial malleoli with the knees together) was not likely to correct with time[17]. Although little support exists in the literature, Howorth believed that excessive genu valgum contributed to awkwardness of gait, subluxing patellae, easy fatigability and joint pains.

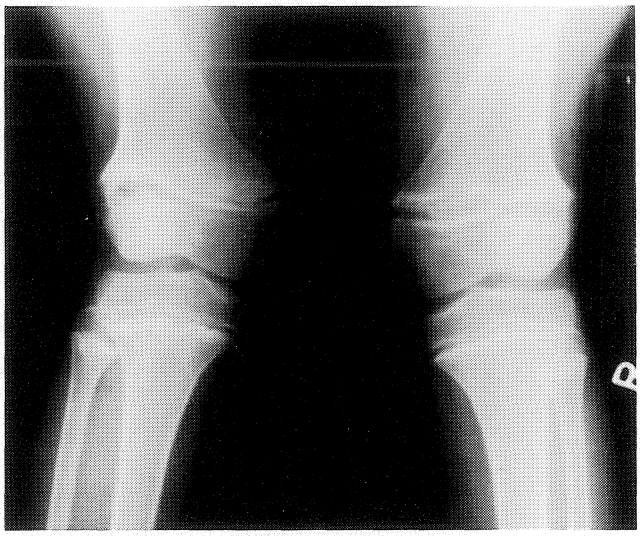


Figure 16
Metaphyseal dysplasia causing genu valgum. These changes are similar to those seen in rickets and renal osteodystrophy.

Corrective surgery may be necessary if the remaining growth potential is limited or the genu valgum is severe. Howorth and others recommended surgical stapling of the medial tibial and femoral epiphysis for correction of excessive genu valgum[17,44]. The amount of correction from stapling is unpredictable since a rebound increase in medial growth occurs when the staples are removed. Zuege et al. suggested allowing for 5 degrees of rebound[44]. Currently, stapling is not recommended prior to a skeletal age of 11 in girls and 12 in boys. The stapling should be timed to coincide with closure of the growth plate. Bowen has devised a simple chart for timing of the stapling procedure to achieve satisfactory alignment at completion of growth[8]. These children should be followed closely because overcorrection may develop. Correction of the valgus deformity that occurs with osteochondromatosis is an ideal use of staples to avoid osteotomy. In these children, the enlarged irregular tibia and femur increase the potential for neurovascular compromise following osteotomy.

SUMMARY

During the early years of childhood, genu valgum and genu varum are common concerns for parents. These problems represent normal physiologic variations in most children. However, a few children will experience pathologic lower extremity malalignment leading to cosmetic and functional deficits. Although several exist, the most frequent causes of pathologic genu varum and genu valgum are Blount's Disease and renal rickets respectively. Treatment of genu valgum and genu varum includes observation for the lesser deformities, bracing for moderate deformities and surgical correction for the excessive deformities.

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GROWTH OF CORNSTALKS AND LONG BONES

DOES LONGITUDINAL BONE GROWTH DEPEND ON A MATRIX DIRECTED HYDRAULIC MECHANISM?

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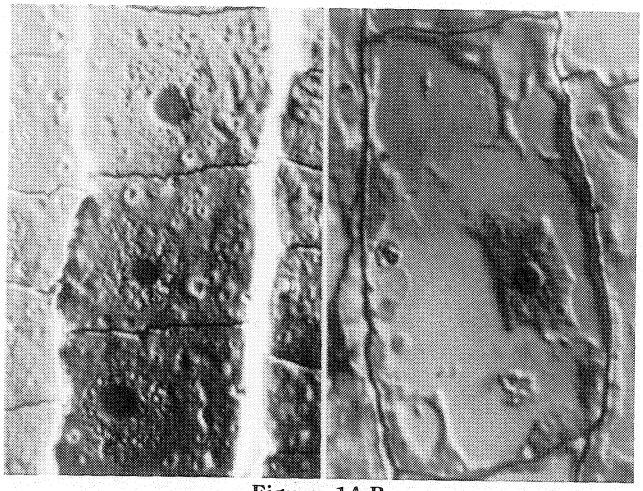
Some Iowans say that on warm, humid, summer nights you can hear the corn grow; that the rapid expansion and elongation of the stalks makes a soft bursting, crackling sound. Those who have never heard it claim that cornstalks can't grow so fast that they make a sound, but no one who has seen the difference in the height of corn between one day and the next denies that cornstalks grow at an astounding rate. Although children's long bones do not grow as rapidly, it seems reasonable to wonder if longitudinal growth of bones and cornstalks depends on the same mechanisms[8,9].

Plant and bone growth result from a controlled or directed increase in tissue volume. Plants increase tissue volume through cell proliferation, cell synthesis of extracellular matrix and through overall cell enlargement. Cells may enlarge by synthesis of cytoplasm and organelles or through accumulation of fluid. Of these mechanisms, fluid accumulation is the most efficient. In terms of metabolic energy requirements, water is "cheaper" than matrix or cytoplasm and does not need maintenance. If rapid plant growth depended on cell proliferation along with the synthesis of cytoplasm, organelles, and cell wall matrices, the process would require large amounts of energy for the production of complex macromolecules and maintenance of the new tissue. Availability of nutrients and the ability of the plant to transport large molecules to expanding tissues would limit the growth rate. Perhaps for these reasons, the majority of volume increase occurs by cell swelling rather than by cell proliferation or synthesis of matrix and cytoplasm[33].

As the plant cells accumulate water within vacuoles, the increasing turgor pressure rapidly expands the cells so they often reach sizes much larger than animal cells. If these cells expanded freely, they would become spheres. Instead, the cell walls direct the rapid increase in cell volume to produce primarily longitudinal growth (Fig. 1A-B). As the cells enlarge, they synthesize and assemble the cell wall matrix which directs their future growth, determines their shape and helps stabilize the tissue. The



Figures 1A-B
Nomarski differential-interference contrast micrographs of corn cells.
Figure 1A shows three corn cells before they swell and Figure 1B shows a single corn cell after swelling. Notice that the corn cell can triple its height without significantly increasing its width.

stiff cell wall cellulose microfibrils commonly lie perpendicular to the axis of cell elongation creating rigid circumferential bands like hoops around a barrel, making the cell grow longitudinally more than horizontally. Localized partial digestion of the cell wall loosens the cellulose microfibril meshwork weakening the matrix in the direction of growth and allows the cells' turgor pressure to expand the cell wall in a specific direction[33]. Completely removing the cell wall by enzymatic digestion allows the cells to expand, becoming spheres called "proplasts". The combination of the turgor pressure of the cells and the rigidity of the cell walls maintain the mechanical stability of the swelling tissue.

At first inspection, longitudinal growth of bones appears more complex than elongation of cornstalks. Bones grow longer and increase in diameter primarily because of increasing volume of the thin cartilaginous growth

plates or physes lying between the bony epiphyses and metaphyses. The transverse growth plate expansion that increases bone diameter occurs at the periphery of the growth plate through addition of new cells and matrix[19]. Bones grow longer by interstitial growth of the physis produced by an elaborate sequence of cell proliferation, matrix synthesis, precise cell and matrix alignment, and changes in cell shape and size[1,2,3,4,7,8,9,16,18,19,20,21,22,24,28,31,35]. As the physeal cells perform these functions, they create the highly ordered structure of the growth plate (Fig. 2). After they have completed their task of increasing growth plate volume, the matrix between cells mineralizes. Metaphyseal capillaries then invade the new tissue, and it is replaced by bone. To produce proportionate skeletal growth, the rate of new tissue production by one growth plate must be coordinated with the activity of other growth plates in the same bone, same limb, opposite limb and spine.

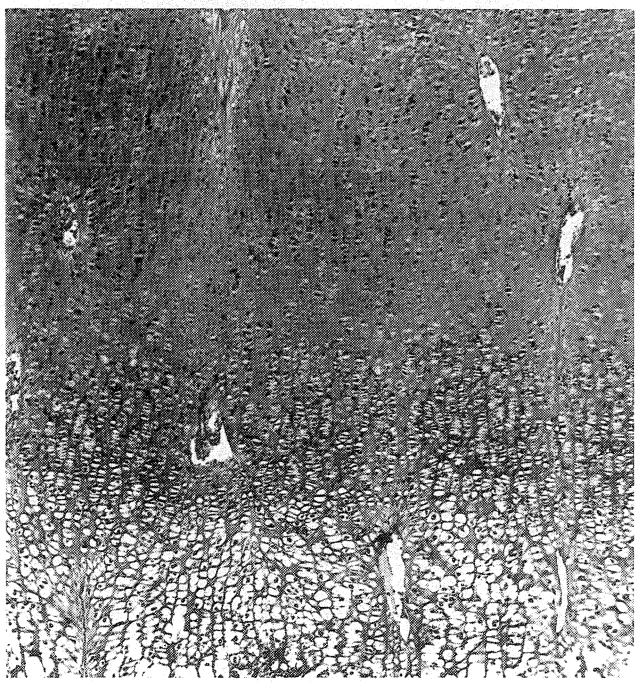


Figure 2
A light micrograph of a fetal bovine rib growth plate. Notice the long columns of proliferative cells oriented parallel to the long axis of the bone, the change in cell shape between the top of the cell columns (upper proliferative zone) and the bottom of the cell columns (lower hypertrophic zone) as well as the striking increase in mean cell volume between the top of the proliferative zone and the bottom of the hypertrophic zone.

Despite the many apparent differences in composition, organization, material properties and function, children's bones and plants both might use the most efficient mechanism of increasing tissue volume: swelling, directed by an extracellular matrix. However, previous discussions of longitudinal bone growth list cell proliferation, matrix synthesis and chondrocyte hypertrophy as the mechanisms of increasing growth plate volume and do not include matrix directed swelling[2,3,4,6,17,18,19,20,21,22,24,28,31]. The longitudinal growth rate of a physis is generally considered to be ". . . a function of the number of cells in the proliferative zone, the average rate of mitosis of these cells and the amount of the matrix with which they surround themselves[28]."

Enlargement of cells following division is commonly referred to as hypertrophy. In general, cell hypertrophy is not considered to be swelling, rather it is defined as a process where ". . . the increased size of cells is due not to an increased intake of water, called cellular swelling or edema, but to the synthesis of more ultrastructural components[27]." True cell hypertrophy increases growth plate volume since enlarging hypertrophic chondrocytes synthesize new organelles[4,8,22], and hypertrophic chondrocytes also accumulate fluid[1,4,16,31]. However, true cell hypertrophy and fluid accumulation have not been identified as primary mechanisms of bone growth[4,8,9,21,22].

Depending primarily on cell division, matrix synthesis and synthesis of cell cytoplasm to increase tissue volume has significant inherent disadvantages. These processes require a considerable supply of energy to produce and maintain new tissue while generating the force necessary to lengthen the bone and soft tissues. Their complexity makes disturbances of growth more likely. Any alteration in cell proliferation, matrix synthesis and assembly, or cellular component synthesis could interfere with longitudinal growth. Furthermore, division of one cell into two does not increase total cell volume; the resultant daughter cells must synthesize new cell components to increase volume. And despite the name usually assigned to them, relatively few proliferative zone chondrocytes continue to proliferate; they might be more appropriately called flattened chondrocytes[17,24].

Matrix and cell component synthesis are inefficient and complex methods of increasing tissue volume; however, this does not prove that physeal swelling is the primary mechanism of bone growth. But, recent evidence suggests that a water driven, matrix directed mechanism, similar to that responsible for the growth of plants, may make an important contribution to increasing growth plate volume. A study of mouse growth plates showed that growth plate chondrocyte enlargement is not true cell hypertrophy[8,9]. In this study, synthesis of new cell organelles contributed only a small part to chondrocyte enlargement, the primary mechanism of increasing cell volume was swelling. Figures 3 and 4 show that as physeal chondrocytes enlarge, they do not increase their organelles or cytoplasm proportionately to fluid accumulation. Cell swelling caused approximately 90% of the more than sixfold increase in chondrocyte volume between the upper proliferative zone and the lower hypertrophic zone while production of new cell organelles added only about ten

percent[7,8]. Thus, it would be more appropriate to call the enlarged chondrocytes swollen cells rather than hypertrophic cells.

As the cells swell, they change their shape so that the expansion of cell volume increases their height rather than their width (Figs. 3). Before they begin to swell, they assume a flattened ellipsoid shape with the major axis perpendicular to the direction of the longitudinal bone growth and a width to height ratio between four and eight[7]. In addition, they align themselves in longitudinal columns of cells separated by longitudinal columns of matrix containing transphyseal collagen fibrils[33] (Fig. 2). After the cells achieve their maximum width, they begin to increase their height and assume a more spherical shape. Because of the pressure of adjacent cells, they eventually become polygonal with a width to height ratio close to one.[7] After reaching maximum size, the cells die, condense rapidly and empty the lacunar space before metaphyseal capillary sprouts penetrate the transverse septum of territorial matrix and enter the lacunar space[14,29,30].

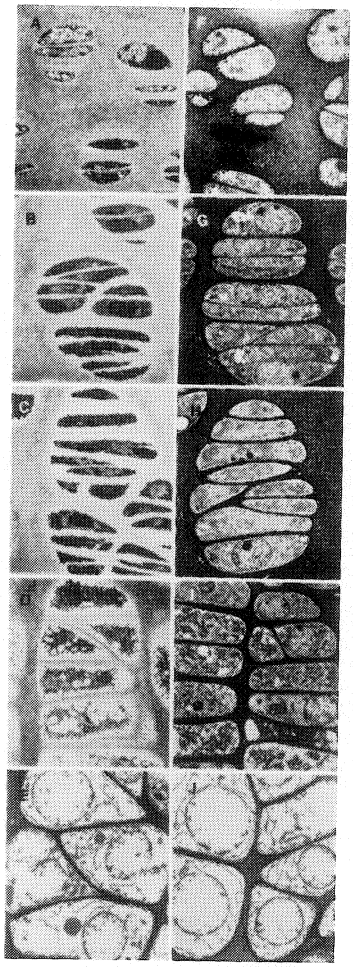


Figure 3A-J
Electron micrographs of longitudinal sections through the proximal tibial growth plate of a fifteen-day-old mouse. A and F, reserve zone. B and G, upper proliferative zone. C and H, lower proliferative zone. D and I, upper hypertrophic zone. E and J, lower hypertrophic zone. Note the increase in cell height with little or no increase in width and the great increase in cell volume between the upper proliferative zone and the lower hypertrophic zone. (A through E, conventional fixation; F through J fixation with ruthenium hexamine trichloride) (X 1000). (Reproduced from J. Bone and Joint Surg., 67-A:942-955, 1985.)

Not only do the physeal chondrocytes swell but their territorial matrix swells[8,9]. Figure 6 shows the marked decrease in collagen content of the territorial matrix between

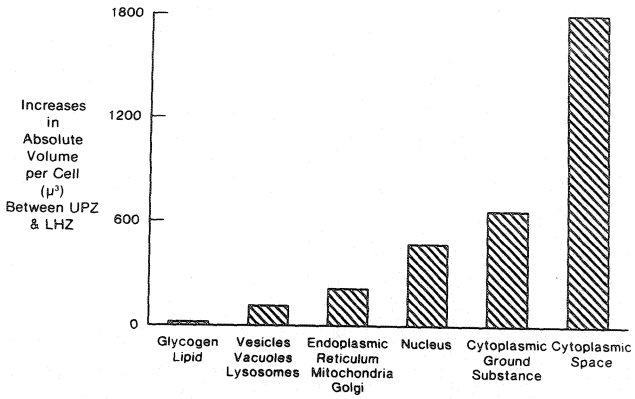


Figure 4
Histogram showing the increases in the absolute volume of cell components per cell from the upper proliferative zone (UPZ) to the lower hypertrophic zone (LHZ). (Reproduced from J. Bone and Joint Surg., 68-A:243-255, 1986.)

the upper proliferative zone and the lower hypertrophic zone. Figure 6 shows the increases in mean cell volume, mean territorial matrix volume per cell and mean interterritorial matrix volume per cell between the upper proliferative zone and lower hypertrophic zone. Synthesis of nonfibrillar collagens and proteoglycans may contribute to the expansion of the territorial matrix, but the relatively constant absolute collagen content as the total volume increases as well as the appearance of the matrix strongly suggests that accumulation of fluid causes most of the nearly fifteen-fold increase in the territorial matrix volume per cell. Meanwhile, the increasing absolute collagen content of the enlarging interterritorial matrix (Fig. 6) as well as the appearance of this matrix (Fig. 5) suggest that most of the increased volume of the interterritorial matrix micromolecules results from accumulation of new matrix. However, if matrix synthesis causes 100 percent of the increased volume of the interterritorial matrix and swelling causes 90 percent of the increased cell and territorial matrix volumes, then swelling produces 70 percent of the total increase in growth plate volume between the upper proliferative zone and the lower hypertrophic zone.

These observations are based on the study of the growth plate of one species at one age, but they suggest that cell proliferation, matrix synthesis and synthesis of cell cytoplasm produce and orient physeal cells and matrix in the uppermost proliferative zone so that swelling of chondrocytes and territorial matrix will produce longitudinal growth[8,9]. The sequence of cell activities and the structure of the physis are consistent with this hypothesis. Soon after cell division, the chondrocytes increase their width by synthesis of cytoplasm and organelles, assume a flattened shape with their major axis perpendicular to the direction of longitudinal bone growth, align themselves with a cell column, and begin to synthesize matrix (Fig. 2 and 7). Shortly thereafter they begin to swell and cause

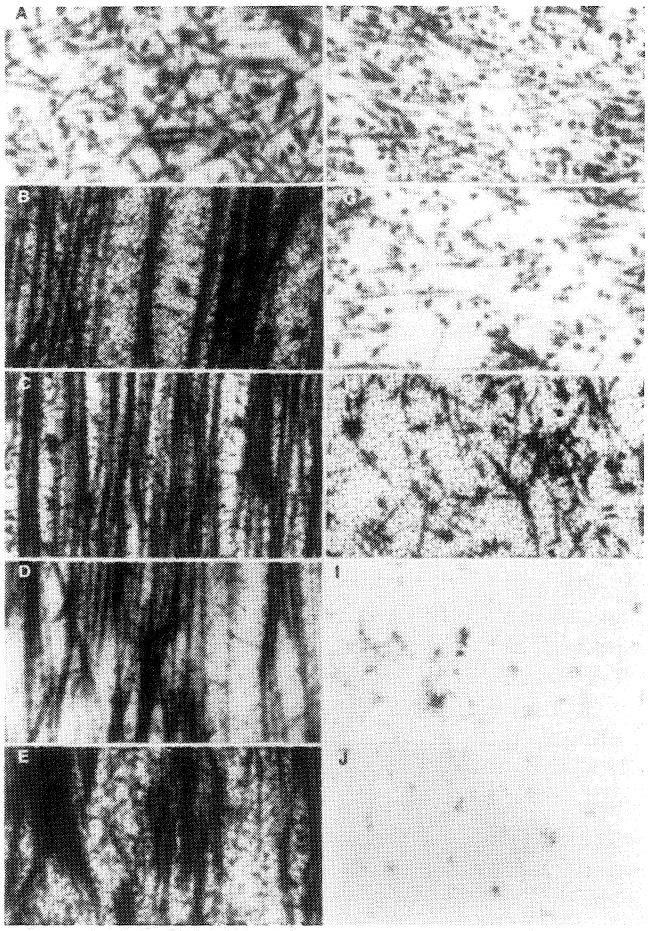


Figure 5A-J

Electron micrographs of longitudinal sections through the proximal tibial growth plate of a fifteen-day-old mouse. A through E, interterritorial matrix: A, reserve zone. B, upper proliferative zone. C, lower proliferative zone. D, upper hypertrophic zone. E, lower hypertrophic zone. F through J territorial matrix: F, reserve zone. G, upper proliferative zone. H, lower proliferative zone, I upper hypertrophic zone, and J lower hypertrophic zone. Note the decrease in territorial matrix collagen content between the upper proliferative zone and the lower hypertrophic zone (conventional fixation).

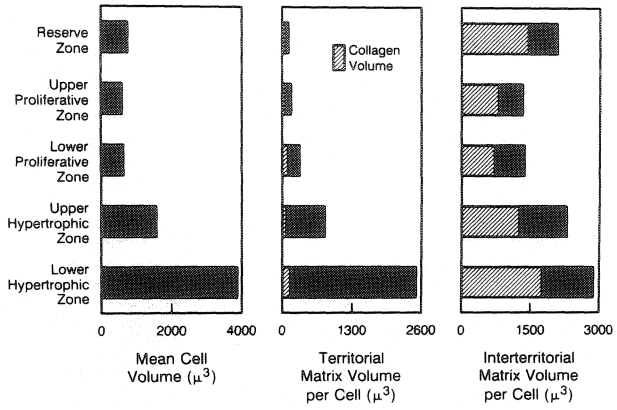


Figure 6

Histograms showing how mean cell volume and matrix volume per cell increase between the upper proliferative zone and the lower hypertrophic zone in the proximal tibial growth plate of the fifteen-day-old mouse. Note that in the territorial matrix collagen volume per cell does not increase as the total volume of the matrix per cell increases while in the interterritorial matrix collagen content per cell increases as the total volume of the matrix per cell increases.

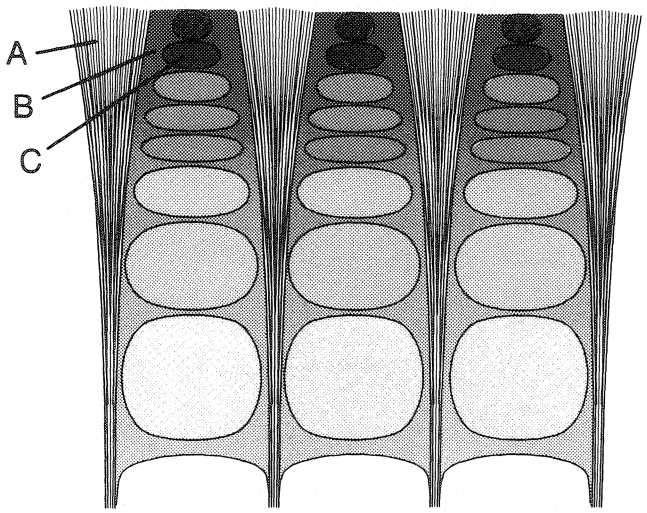


Figure 7

A schematic diagram showing how swelling of cells and territorial matrix, directed by the longitudinal collagen columns of the interterritorial matrix contained within the circumferential perichondral ring (not shown), could produce longitudinal bone growth. To illustrate the directed swelling effect the cell columns are shortened and begin distal to the level where the chondrocytes divide. A, Transphyseal collagen columns of the interterritorial matrix. B, Territorial matrix. C, Chondrocyte. The density of the cells and territorial matrix decrease from the upper proliferative zone to the lower hypertrophic zone to indicate increasing water content. The density of the transphyseal collagen columns remains constant to indicate their relatively constant collagen concentration.

their territorial matrix to swell. Rates of matrix synthesis and cell division correlate with the rate of longitudinal bone growth[28] because these processes create the structure that makes matrix directed swelling possible. Like the arrangement of plant cell wall cellulose, microfibrils direct the swelling of a plant cell, the transphyseal collagen columns of the interterritorial matrix and the surrounding perichondrial-periosteal ring[32] may direct the increasing volume of the chondrocytes and territorial matrix to elongate bone (Figure 7). The transphyseal columns may help maintain the longitudinal alignment of cell columns and the perichondrial-periosteal ring may restrain lateral expansion of the swelling physis creating longitudinal bone growth. The chondrocytes may synthesize and assemble the matrix column before they begin to enlarge. As they enlarge, they may add new, more highly oriented collagen fibrils and condense the previously synthesized fibrils to form the transphyseal collagen columns. As in plants, turgor pressure combined with an ordered fibrillar extracellular matrix could give the rapidly growing physis mechanical

stability. Swelling of the territorial matrix may not only contribute to increasing growth plate volume, it may also facilitate vascular invasion and chondrocyte swelling. Sprouts from metaphyseal capillaries may penetrate an edematous matrix more easily than a matrix filled with densely packed collagen fibrils, and an edematous matrix lacking a tightly woven fibrillar collagenous meshwork would not resist rapid cell swelling.

A recent study of growth plates treated with EDTA, a chelator of divalent cations, provides evidence that growth plates have tremendous capacity for swelling[11]. Growth plate slices immersed in solutions containing EDTA swelled more than epiphyseal, nasal and manubrial cartilage subjected to the same treatment. Addition of divalent cations (Ca, Mg, Zn) to the solution or extraction of growth plate proteoglycans prior to EDTA treatment prevented swelling. Immersion of growth plates with their metaphyses attached in the EDTA solutions resulted primarily in longitudinal swelling. These results show that growth plates have greater potential for swelling than other cartilages and that the interaction of matrix proteoglycans with water produces most of the swelling. The perichondrial-periosteal tissues can help direct the swelling in a longitudinal direction, and the concentration of divalent cations in the growth plate may help control swelling.

Once chondrocyte swelling begins, the cells reach their maximum size quickly and can do so without causing a dramatic change in the total growth plate water content. Careful measurement of the changes in growth plate cell volume has shown that in a column of 34 cells, cell volume remains relatively constant between cell one and cell 20, increases slightly between cell 20 and cell 27, nearly doubles between cell 27 and cell 28 and increases almost two and one half times between cell 28 and cell 34.[35]. Thus, the significant increases in cell volume occur in a narrow zone consisting of approximately the last 20 percent of the cell column. The overall water content of the cartilaginous growth plate and adjacent epiphyseal cartilage is about 80 percent[20]; therefore, the potential increase in water content of the last 20 percent of the growth plate is relatively small. For example, if the water content of tissue was 80 percent and if the volume of the tissue increased five fold, with 90 percent of the increase due to swelling, the resulting water content of the tissue would be 88 percent. However, in the physis the increase would be smaller; the interterritorial matrix does not swell and as it begins to mineralize its water content decreases.

If normal longitudinal bone growth depends on swelling of chondrocytes and territorial matrix, mechanisms must exist to initiate and control these processes. Cell swelling may result from an injury such as hypoxia that compromises the cells' ability to exclude sodium. However, growth plate chondrocytes do not show the complete pattern of changes found in injured or ischemic cells, and chondrocyte swelling, like plant cell swelling, may be a specialized form of cell growth rather than a result of injury[8,9]. The *in vitro* treatment of the physis with chelating agents suggests that hypertrophic cells or the mineralization of the interterritorial matrix may control the matrix swelling by altering the concentration of calcium and other divalent cations[11]. Plant cells selectively alter their surrounding matrix to allow cell elongation, and chondrocytes may use a similar mechanism[33]. Collagenase activity as well as alterations of proteoglycans have been identified in the hypertrophic zone suggesting that enlarging chondrocytes secrete enzymes and other molecules that modify the territorial matrix by loosening the collagen fibril meshwork, producing swelling[25,26,34] and preparing for vascular invasion[9,10,13]. These studies suggest that swelling chondrocytes perform a series of complex, precisely coordinated activities to increase growth plate volume, synthesize new matrix molecules, prepare the territorial matrix for vascular invasion, and prepare the transphyseal columns for mineralization.

Several studies support the suggestion that chondrocyte swelling and the associated change in chondrocyte shape are not passive, uncontrolled processes. Investigations of longitudinal bone growth in rats and mice showed that slowing of growth was associated with 55 and 70 percent decreases in mean hypertrophic cell volumes respectively[6,21]. The investigation of rat growth plates also showed that an increased rate of longitudinal bone growth was not associated with increased cell proliferation, matrix synthesis or hypertrophic cell volume, but it was associated with increased hypertrophic cell height[21]. A recent meticulous study of bone growth in pigs and rats documented a strong positive correlation between hypertrophic cell volume and the rate of longitudinal bone growth. This suggests that during rapid longitudinal bone growth, swelling makes a major contribution to the increase in growth plate volume[35]. Furthermore, the extent of hypertrophic cell swelling appears to be independent of the cell proliferation rate and the cell death rate, indicating that some other mechanism controls chondrocyte swelling[15,35]. These observations indicate that cell swelling makes an important contribution to increasing growth plate volume, that variations in cell swelling help determine the rate of bone growth and that swelling with associated changes in cell shape and matrix composition are active processes controlled by mechanisms not yet identified.

A tissue that swells without restraint would expand equally in all directions. Therefore, if swelling of chondrocytes and territorial matrix produce longitudinal bone growth the perchondrial-periosteal sleeve surrounding the cartilaginous physis must contain and direct the swelling tissue; also, significant defects in the perichondrial-periosteal sleeve should lead to disturbances of longitudinal growth. Con-

versely, if increased growth plate volume results only from highly ordered cell proliferation and synthesis of matrix, cytoplasm and organelles; it might be less likely to expand equally in all directions, and mechanical containment of the physis would be less important. Examination of the structure of the perichondrial-periosteal physeal sleeve shows that it consists of a dense array of collagen fibrils aligned longitudinally and circumferentially[32]. Polarizing light microscopy shows that the perichondrial ring consists of ". . . circumferential bands of collagen fibers which collectively form a cylindrical collar about the growth cartilage . . ." and the overlying periosteum contains a preponderance of circumferential collagen fibers[32]. This arrangement of collagen fibers closely parallels the arrangement of cellulose fibers in the walls of swelling plant cells, exactly the arrangement that would resist lateral expansion of a swelling structure and cause longitudinal growth. Like the plant cell wall, the perichondrial-periosteal sleeve combined with the swelling pressure of the cartilaginous physis could provide mechanical stability. In addition, defects in the perichondrial-periosteal sleeve may lead to exostoses, that is, lateral growth from the physis and decreased longitudinal bone growth[12,23].

SUMMARY

Matrix directed swelling is a simple, efficient method of producing rapid longitudinal growth. It is the primary mechanism of plant growth and may have a similar role in bone growth. Proliferation, flattening and alignment of chondrocytes, synthesis of matrix, and synthesis of cell cytoplasm and organelles form and organize the internal structure of the growth plate relative to the direction of longitudinal bone growth. Matrix synthesis along with synthesis of cell cytoplasm and organelles increase growth plate volume, but matrix directed swelling, similar to that responsible for rapid growth of cornstalks, may be the primary mechanism of increasing physeal volume, and therefore an important mechanism of longitudinal bone growth.

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ARE WE PERFORMING TOO MUCH SPINAL SURGERY?

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Lumbar spine surgery is a twentieth century phenomenon. Laminectomy was first attempted over 1000 years ago, and attempts at spinal stabilization were made by Hadra in 1886[1]. Lumbar spinal fusion was introduced in 1911 independently by Hibbs and Albee, and the first lumbar disc excision was performed 15 years later[2,3,4]. The era of modern lumbar spine surgery truly began with Mixter and Barr's classic description of the lumbar disc syndrome in 1934. Since that time, and particularly since World War II, the annual rate of lumbar spine surgery has grown dramatically. In 1988, it is important to ask, are we performing too much lumbar spine surgery? This paper addresses that question from the perspectives of epidemiology, success rates of spinal surgery, and the knowledge of what determines success or failure.

EPIDEMIOLOGY

The general public as well as medical professionals have significant misperceptions about low back disease as a health problem. The lifetime incidence of low back pain in American adults is 70 to 80 percent, and the socioeconomic costs are significant in making low back pain (LBP) a serious health problem.

If the epidemiologic data is analyzed more critically, a less ominous sense of LBP as a health problem is obtained, although the resultant psychosocial and legal impact is real. It is estimated that LBP affects five percent of the adult population each year[6,7]. Given a population of 200 million adults in the United States, approximately 9,000,000 individuals are affected annually. The recovery process is illustrated in Figure 1, and shows the majority of these patients are well within two weeks[8]. Table 1 emphasizes that of the original 9,000,000 a significantly smaller number have persisting symptoms, and fewer yet receive an operation. Moreover, this recovery curve can be significantly improved by more aggressive early intervention, in both industrial low back "injury" and in the non-industrial setting. For example, Wiesel et al. have shown that the simple expediency of on-the-job site evaluations by knowledgeable, impartial physicians or computer-based case management can reduce work loss, duration of symptoms and rates of surgery with attendant significant cost saving[9]. Similarly, Cohler studied all individuals with LBP derived from a population of 100,000 residents in Gothenberg[10]. Her treatment protocol included minimal rest, education and aerobic exercise combined with early appropriate specialist intervention. Although the population she studied had a different health care delivery and legal system from the United States, this study also indicates the already favorable natural history of LBP can be improved by simple, well controlled management. Simply stated, LBP does not need to be a serious health problem.

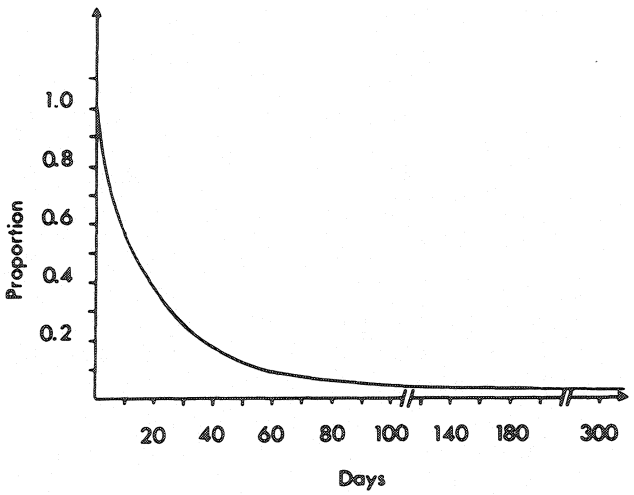

Figure 1:
The recovery from acute low back pain is depicted. Note 50 percent of the patients have returned to function within two weeks, and only five percent remain disabled after three months. (Reproduced from Svensson and Andersson[8] with the permission of the authors and publisher.)

Table I

Low Back Pain Incidence and Recovery	
Annual incidence	9,000,000 (5%)
Recovery	
1 month	5,400,000 (60%)
2 months	7,200,000 (80%)
3 months	8,100,000 (90%)
Disabled after 3 months	900,000 (10%)
Annual surgery	286,000 (2.8%)

EPIDEMIOLOGY OF DISABILITY

In the past ten years, there has been a lot written about the significant socioeconomic impact caused by back pain syndromes. At the present time, there are 5.4 million

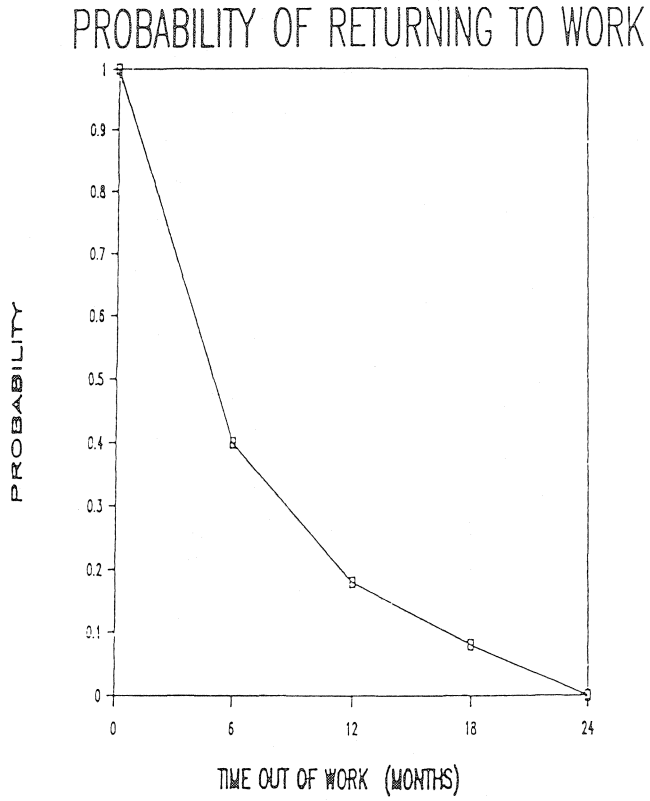

Figure 2:
The recovery, as measured by return to work, is illustrated for the five percent of patients whose disability extends beyond three months. By two years, the possibility of functional restoration is non-existent.

Americans disabled by LBP (NCHS), 2.6 million of them permanently[11]. Moreover, back pain disability is increasing at a rate 14 times greater than the population growth[12]. Workers disabled from LBP account for 85 to 90 percent of the total societal cost of LBP; this is estimated to be between 16 and 60 billion dollars a year. There is compelling evidence that the major determinants of disability are workplace dissatisfaction, a complex and often counterproductive compensation and legal system, psychologic dysfunction and delayed or unnecessary medical interventions[8,15,16,17]. Moreover, the outcome of treatment as measured by return to work, adds to the perception that LBP is an incurable disease, rather than a benign syndrome (Figure 2)[18]. Newer forms of active intervention at the onset of symptoms, predictive models which allow early identification of the person at risk for disability and aggressive rehabilitation programs with up to 85 percent success rate are altering this dismal prognosis[19]. It is also clear that the disability problem in LBP is not favorably influenced by surgical intervention, except in a very small subset of patients.

EPIDEMIOLOGY OF LUMBAR SPINE SURGERY

Surgery plays only a small role in the overall perspective of low back pain (Table 1). Figure 3 demonstrates further how the rates of surgery have increased in the early part of this decade[21]. Lumbar disc excision accounts for three-quarters of the total, while the remaining one-quarter represents spinal fusions and other spinal procedures. Not included in this analysis is the impact of an additional 60,000 to 70,000 chymopapain injections performed in 1985. The question is whether this surgery rate is too high.

First, it is known that approximately one to 20 percent of all back episodes are accompanied by sciatica[8]. Thus, of the 9,000,000 annual LBP episodes, an estimated range of candidates for lumbar spine decompression is 90,000 to 1.8 million. However, it is also estimated that only one to 20 percent of patients with sciatica need surgery. This produces a range of 9,000 to 360,000, or a midrange of 180,000. This approximates the 1984 United States lumbar disc excision rate of 186,000. Cauda equina syndrome is the only absolute indication for prompt lumbar disc excision. It is estimated that between .33 percent and two percent of all patients with known lumbar disc herniation present with cauda equina syndrome[23,24]. Based on calculations that annually 90,000 to 1.8 million patients have sciatica, a range from 300 to 38,000 cases per year could have an absolute indication for disc excision. Obviously, this calculation does not account for patients with a strong relative indication for surgical intervention such as progressive muscle weakness.

Second, we can look at other countries whose work environments are comparable to the United States, recognizing their significant differences in both the social and health care delivery systems. Because other countries use statistics based on the entire population (i.e., children and adults), a United States population of 260 million will be used rather than the previously used adult population of 200 million. Utilizing the 1983 figure of 188,000 lumbar disc excisions, the annual rate of disc surgery is estimated as 71/100,000 while the rate of all lumbar spine surgery, based on 250,000 total operations, is 100/100,000. This figure approximates the rate of 69.5/100,000 calculated for disc excision in 1979[25] (Figure 3). In the United Kingdom, the recent rate for lumbar disc excision was 10/100,000 while in Finland the rate was 41/100,000[25]. It is apparent that there is a significant variation in lumbar disc excision rates among countries while the annual incidence of LBP is comparable.

Third, one can evaluate the regional variations in lumbar spine surgery in the United States. Kane estimated the variations to be as much as ten-fold, with the highest surgery rates in the west coast, and the lowest in the Midwest and Northeast[26]. These differences cannot be explained by variations in the incidence of LBP and sciatica, although there may be regional differences in occupational requirements. Regional variations of operative

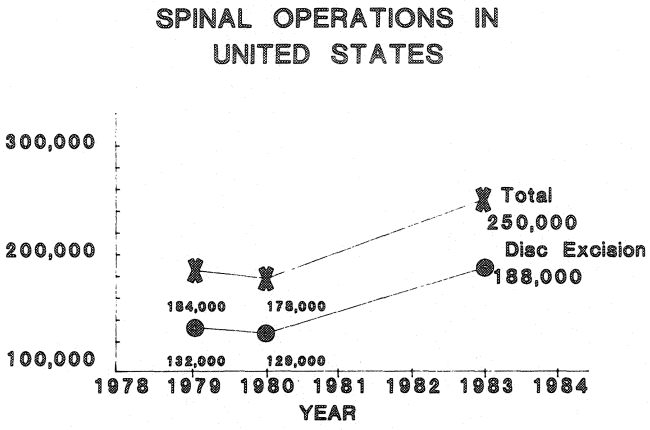

Figure 3:
The rate of lumbar spinal surgery is given including total lumbar operations, as well as operations specifically for lumbar disk herniation. (Adapted from Rutkow[21] and reproduced with the permission of the author and publisher.)

rates within the United States have been demonstrated by Wennberg[27]. In fact, he has shown that within a given state, there may be a ten-fold increase in such elective procedures as hysterectomy, prostatectomy and carpal tunnel decompression. He concluded the major determinant in surgery rate variations was the surgeon's preference, rather than a basic difference in the incidence of disease. In contrast, the rate of operation varies minimally when one treatment is clearly preferred, such as in the surgical management of a fractured hip. Based on these data, it can be concluded that lumbar surgical rates have wide variation within the United States and between countries; there exists no apparent reason for these differences other than health care delivery standards and the preferences of surgeons.

WHAT ARE THE MEASURES OF SUCCESS AND FAILURE, AND WHAT ARE THE CONSEQUENCES OF FAILURE IN LUMBAR SPINE SURGERY?

Many scales have been developed to assess success rates after lumbar spine surgery. These have varied from simple measurements of patient satisfaction to complex rating systems assessing work capacity, treatment utilization and pain. A comparison of 14 different rating systems revealed significant differences in what was termed "satisfactory" surgical results[28]. In this study, a questionnaire applied these 14 different rating systems to the same population of spine surgery patients; the results showed only differences inherent to the rating systems. The rating systems that gave the most pessimistic results were those which heavily weighted return to work, while the scales with the most optimistic results placed heavy emphasis on "patient satisfaction." Therefore, the rating scale selection has significant bearing on the determination of lumbar spine surgery success rate.

Determination of lumbar spine surgery success rates have major socioeconomic consequences. As mentioned earlier, the cost of LBP in the United States is between 16 and 60 billion dollars each year and it is known that 85 to 90 percent of that cost is attributable to those workers with disabling low back disease[29,30]. This subset includes a disproportionate number of individuals with prior low back surgery. For example, in a cross-sectional study of males ages 18 to 55, we found the lifetime incidence of lumbar spine surgery to be 2.5 percent. This incidence increased four-fold when the disabled subset was analyzed. Norton analyzed the cost of lumbar laminectomy and chymopapain for all Oregon workmen's compensation cases treated in one particular year[31]. He found the success rate was 59.4 percent for a laminectomy and 29.1 percent for a chymopapain injection. Based on this particular success rate calculation and a total cost assessment for surgical treatment, the cost per successful result was $49,007 for a laminectomy and $126,610 for a chymopapain injection. Based on the rating system employed by Norton, the cost to society for successful spine surgery is unacceptably high.

WHAT ARE THE DETERMINANTS OF SUCCESS AND FAILURE?

First, the patient must have well-defined pathology. There is ample information that well-defined lumbar spine pathology combines clinical history, objective physical findings and supporting radiographic imaging studies. For example, a patient likely to have a favorable result from lumbar disc excision would have the following: a clear history of sciatica; straight leg raising of less than 30 degrees reproducing sciatica; objective neurologic signs such as reflex, sensory or motor loss; and myelographic, CT or MRI evidence of disc herniation. A similar result can be expected in a patient with neurogenic claudication, degenerative spondylolisthesis and a complete myelographic block. In contrast, the results for surgical intervention are poorer for less well-defined pathology such as "disc disruption syndrome", "degenerative segmental instability" and "bulging lumbar disc" where the confirmatory clinical symptoms, signs and radiographic images are less certain. In fact, surgical success rates have been reported to be as low as 20 percent when the indication was termed "low back pain." This situation is further complicated by the presence of disc degeneration, disc bulge and facet degeneration in 30 to 40 percent of individuals who have never had low back pain or sciatica[32,33,34].

Second, the operation performed must conform to the pathology being treated. Probably no more than two to three percent of failures are attributable to gross errors in judgement such as wrong side, operating at the wrong

level, discitis or nerve root injury. More subtle causes of surgical failures are inadequate decompression in patients with lateral recess stenosis combined with disc herniation, or instability produced by extensive decompression[33]. Although there are no clinically certain guidelines, Panjabi has shown that removal of more than 50 percent of both facets, or removal of one entire fact, produces a significant loss of stiffness in all planes of motion.

Furthermore, the notion that there is one best operation for a particular diagnosis (disc herniation, for example) is an unproven hypothesis. There is little evidence that the results of disc surgery are influenced by microsurgical techniques, standard laminectomy, complete disc excision or partial disc removal. However, some subtle variations in technique may have an effect on the outcome such as fat grafts to prevent perineural fibrosis.

Third, the patient must be chosen correctly. All published series strongly implicate patient selection as the greatest determinant of surgical success or failure[35,36]. The determinants of failure are well understood and include compensable injuries and psychologic dysfunction. The latter has been typically identified using the Minnesota Multiphasic Personality Inventory, pain drawings, and other psychologic tests. However, there is evidence from Weber's studies that patients with both psychologic disturbance and unequivocal pathology faired better if they were surgically treated[35].

Fourth, it must be understood that surgery will not alter the normal progression of a degenerative process. Weber's studies have clearly demonstrated that the long-term outcome for lumbar disc herniation is not altered by surgery. In fact, most long-term results demonstrate that failures ultimately result from progression of degenerative process, such as spinal stenosis, segmental instability and recurrent disc herniations[37,38]. The belief that spinal fusion would prevent these problems has not been substantiated. In fact, spinal fusion in cases where the indications are at best equivocal (e.g. as part of the routine treatment of disc herniation), the recognized complications of pseudoarthrosis, spinal stenosis and instability are more difficult to manage[37].

ARE WE PERFORMING TOO MUCH SPINAL SURGERY?

The question, "Are we performing too much spinal surgery?", has been analyzed in this discussion from many perspectives; by any measure, the answer appears to be "yes." Although the data is not complete, the consequences of erroneous decision-making and inappropriate surgery seems to be difficult from a socioeconomic perspective as well as from a human suffering perspective. Given the increasing involvement of government in medical decisions, the challenge to the medical profession is finding a means to reduce inappropriate lumbar spine surgery. If Wennberg's impressions are correct, the major determinant identifying inappropriate surgery is simply an elevated rate of lumbar spinal surgery.

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A PROTOTYPE CYCLE ERGOMETER DESIGNED TO EXPEDITE KNEE REHABILITATION

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INTRODUCTION

We hold the clinical impression that continuous passive motion (CPM) aggravates quadriceps atrophy and may be deleterious to complete knee rehabilitation. This clinical impression was the stimulus for the authors to introduce the concept of continuous active motion (CAM) as a method to gain both motion and strength early in the rehabilitation program of postoperative knee surgery patients.

A cycle ergometer is known to be one of the best rehabilitation devices in muscle development[3]. One limitation which restricts its early use is that at least 90° of knee motion is necessary to turn the pedal an entire revolution. Thus many rehabilitation programs utilize CPM to gain motion in postoperative knee patients[1,6,8]. Our objective was to design a cycle ergometer with a series of shorter cranksets which would allow patients to cycle with less initial knee motion. This device could then be used earlier in the rehabilitation program, adding active exercise to gain quadriceps and hamstring strength as well as knee motion.

MATERIALS AND METHODS

A: Mathematical Model of Knee Motion During Ergometric Cycling

A simple model was created with a patient seated on a cycle ergometer. A triangle was formed with its sides consisting of the femur, the lower leg (knee to the bottom of the heel) and the hip-to-heel distance (Fig. 1). For this model, the heel is assumed to maintain the same relative distance to the hip as the pedal. While cycling, both the femur and lower leg lengths remain constant. The variable length is the hip-to-heel distance. For a specific hip-to-heel distance, the popliteal angle (A) can be calculated using the Law of Cosines:

$$a^2 = b^2 + c^2 - (2bc \cos A)$$

The above equation can be solved for angle A:

$$A = \cos^{-1}\left[\frac{b^2+c^2-a^2}{2bc}\right]$$

$$A = \cos^{-1}\left[\frac{(\text{femur})^2+(\text{lower leg})^2-(\text{hip to heel})^2}{2\ (\text{femur})\ (\text{lower leg})}\right]$$

By convention, knee flexion (K) is defined as the supplement of the popliteal angle:

$$K = 180° - A$$

Thus, knee flexion can be calculated for any given hip-to-heel distance, with fixed femur and lower lengths.

As a patient rides a cycle ergometer, knee flexion changes as the crank turns and the hip-to-heel distance changes. Using the formula above, the excursion of flexion neces-

Figure 1
When a patient sits on a cycle ergometer, the popliteal angle (A) is the supplement of knee flexion (K).

sary to complete a full rotation on the bicycle can be calculated for any individual at any seat height.

A computer program was written to calculate knee flexion using the above formula when the femur, lower leg, as well as minimal and maximal hip-to-heel measurements were entered. The maximal hip-to-heel measurement is the sum of the femur and lower leg lengths with the knee in full extension and the pedal at the bottom of its crank cycle. The minimal hip-to-heel distance is the maximal hip-to-heel distance minus two times the crank length. The hip-to-heel measurements may also be varied by decreasing the maximal hip-to-heel distance by lowering the seat on the cycle ergometer. In this situation, the patient never reaches full knee extension.

Using the standard crank of a cycle ergometer measuring 170 mm as well as modified cranks measuring 140 mm, 110 mm and 80 mm and varying the height of the ergometer seat, a range of motion profile can be computed for any given patient. For a 178 cm (5 foot 10 inch) man with a femur length of 47 cm and a lower leg length of 52 cm, 98° of knee flexion is required to use the standard 170 mm crank when starting from a position of full knee extension. By shortening the crank to 140 mm, the required knee flexion is 88°. A 110 mm crank requires 78° of flexion and an 80 mm crank requires 66°. Therefore, by shortening the crank 90 mm (from 170 mm to 80 mm), the knee flexion required to cycle is 32° less. The range of knee flexion necessary to cycle with the seat lowered by 3 mm intervals can also be determined, thus creating an individualized range of motion profile for any patient with varying crank lengths and seat heights. A range of motion profile is depicted in Figure 2 for the previously mentioned 178 cm man.

B: Construction of a Custom Cycle Ergometer

A model W2 Tunturi cycle ergometer was obtained with the conventional one piece 170 mm crank[9,10]. A Shimano Dx Conversion Bottom Bracket Spindle allowing for exchangeable crank arms was installed. Four 170 mm Shimano #105 cranksets and pedal assemblies were obtained. One was left intact and the other three were cut, redrilled and tapped to make crank lengths of 140 mm, 110 mm and 80 mm (Fig. 3). Each crankset had a 52 tooth chainring. The four cranksets could be rapidly exchanged for one another with a Sugino Autex Yone-key release using a 6 mm hex socket bolt.

C: Clinical Protocol

The following protocol has been developed to gain flexion in patients after knee surgery excluding cruciate ligament repair or reconstruction:

1. Approximately 60° of initial knee flexion was required in most patients prior to cycling.
2. The patient's femur and lower leg lengths are measured and entered into the program using a Macintosh Plus computer. An individualized range of motion chart or graph is then printed for each patient.
3. The cycle ergometer is set up with the 80 mm crankset and the seat at full knee extension for that patient. To clear the top of the pedal's rotation, the patient may need to "hip-hike". The hip-hike is a method of clearing the top of the pedal's revolution by lifting the buttocks off of the seat as the pedal reaches its highest position and the knee achieves its maximum flexion. This essentially increases the hip to heel distance and decreases the amount of flexion required. As the patient rides for a few minutes, the hip-hike gradually diminishes as the knee flexion improves.
4. Once the patient becomes comfortable riding without a hip-hike, the seat is lowered 3 cm, thus decreasing the hip-to-heel distance and increasing the necessary knee flexion. Again the patient will hip-hike, then gradually stretch and gain more motion. Step 4 is then repeated.
5. After the seat has been lowered 6 cm, which is the difference in the lengths of the consecutive cranksets, the next larger crank is inserted while raising the seat to the point of full knee extension once again. The arc of motion is now increased with cycling. Steps 3 and 4 are then repeated with the larger crankset.

The use of the custom cycle ergometer in patients having anterior cruciate repairs or reconstruction follows a

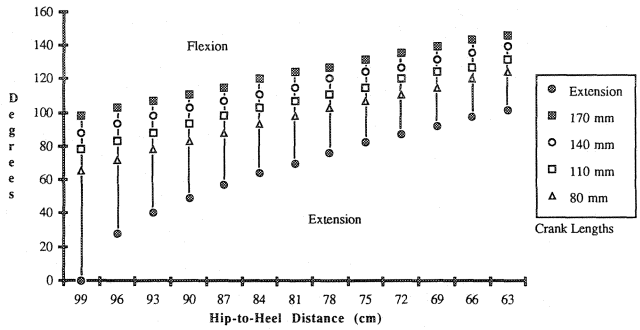

Figure 2
Example range of motion profile for 178 cm man entering the measurements of femur (47 cm) and lower leg (52 cm) lengths.

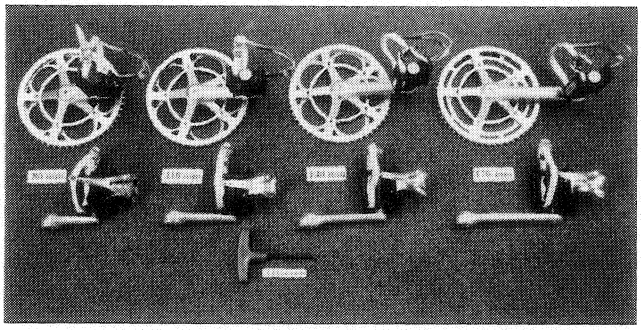

Figure 3
A custom cycle ergometer has cranksets of 80 mm, 110 mm, 140 and the standard 170 mm.

different protocol. In these cases utilizing a smaller arch of motion with the shorter crank lengths becomes important. Early complete function of the quadriceps is not desired due to its action as an ACL antagonist at 20°-50° of flexion[7]. The patient's individual range of motion profile can be used to set the hip-to-heel distance by adjusting the seat height so that the knee is at 50° of flexion when the pedal is at its lowest position. Using the 178 cm man as an example, a hip-to-heel distance of 90 cm will produce 49° of knee flexion (Fig. 2). The total flexion required at this seat height will vary according to the crankset lengths, the standard 170 mm crank requiring 111° of flexion to complete the pedal cycle while the 110 mm and 80 mm cranks require 93° and 83° respectively.

DISCUSSION

The CPM machine and cycle ergometer each have specific advantages and disadvantages. The advantage of a CPM machine is in the immediate post operative period when it is used through smaller ranges of flexion. One disadvantage of the CPM machine is lack of muscle contraction and resultant atrophy. Another disadvantage is that they rarely give actual knee flexion to 110°. The cost of a CPM machine used in our sports rehabilitation facility is two and one half times that of a custom cycle ergometer[2].

The shortcoming of the cycle ergometer is that it can not be used readily when a patient has less than 60° of knee flexion. Its advantages include active knee motion, no need for external power, knee flexion reliably past 110° and its relative lower cost.

Biomechanical studies have shown ergometric cycling to be an excellent mode of rehabilitation. The tibiofemoral compressive forces induced during cycling (1.2 time body weight) were significantly lower than many activities of daily living such as level walking (2-4 times body weight) or stair climbing (3-7 times body weight)[5]. The stress on the anterior cruciate ligament during ergometric cycling was also found to be low compared to other activities of daily living, making it valuable in early rehabilitation of patients with repaired anterior cruciate ligaments[5].

The custom cycle ergometer has been used successfully on thirteen postoperative patients at our institution. The surgeries included total joint replacement (5), anterior cruciate ligament reconstruction (4), internal fixation of a distal femoral fracture (1), internal fixation of an osteochondral fragment of the medial femoral condyle (1), repair of a quadriceps tendon rupture (1) and retinacular repair of a lateral patella dislocation (1). This device has been enthusiastically accepted by the patients.

SUMMARY

We have developed a new design in cycle ergometry to expedite postoperative knee rehabilitation. Continuous active motion is utilized with initial knee flexion of 60° progressing to greater than 110° by varying the crank length and seat height on the custom cycle ergometer. An individualized knee range of motion profile may be generated for each patient with varying crank lengths and seat heights. When compared to continuous passive motion, continuous active motion utilizing a custom cycle ergometer may be more reliable in attaining 110° of knee flexion and may help prevent quadriceps and hamstring atrophy.

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CARPAL TUNNEL SYNDROME IN HEMODIALYSIS PATIENTS

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INTRODUCTION

Carpal tunnel syndrome is common in patients undergoing hemodialysis. Symptoms and signs of carpal tunnel syndrome in these patients tend to be more severe than those seen in idiopathic cases, and the etiology is probably multifactorial[1,2,3]. The presence of polyneuropathy, arteriovenous fistulae, amyloid deposits, atherosclerosis, and generalized edema have all been advanced as reasons for an increased incidence in hemodialysis patients[3,4,5, 6,7,8,9].

The purpose of this study was to review our experience with carpal tunnel syndrome in hemodialysis patients and to evaluate the results of carpal tunnel release in these patients.

MATERIALS AND METHODS

Since 1978, the senior author (WDE) has operatively treated 21 wrists in 13 hemodialysis patients for carpal tunnel syndrome at the University of Wisconsin Hospitals and Clinics and the adjacent Veterans Administration Hospital. Eight patients were male and 5 female. The age of the patients at the time of their first visit ranged from 55 to 73 years (average 65.1 years). The cause of renal failure was chronic pyelonephritis in 4 patients, membranous glomerular nephritis in 2, polycystic disease in 2, and hypertension, Goodpasture's disease and obstructive uropathy in one each. The cause of renal failure was unknown in 2 patients.

The time on hemodialysis prior to the onset of carpal tunnel symptoms ranged from 3 years and 9 months to 17 years (average 9.1 years). One patient had stopped hemodialysis 12 and 15 months prior to right and left carpal tunnel release respectively, but the symptoms of carpal tunnel syndrome had been present 18 months prior to discontinuation of the hemodialysis. The symptoms of carpal tunnel syndrome had been present in these patients ranging from 3 to 44 months (average 13.9 months) prior to surgical treatment.

All patients in this series had pain in the median nerve distribution, and 19 of 21 wrists were worse at night. Three patients had pain in the fifth finger. No patient had constant pain, but burning paresthesias were noted in 18 of 21 involved hands. Subjective numbness in the median nerve distribution was seen in all cases, and 4 patients had numbness in all fingers. Fourteen of 21 hands were regarded by the patient as weak and clumsy. Most patients felt the symptoms were worse during hemodialysis, and many reported relief with vigorous shaking or use of the hand.

Physical findings included a positive Tinel's sign in 14 hands, and a Phalen's sign in 17 (in one wrist the arteriovenous fistula precluded this latter test). Atrophy of the thenar eminence was seen in 15 and weakness in the abductor pollicis brevis was noted in 17. Sensory loss in the median nerve distribution to the fingers was seen in all hands, and 6 had involvement of the fifth finger.

Preoperative nerve conduction velocity tests and electromyography were performed in all cases except one. In every case, there was prolongation of the distal motor latency in the median nerve as defined by Melvin[10]. All but one case had an increase in the distal sensory latency. Evidence of denervation of the abductor pollicis brevis was seen in 11 hands. In addition, prolongation of the distal ulnar sensory latency was present in 7 hands. Generalized slow neural conduction, indicative of polyneuropathy, was seen in 11 of the 13 patients.

A complete physical examination, radiographs of the involved hand and wrist, and laboratory studies were conducted preoperatively on all patients. Eight patients had lower extremity weakness or stocking glove sensory loss consistent with uremic polyneuropathy. Eight patients had trace to one plus pretibial edema. Radiographic examination of the hand and wrist demonstrated diffuse demineralization in most patients, a brown tumor of the radius in one and an old scaphoid fracture in one.

An arteriovenous fistula for hemodialysis access had been placed in all involved wrists. At the time of surgery, active arteriovenous fistulae were present in 16 wrists and inactive fistulae present in 5. Thirteen patients were known to have had at least one fistula revision.

Of the 21 hands in this study all had operative treatment by division of the deep transverse carpal ligament. Release of Guyon's canal was also performed in 6 hands for ulnar nerve symptoms, or a delay in conduction of the ulnar nerve at the wrist. Superficial median nerve epineurolysis was done in nine hands, usually when there was evidence

of thenar denervation. Surgery in twelve hands was done under general anesthesia, five with intravenous regional (Bier) anesthesia, three with axillary block, and one with local infiltration. In the five cases where a tourniquet was necessary for intravenous anesthesia, it was placed below an antecubital fistula, or used in an extremity with a nonfunctional fistula.

In all cases, operative findings included "hourglass" compression of the median nerve, with synovitis noted in 5 wrists. Synovectomy was carried out in two patients, but neither showed microscopic evidence of amyloid deposition. Encroachment on the nerve by a mass in the carpal canal was not noted in any wrist. However, one radius contained a biopsy proven brown tumor. We encountered carpal canal serous fluid in only one patient.

A bulky dressing and volar splint was applied in the operating room and worn for approximately two weeks. Sutures were removed at 2 weeks and patients were placed in a wrist splint or short arm cast and instructed in finger motion exercises. After three additional weeks, all immobilization was discontinued and the patients were instructed in wrist motion exercises.

RESULTS

Follow-up ranged from one month to three years and averaged 8.4 months. One patient died of cardiac arrest one month following surgery. The minimum follow-up for the remaining patients was 3 months. Subjectively, the results were excellent. All patients were relieved of hand pain, and only three hands had minimal residual paresthesias. Eight of the 21 hands had no postoperative numbness, and the remaining 13 had improved significantly. Most patients with preoperative weakness felt their strength was improved.

Objectively, the results were less encouraging. Preoperative sensory loss was completely eliminated in only three hands, but improved in 17. Only one hand demonstrated no improvement in sensory loss. Weakness showed minimal improvement, and no patient had reversal of thenar atrophy. There were no complications.

DISCUSSION

In the last 10 to 15 years, there has been an increasing awareness of carpal tunnel syndrome in hemodialysis patients. Using electrodiagnostic criteria, the incidence of carpal tunnel syndrome has been reported to be as high as 32% in hemodialysis patients[1]. The condition is often bilateral and usually more severe than idiopathic cases[1,2]. In our series, bilateral carpal tunnel syndrome was seen in 8 of 13 patients, and thenar denervation was present in over 50%.

The etiology of carpal tunnel syndrome in hemodialysis patients is probably multifactorial, but most authors have reported a strong association with an arteriovenous shunt placed in the affected extremity[1,2,3,5,7,8,9]. All patients in the series of Minami, Kenzora and Bradish had an arteriovenous shunt on the involved side[1,2,3]. Similarly, our patients all had at least one prior arteriovenous shunt on the involved side. The shunt does not have to be active to be associated with carpal tunnel syndrome. In Tietz's series, six inactive shunts were seen in 22 hands, and Minami noted eight inactive shunts in 20 hands[2,8]. In the present series 5 of 21 involved hands had inactive arteriovenous shunts.

In all his cases, Kenzora noted a serous transudate in the operative field at the time of carpal tunnel release and felt this was due to high venous pressure. Additionally, Kenzora used a Penrose drain as a tourniquet below the arteriovenous fistula in each case. In contrast to the findings of Kenzora, we noted a serous transudate in only one patient; in this case, the carpal tunnel release was performed without a tourniquet. In our five cases where a tourniquet was used, no serous fluid was noted. No other author has reported serous transudate as an operative finding, and we cannot explain this discrepancy.

It is well known that patients with polyneuropathy are more susceptible to compression neuropathy and that polyneuropathy is common in uremia[14,15]. Minami stated that none of his patients had polyneuropathy by electrodiagnostic criteria[8]. In our study, 11 of 13 patients had electrical studies indicative of polyneuropathy, and there were physical findings to support this in eight.

Engel and Kenzora did not feel that time on hemodialysis was an important factor in the development of carpal tunnel syndrome[1,5]. However, the average time on hemodialysis in Minami's patients was over seven years[2], and Tietz found that patients on hemodialysis longer than five years had a four-fold increase in the incidence of carpal tunnel syndrome[2,8]. We agree that time on hemodialysis probably does make a difference. Only one patient in this series had been on hemodialysis less than five years, and the average time was over nine years.

The subjective results of carpal tunnel release in hemodialysis patients have been excellent in previous studies, but the objective results have been less dramatic[1,2,3,8]. As in other studies, our major presenting symptom was pain, and this was completely relieved in all patients soon after surgery. Likewise, paresthesias were relieved completely in 18 of 21 hands and vastly reduced in the remaining 3. Numbness improved in nearly all patients, but objective sensory loss persisted in over 80%. Atrophy of the thenar musculature was seen in 13 patients preoperatively and there was no improvement after surgery.

We conclude that carpal tunnel syndrome in hemodialysis patients is multifactorial and associated with long periods on hemodialysis. The nerves of most of these patients are susceptible to compression neuropathy because

of an underlying uremic polyneuropathy. An arteriovenous shunt contributes to the development of carpal tunnel syndrome in these patients. Carpal tunnel release has good subjective results in hemodialysis patients, but the severity of their underlying disease and advanced involvement compromise objective results.

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MAGNETIC RESONANCE IMAGING OF ATHLETIC SOFT TISSUE INJURIES

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INTRODUCTION

While insults to cortex and marrow can be appreciated on magnetic resonance images, plain film radiography is sensitive and cost-effective in detecting and following fractures. However, athletes often sustain soft tissue injuries such as disruption in muscle-tendon units, myofascial hemorrhage, tendinitis, bursitis, meniscal tears, ligamentous injuries, joint effusions, and hemarthrosis. Magnetic resonance imaging (MRI), with it ability to detect subtle changes of tissue water content, is well suited for demonstrating soft tissue injuries. Its multiplanar capability, noninvasive nature, exquisite anatomic detail and high degree of contrast provide distinct advantages over other imaging techniques for evaluating athletic injuries.

MATERIALS AND METHODS

Fifty-four cases of sports-related soft tissue injuries examined by magnetic resonance imaging were reviewed retrospectively. The majority of the patients were young adults (average age 19.4 years) with males outnumbering females approximately 4:1. Many patients were collegiate athletes although patients with high school and recreational athletic histories were also included.

Magnetic resonance (MR) examinations were performed on either a 0.5-T superconducting magnet (Picker International, Cleveland) or a 1.5-T superconducting magnet (Signa, General Electric, Milwaukee). Standard T1 and T2 weighted spin-echo images were obtained with parameters such as (SE TR 500, TE 20) and (SE TR 2000, TE 100) respectively; however, imaging planes, slice thickness, field of view, use of surface coils and number of excitations varied depending on the region examined and the injury. For each study, images were obtained in at least two orthogonal planes.

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SUMMARY OF FINDINGS

In our series of 54 patients, the athletic injuries evaluated by MRI included: 1) muscle tears (biceps brachii, quadriceps, hamstring, and adductor muscle groups), 2) localized hematomas (gastrocnemius/soleus, quadriceps and adductor muscles groups), 3) tendon avulsions and intrasubstance tears (Achilles, patellar and hamstring tendons), 4) tendinitis (posterior tibial tendon), 5) bursitis (bursa deep to patellar tendon, bursa anterior to Achilles tendon), 6) meniscal tears (medial and lateral menisci), 7) ligamentous sprains (anterior and posterior cruciate ligaments, medial and lateral collateral ligaments) 8) joint effusions and hemarthrosis (knee joints). All injuries studied involved the lower extremity with the exception of one case with intramuscular hemorrhage of the biceps brachii muscle. Although a wide variety of athletic endeavors caused the injuries in this series, football-related activities were the most numerous. Incidentally found were cases of otherwise unrecognized bony abnormalities including femoral fractures, tibial fractures and bone contusions.

CASE EXAMPLES

Case 1:

A 21 year old collegiate football field goal kicker noted soreness in his thigh following a football game. The next day clinical exam showed increased swelling and a palpable defect in the region of the adductor longus muscle. The patient had a history of previous intramuscular hemorrhage in the right rectus femoris muscle.

A T1 weighted image (T1-WI) (Figure 1A) revealed decreased signal intensity in the rectus femoris muscle due to scar or hemosiderin from a previous intramuscular hemorrhage and moderately increased signal in the adductor longus muscle due to subacute hemorrhage. The corresponding T2 weighted image (T2-WI) (Figure 1B) better shows the changes produced by acute and subacute intramuscular hemorrhage. In general, the T2-WI provide better tissue contrast between lesions and surrounding structures whereas T1-WI provide better anatomical detail.

Case 2:

A 39 year old male physician was sprinting toward first base during a softball game and developed acute pain in his right posterior thigh.

An axial T2-WI (Figure 2A) of the upper thigh shows

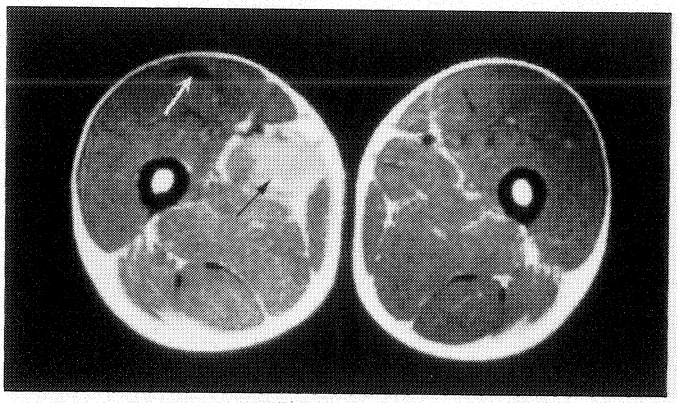

Figure 1A
Intramuscular hemorrhage. An axial relatively T1-weighted image shows increased signal intensity due to subacute hemorrhage (black arrow) in the adductor longus muscle and decreased signal intensity due to scar or hemosiderin (white arrow) in the rectus femoris muscle.

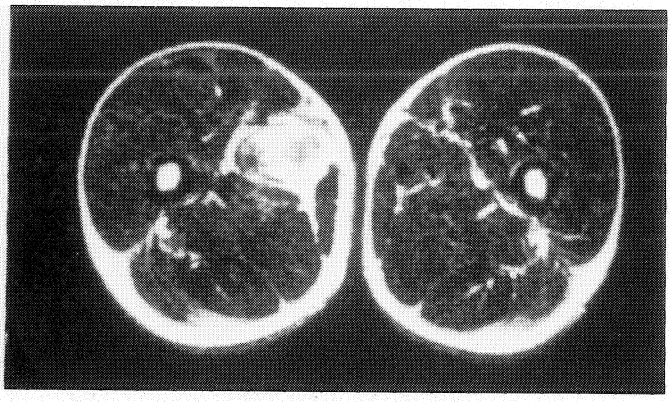

Figure 1B
The T2-weighted image corresponding to Figure 1A. There is striking contrast between the hemorrhagic and normal muscle.

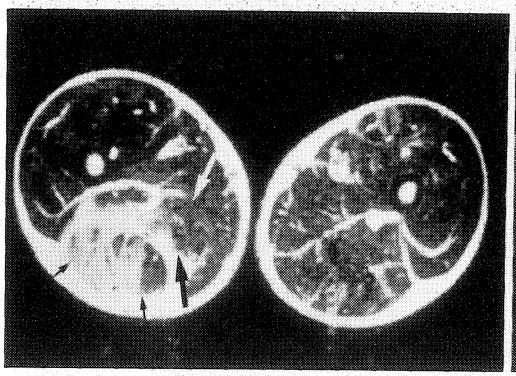

Figure 2A
Second degree muscle strain. An axial T2-WI shows asymmetric size of the margins of the hamstring muscle groups (short arrows) and adductor magnus muscles (long arrows) when compared with the normal left side. The increased signal on the right is due to hemorrhage or edema fluid.

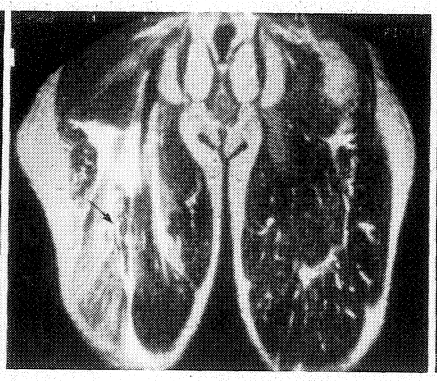

Figure 2B
A T2-WI showing interruption in biceps femoris muscle (arrow). Areas of bright signal represent hemorrhage or edema of the adductor and hamstring muscles.

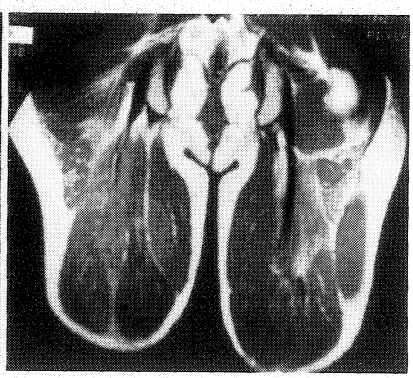

Figure 2C
A relatively T1-WI at the same level as Figure 2B. The slight increase of signal in the muscle as compared with the contralateral side is consistent with intramuscular hemorrhage.

asymmetric size of the margins of the hamstring muscle groups and adductor magnus muscles. Areas of hemorrhage and edema in muscle and along fascial planes have caused regions of increased signal intensity in the right leg appearing bright on MRI. Figure 2B demonstrates interruption of muscle fibers of the biceps femoris muscle. Hemorrhage and edema of the adductor and hamstring muscles are apparent as bright areas on this T2-WI. The contrast between normal and abnormal muscle is much less dramatic on the corresponding T1-WI (Figure 2C); the patchy areas of mildly increased signal are most likely intramuscular hemorrhage.

Case 3:

A 23 year old collegiate wrestler injured his right thigh during competition.

A coronal T1-WI (Figure 3A) shows the hamstring tendon avulsed from the ischial tuberosity on the right. The intact hamstring tendon is seen on the normal left side. A coronal T2-WI (Figure 3B) again demonstrates the avulsed hamstring tendon but better shows the result of bleeding and edema (bright areas) around the sciatic nerve and along fascial sheaths encircling the hamstring muscles.

Case 4:

This 19 year old collegiate gymnast presented with a nine month history of ankle pain particularly when tumbling. Physical examination revealed tenderness to palpation over the Achilles tendon especially at its insertion. She had decreased dorsiflexion of the left ankle (0 degrees compared to 25 degrees of the right ankle). Routine films showed bony ossicles interposed between the Achilles tendon and the calcaneal tuberosity.

On a sagittal T1-WI (Figure 4A), the intermediate signal of fluid within the distended bursa anterior to the Achilles tendon surrounds the bony ossicles. There is a very thin

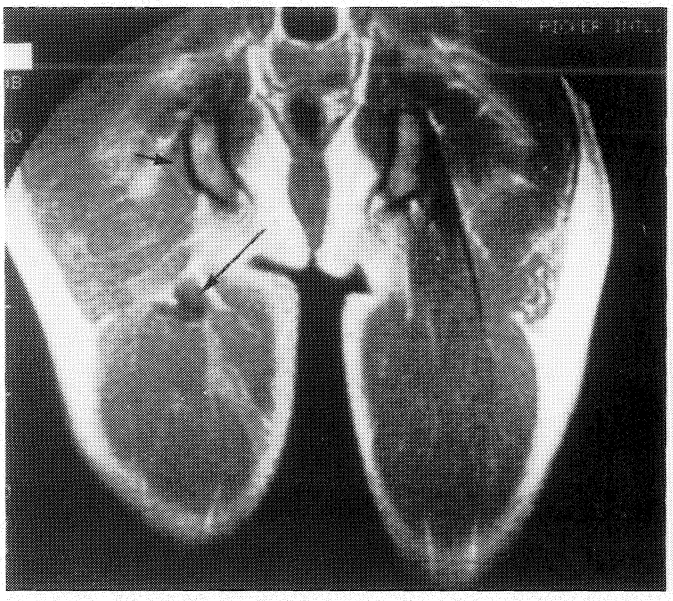

Figure 3A
Tendon avulsion. This coronal relatively T1-WI shows the avulsed right hamstring tendon (long arrow) retracted distally. The right ischial tuberosity (short arrow) no longer has the normal low signal area of tendon insertion. Areas of mildly increased signal compared to the contralateral side represent subacute bleeding.

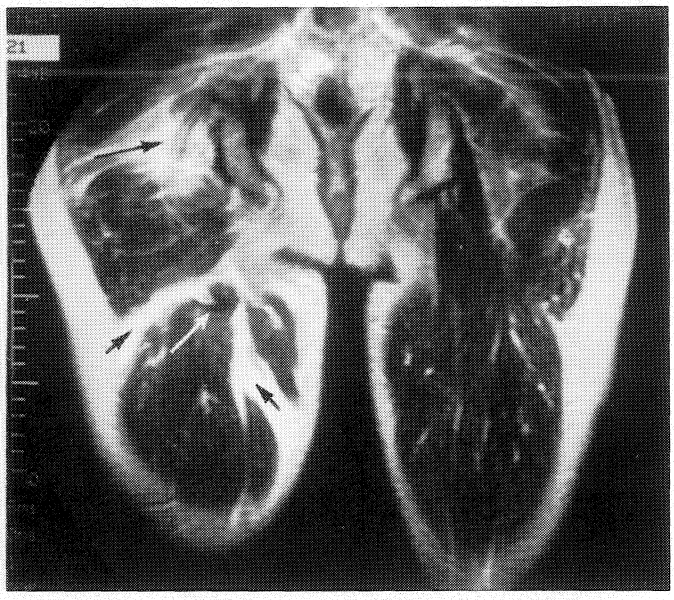

Figure 3B
Bleeding and edema (bright signal) around the sciatic nerve (long black arrow) and along the fascial sheath (short black arrows) surrounding the hamstring muscles are well demonstrated on this T2-WI. The avulsed tendon (white arrow) is again visualized.

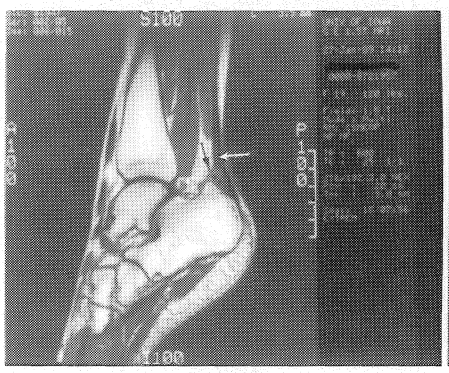

Figure 4A
Bursitis secondary to chronic tendon tear. On this T1-weighted sagittal image, the distended bursa (black arrow) produces intermediate MR signal intensity (gray). A white arrow indicates the vertical signal within the Achilles tendon which is due to a chronic tendon tear. The two rounded areas of low signal intensity within the bursa represent bony ossicles.

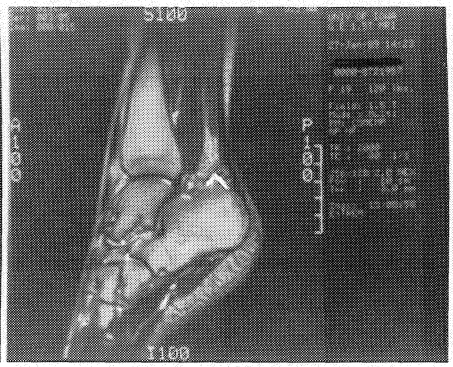

Figure 4B
On the T2-WI, bursal fluid stands out as bright signal contrasted to adjacent tissues.

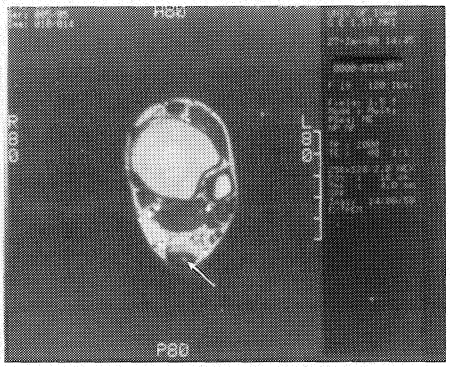

Figure 4C
An axial T2-WI image above the ankle joint again demonstrates the tear (arrow) within the substance of the Achilles tendon.

vertical band of increased signal representing a tear within the Achilles tendon. The corresponding sagittal T2-WI (Figure 4B) strikingly displays the bursal fluid with very high signal intensity. An axial T2-WI 15mm. proximal to the ankle joint (Figure 4C) demonstrates an abnormally increased signal within the substance of the Achilles tendon demarcating a partial tear.

Case 5:

A 16 year old high school football player sustained a clipping injury with his knee in extension. MRI demonstrates a tear in the posterior horn of the lateral meniscus on T1-WI (Figure 5A), and T2-WI (Figure 5B). The tibia is displaced anteriorly on the femur secondary to an anterior cruciate ligament injury. Other images of the MRI study (not shown) documented the tears of both the anterior cruciate ligament and the posterior horn of the medial meniscus. An arthroscopic examination confirmed each of these injuries.

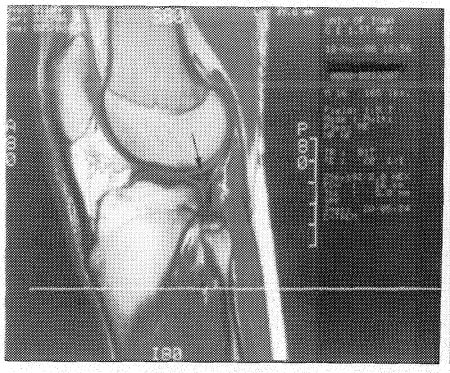

Figure 5A
Meniscal tear. A tear in the posterior horn of lateral meniscus (arrow) is shown on the T1-WI. Also noted is anterior translation of the tibia on the femur consistent with the concommitant anterior cruciate ligament tear.

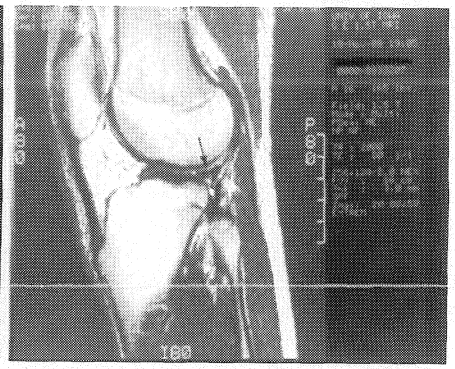

Figure 5B
The T2-WI also shows the meniscal tear.

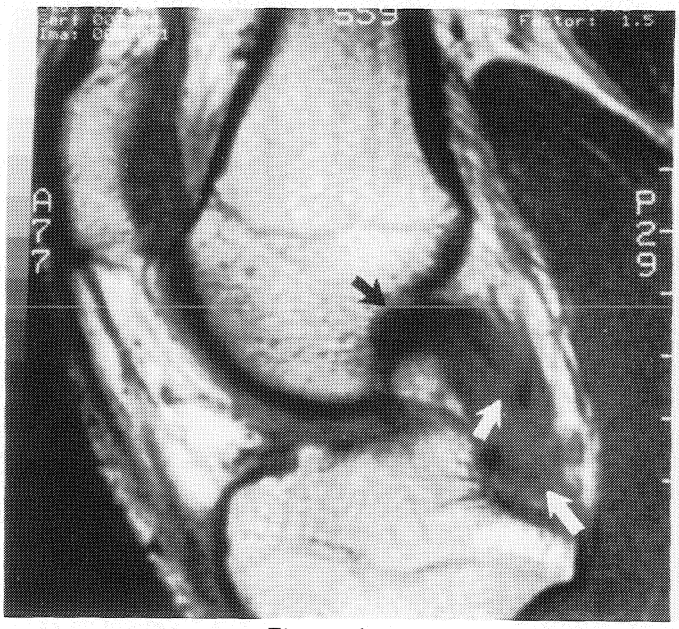

Figure 6A
Cruciate ligament tear. This relatively T1-WI shows a tear in the posterior cruciate ligament in its mid and distal positions (white arrows) but intact femoral attachment (black arrow).

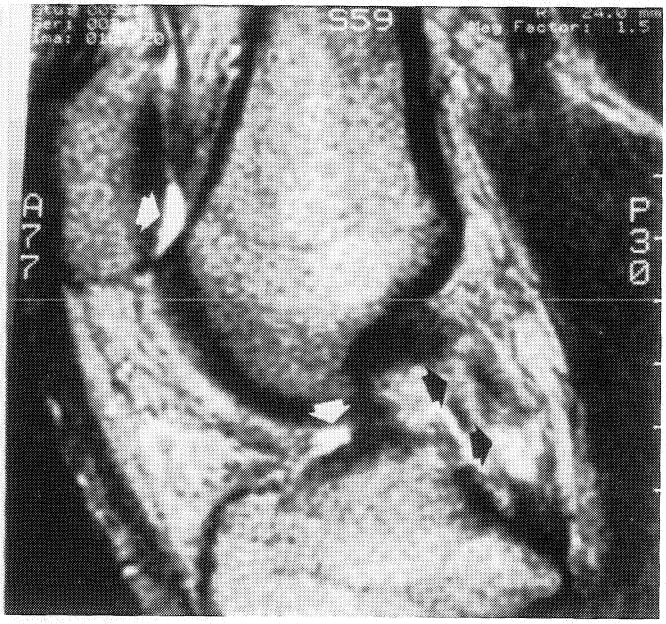

Figure 6B
The T2-WI at the same level as Figure 6A. The contrast between ligamentous edema (black arrows) and adjacent structures is more visible than on Figure 6A. The joint fluid (white arrows) shows very bright signal intensity.

Case 6:

An 18 year old collegiate football player was tackled landing on his flexed knee. Clinical examination revealed a mildly abnormal posterior drawer test. A representative slice from the T1-WI (Figure 6A) shows a posterior cruciate ligament tear in the mid and distal portions. The increased signal in the mid-substance and tibial attachment of the posterior cruciate ligament is also appreciated on the T2-WI (Figure 6B). The small hemarthrosis produces high signal intensity areas. Arthroscopy confirmed complete disruption of the posterior cruciate ligament.

Case 7:

A 20 year old collegiate football player received a blow to the anterolateral aspect of the knee producing valgus stress. The coronal T1-WI (Figure 7) reveals discontinuity of the medial collateral ligament with an undulating contour of the ruptured ligament. The ligament is surrounded by edematous soft tissues. Incidentally noted is lowered signal (dark) intensity in the marrow subjacent to the lateral tibial articular surface and in the lateral femoral condyle representing marrow contusion and microfracture. The area of contusion also shows increased signal intensity relative to adjacent marrow on T2-WI.

DISCUSSION

The number of sport-related musculoskeletal injuries has been steadily increasing. This trend is largely due to

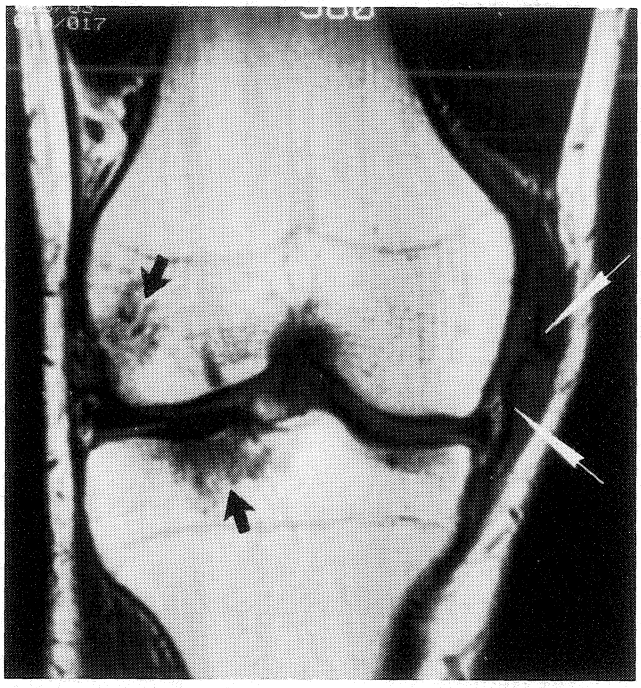

Figure 7
Collaterial ligament tear. The coronal T1-WI displays the ruptured medial collateral as a wavy, black ribbon-like structure (white arrows). The lower signal areas within the marrow cavity represent bone contusion and/or microfracture (black arrows).

a renewed interest in physical fitness and organized sports[25]. Although clinical use of magnetic resonance imaging initially developed for neurologic applications, a dramatic expansion for musculoskeletal applications has been seen in recent years. Two key factors leading to the increased utilization of MRI are its ability to directly image in multiple planes and its excellent soft tissue contrast. These features contribute to improved demonstration of soft tissue injuries.

By directly imaging structures in coronal, sagittal and oblique planes, MRI eliminates the artifact at slice interfaces found on computed tomographic reconstructions thus greatly improving image quality. The multiplanar capability also poses fewer positioning constraints with MRI than with CT. Many anatomic structures are optimally demonstrated in non-axial planes. For example, coronal slices are more useful for collateral ligaments while sagittal or oblique images are better suited to visualize the cruciate ligaments. Additional advantages of MRI over CT imaging include diminished artifacts from non-ferromagnetic metals and photon starvation (beam hardening).

The degree of soft tissue contrast achieved with MRI exceeds that of xerography and low kilovolt peak (kVp) technique radiography. Tenography is invasive, difficult to perform and interpret, and correlates poorly with surgical findings[1,11]. MRI surpasses arthrography in evaluating extracapsular structures in the knee such as collateral and patellar ligaments, quadriceps tendon and muscles[4]. For the detection of meniscal tears, 80-93% accuracy rates are reported for MRI compared to 60-91% accuracy rates quoted for arthrography[2,4,17,21]. Only MRI and sonography allow imaging of anatomic structures without exposure to ionizing radiation.

On both T1 and T2 weighted images, fat and muscle provide excellent background to visualize abnormalities. T1 weighted images are particularly useful in displaying changes in fat containing structures such as subcutaneous adipose tissue and bone marrow; the very high intensity signal of fat contrasts strikingly with virtually any process which infiltrates, interrupts or displaces the fatty tissue. In edematous states, the reticular fibrous septa in subcutaneous adipose tissue become thickened and change intensity on MRI tissues. MRI facilitates detection of bone contusion and occult fractures primarily by marrow signal changes although subtle or imperceptible increases in cortex signal also occur. While stress fractures can often be detected on MRI, radiographic and scintigraphic procedures are more cost-effective and sensitive means of diagnosis.

Muscle appears relatively hypodense (dark gray) on both T1 and T2 weighted sequences. The layers of fat along fascial planes, in subcutaneous adipose tissue and surrounding neurovascular bundles usually allow identification and definition of individual muscles. The axial plane is particularly helpful for outlining specific muscle contours to precisely determine location of lesions and to compare atrophy or expansion of muscle. Frequently, the opposite side can be used as a control. This feature is especially important for detecting subtle changes. In rare cases, it may be difficult to determine whether asymmetry of muscle size results from atrophy of one side or hypertrophy on the contralateral side; however, historical or clinical findings usually allow differentiation.

T2 weighted images show the effects of hemorrhage and edema in appendicular locations better than T1 weighted images; the bright signal from high water content stands out against the relatively low signal of normal muscle and the intermediate signal of fat on T2 weighted images. The contrast between muscle and hemorrhage or between muscle and edema may be subtle or imperceptible on T1 weighted images.

On MRI, first and second degree strains manifest as areas of increased signal intensity within the relatively hypodense muscle. Tracking of blood and edema fluid along fascial planes results in MRI changes far from the site of muscle tear. Although disruption of muscular continuity and retraction of ruptured muscle can be seen on MRI, it is primarily the effects of dissecting hemorrhage and associated edema which help demonstrate muscular strains. Hemorrhage is detectable on MRI due to increased presence of free water or changes in soft tissue relaxation time (produced by hemoglobin or its breakdown products).

Ehman and Berquist describe MRI findings in acute and recurrent forms of compartment syndrome; they found unilateral swelling and diffuse increased signal intensity on T2-weighted images[8]. However, normal muscle can increase in volume by as much as 20% during exercise[23]. Fleckenstein, et al. have demonstrated an increased T2 signal in exercised muscle of normal volunteers[10]. Thus exercise within minutes of MR scanning might reveal muscle images resembling compartment syndrome.

Tendons, menisci and ligaments normally have very low signal intensity on MRI. Detection of increased signal intensity within these structures can help identify subtle injuries. Tears of the Achilles tendon, posterior tibial tendon, quadriceps tendon and patellar tendon have been previously reported[6,7,19,22,23,24]. Hartzman, et al., described an MR examination in a case of Osgood-Schlatter disease showing high intensity signal at the patellar tendon insertion corresponding to the site of soft-tissue swelling and edema[12].

On both T1 and T2 weighted images, acute tendon strains can be visualized as increased signal intensity within the tendon or as diffuse tendinous thickening. Axial images are often important in identifying intratendinous signal change and defining changes in tendon size or location. Attenuation is seen with second degree strains while areas devoid of tendon are seen with third degree strains. Fluid within tendon sheaths may also be apparent.

Overuse-related syndromes can cause thickening of tendon-sheath complexes[5]. Scar has MRI characteristics similar to tendons. Thus, areas of scar can contribute to the width of the low signal at the site of a tendon injury. Increased signal intensity, particularly on T2 weighted images, can result from inflammatory changes or bursal effusion. Therefore, from MRI appearances alone it may not be possible to differentiate acute from chronic tendon tears.

Since 1983, when Kean, et al., first demonstrated the ability of MR to show articular structures in the knee, magnetic resonance imaging of the knee has evolved from a research tool to an accurate diagnostic examination commonly used for meniscal injuries[6,14,16]. Technical advances in surface coils have augmented resolution. Reicher, et al., reported an approximately 80% accuracy for detection of meniscal tears with MRI[21]. More recently in a series of 459 surgically correlated menisci, Mink, et al., found the sensitivity for detecting meniscal tears with MRI to be 95%, the specificity 91%, and the overall accuracy 93%[15].

An early report by Turner, et al., showed the usefulness of MRI in displaying ligamentous injuries of the knees and correlated their studies with surgical findings[24]. In the large series conducted by Mink and associates, the overall accuracy for detection of anterior cruciate ligament injuries by MRI was 95% and the false negative rate was 0%[17]. These figures compare to the 96.6% accuracy reported by Jackson, et al.[13]. An anterior cruciate ligament tear characteristically appears as a fusiform area of increased signal intensity replacing the normal ligament[6]. In chronic ACL deficient knees, MRI may not show the ligament or increased signal in the intercondylar notch. Attenuation of ligament or joint subluxation may also be demonstrated. Sagittal imaging is ideal for demonstrating the posterior cruciate ligament. MRI can identify whether an avulsion at the PCL attachment or an mid-substance tear has occurred.

The medial and lateral collateral ligaments of the knee lend themselves to coronal imaging. A normal band of signal in the deep fibers of the medial collateral ligament should not be misconstrued as a separation of the ligament from the medial meniscus. Since patients usually have the leg in mild external rotation when positioned in the knee coils for MRI, the lateral (fibular) collateral ligament is commonly visualized on the posterior coronal images. The crisp, linear black margins of normal collateral ligaments become thickened intermediate or high signal regions when tears occur. The disrupted ligament may be seen as an incomplete, buckled structure within the edematous soft tissues as in Case 7.

Until recently, positioning problems limited upper extremity studies from achieving the same high quality MR examinations possible for the lower extremities. Developments in surface coil design and improvements in software have led to a recent upsurge in MRI shoulder examinations for evaluation of rotator cuff tears and impingement syndrome[20]. The clinical utility of MRI has also been demonstrated in the hand and wrist; however, Koenig warns that visualizing cartilage and ligamentous defects in the wrist requires special attention to technique since partial volume effects can falsify perceived contrast[15,28].

Although fluid within a joint can be seen as an area of intermediate signal on the T1 weighted images, it is the T2 weighted image that strikingly displays these collections. Although MRI studies are unable to distinguish hemarthrosis from non-bloody fluid, it is likely that differentiation will be possible with better understanding of relaxation time characteristics[3,8].

CONCLUSION

These cases illustrate that MRI's multiplanar capability, noninvasive nature and excellent anatomic detail are extremely useful in evaluating athletic soft tissue injuries. Furthermore, MRI is helpful in establishing a rapid, accurate diagnosis in the competitive athlete obviating the need for invasive studies and possibly hastening definitive treatment or the confident return to competition.

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COMPARISON OF HIP RATING ASSESSMENTS IN PATIENTS WITH PRIMARY UNCEMENTED POROUS COATED TOTAL HIP ARTHROPLASTY

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INTRODUCTION

A number of hip rating systems have been developed to assess patients' functional status based on clinical examination and questionnaires[4,9,10]. A more recent hip assessment method has included radiographic criteria for patients with total hip replacement[7]. Unfortunately, no uniform method of evaluating and reporting the results of hip replacement surgery has been developed. A uniform method of evaluating would simplify interpretation of data, facilitate comparisons between results of different techniques and simplify clinical research in this area[3,5,7]. The purpose of this study was to evaluate five frequently used hip rating systems used in assessing the one and two year results of patients who had undergone primary uncemented porous coated total hip arthroplasty. Although in 1972 Anderson reported a study on the comparison of different hip assessments, no recent comparisons have been made. The evolving techniques and designs of hip arthroplasty need to be critically compared in order to define optimum hip procedures for various patient populations[1]. We attempted to examine the differences between rating systems as they compare to patient perception and to identify the critical areas of these rating systems.

MATERIALS AND METHODS

Merle d'Aubigne, Iowa, Harris, Hospital for Special Surgery and Mayo hip ratings were obtained on 100 consecutive patients seen at Walter Reed Army Medical Center returning for one and two year follow-up of primary uncemented P.C.A. total hip arthroplasty. The two surgeons involved in the surgical procedure completed the hip rating assessments, and each patient completed a questionnaire of two questions. The first question asked whether the patient considered his or her hip arthroplasty excellent, good, fair or poor. The second question asked the patients if they had pain, limp, function or limited motion. Charnley functional classification was also recorded for each patient (A = single hip disease with no other condition interfering with walking. B = bilateral hip disease with no other condition interfering with walking. C = systemic disease interfering with normal locomotion)[3]. The Friedman rank sum and Wilcoxan signed rank tests were used to compare overall results among rating systems and to contrast the proportion of excellent responses among the three Charnley functional classes. Mann-Whitney and chi square test for homogeneity were also used when appropriate.

The patient population consisted of 75 males and 25 females. The age range was 22 to 81 years with a mean of 60, and all patients had undergone a primary uncemented P.C.A. total hip arthroplasty. The procedures were all performed in the same manner by one or both surgeons, except that the first 60 procedures were performed through the direct lateral approach and the last 40 through the posterior lateral approach.

RESULTS

The results of all hips for each rating scale as well as the patient perception are tabulated according to excellent, good, fair and poor results (Figure 1). As illustrated, the Hospital for Special Surgery rating system was the most optimistic and the Merle d'Aubigne most pessimistic by the Friedman test survey of rank sums. Pairwise comparisons as analyzed by Wilcoxan signed rank test are illustrated in Table I. There was no statistically significant difference between The Hospital for Special Surgery assessment and patient impression; between patient impression, Mayo, and Iowa assessments; between the Iowa and Harris assessments; nor between the Harris and Merle d'Aubigne. All other assessments were significantly different ($P < .05$).

In analyzing patient perception, 84% of hips were excellent, 12% good, 4% fair and 9% poor. As illustrated in Table II, the patient perception did not necessarily agree with each rating scale result.

In comparing the rating scale results of hip replacements performed through the lateral approach versus the posterolateral approach, there were not statistically significant differences among the rating scales ($P = 0.2$ per Mann-Whitney test).

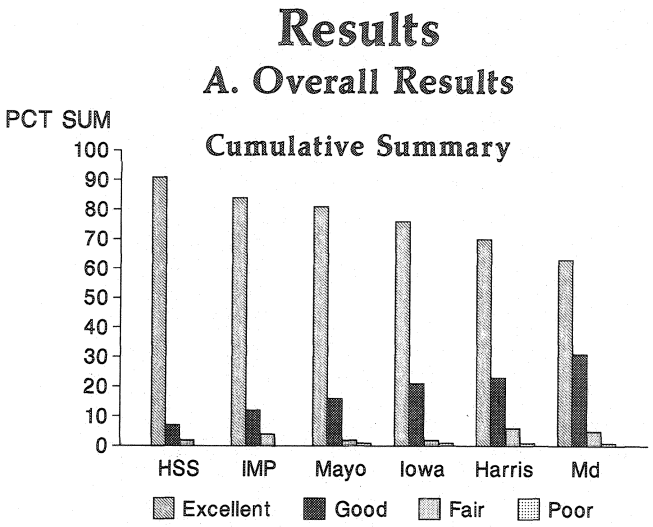

Figure 1
Cumulative results of each hip rating system and patient impression.

As noted in Table III, 42% of the patients were Charnley Class A, 41% Charnley Class B and 17% Charnley Class C. The proportion of hips judged to be excellent by each rating scale was significantly less ($P < 0.2$) for Charnley Class C patients. This finding is demonstrated in Table III. Twenty-nine percent of patients complained of limp, 18% of pain, 12% of decreased function and 6% of limited motion. Thirty-five percent of patients had no complaint. Patients with posterolateral approach hip arthroplasties had more "no complaints" than patients with lateral approach hip arthroplasties ($P = .06$), 48% compared to 27%. Patients with the posterolateral approach hip arthroplasties also had significantly fewer complaints of limp than patients with lateral approach hip arthroplasties ($P = .008$), 13% versus 40%.

Table 1

Statistical Analysis by Friedman Ranked Sum and Wilcoxan Signed Rank Tests

HSS	IMP	MAYO	IOWA	HARRIS	MD
303.0	328.0	338.5	353.0	379.0	398.0

Any two means underscored by the same line are NOT different (.05 level)

Table 1
Pairwise comparisons of the hip rating systems.

Table 2

Patients Own Perception	Rating System Result	HSS	MAYO	IOWA	HARRIS	MD
Excellent	%E	95.3	87.0	84.5	76.4	71.4
#84	%G	4.7	13.0	15.5	20.0	27.4
	%F				3.6	1.2
	%P					
Good	%E	83.3	58.3	33.3	41.7	25.0
#12	%G	8.3	33.3	58.3	41.7	50.0
	%F	8.4	8.4	8.4	16.6	25.0
	%P					
Fair	%E	25.0	25.0	25.0	25.0	0.0
#4	%G	50.0	25.0	25.0	25.0	25.0
	%F	25.0	25.0	25.0	25.0	25.0
	%P		25.0	25.0	25.0	25.0
Poor						
#0						

(E = Excellent, G = Good, F = Fair, and P = Poor)

Table 2
Hip rating scores for each patient impression.

DISCUSSION

Although hip rating systems have been widely used in the clinical review, their utility has been questioned due to the following factors:

1. The weighting applied to each parameter lacks objectivity and uniformity among rating systems.
2. The rating of each parameter by the patient or the examiner remains highly subjective, and the rating system results can be quite variable depending upon personal and environmental factors.
3. Pain may significantly influence joint function. Thus, including it in a clinical rating system introduces a double penalty for poor hip function.
4. There is no standard of comparison for the available clinical rating systems.
5. Clinical assessment in rating scales are nonspecific; they may not provide refined gradation to differentiate the patients' hip functional changes caused by factors

Table 3

Survey	Results	Charnley A	Charnley B	Charnley C
HSS	Excellent	41	39	11
	Not excellent	1	2	6
Mayo	Excellent	37	36	8
	Not excellent	5	5	9
Iowa	Excellent	34	36	6
	Not excellent	8	5	11
Harris	Excellent	35	32	3
	Not excellent	7	9	14
MD	Excellent	28	30	5
	Not excellent	14	11	12

Table 3
Categorization of hip rating results by Charnley functional class.

related to implant design, surgical technique and rehabilitation regime[3,5,7].

Although not clinically practical, a functional analysis, such as investigated by Chao, may eliminate some of the biases mentioned above[3].

In the present study, we evaluated five existing hip assessment scales in order to analyze their relative merits. We found The Hospital for Special Surgery rating system to be the most optimistic and the Merle d'Aubigne system to be the most pessimistic. We found no statistically significant differences between Hospital for Special Surgery system and the patient impression, between the Iowa and Harris systems or between the Harris and the Merle d'Aubigne system. In many cases, the patient perception was more optimistic than the surgeon's impression (i.e., a hip with a severe limp and no pain). We postulate that patients' impressions may have been overly optimistic because of their enthusiasm for this new, theoretically appealing form of implant fixation (bone ingrowth rather than acrylic fixation) or because the assessment and questionnaires were answered in the presence of the operating surgeon. Although four of five rating systems were more pessimistic than the patient impression, they are not necessarily a less accurate measure of the patients' results. Supporting this statement, we found patient impression not to be significantly different between the lateral and posterolateral approach patients; however, the number of patients with no complaints and with limp complaints were significantly different between the two approaches.

In all rating systems, the percentage of excellent results was significantly less in Charnley Class C patients. We believe this data demonstrates that in reporting the results after hip surgery it is important to know the proportions of patients in the various Charnley functional classes.

Our study demonstrates that the most common complaint of patients who have undergone uncemented primary porous coated total hip arthroplasty are limp (29%) and pain (18%). Therefore, in order to compare series of patients with uncemented replacements, it would be optimal to incorporate a more specific grading assessment for pain and limp.

CONCLUSIONS

When evaluating the follow-up results of uncemented primary porous total hip arthroplasty, we have found that the various hip rating systems produce different results. In order to critically compare follow-up series of patients after primary uncemented total hip arthroplasty, our data suggests the need to document the patients' Charnley functional classes and to document the pain and limp proportions of the rating assessment with as many gradations as possible. Our data comparing the lateral and posterolateral approaches, shows less limp complaints and more "no complaints" in the posterolateral approach patients, yet there was no statistically significant difference in overall impression. Therefore if only the hip rating scores are reported, the frequency of limp and of "no complaints" would not be apparent.

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SUPERIOR GLUTEAL ARTERY DISRUPTION WITH PELVIC AND ACETABULAR FRACTURES

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INTRODUCTION

Hemorrhage from pelvic and acetabular fractures is usually diffuse, arising from the fractured cancellous bed or from venous lacerations. Major arterial bleeding is less common, and for this reason the efficacy of early intra-arterial embolization has been questioned[1,13,14,18,19]. However, fractures displaced into the greater sciatic notch have been reported to cause bleeding from superior gluteal artery (SGA) laceration[2,7,18,19]. This is a report of two cases of SGA bleeding, with displaced fractures into the greater sciatic notch. In both cases, the diagnosis was delayed, and bleeding was slow yet persistent (seven days in the second case). Intra-arterial embolization was successful in both cases. Indications for use of intra-arterial embolization are discussed.

CASE 1

TH, an 18 year old male involved in a motorcycle accident, sustained a fracture of his left ilium and ischium with extension into and displacement of the greater sciatic notch (Fig. 1). Other injuries included a right pneumothorax as well as left femoral shaft and left humerus fractures. Peritoneal lavage in the emergency room was positive for gross blood. During exploratory laparotomy the patient underwent splenectomy, repair of a liver laceration and exploration of the left renal pedicle. In the first 18 post-operative hours, he required 10 units of packed red blood cells, 4 units of fresh frozen plasma and 6 units of platelets. Twenty hours post injury, concern due to continued bleeding led to abdominal re-exploration with no active bleeding identified. Over the ensuing 24 hours, he required 4 units of packed red blood cells and 2 units of fresh frozen plasma. By this time, a large left buttock and flank hematoma had developed. Angiography was performed 72 hours after injury. A terminal branch of the superior gluteal artery was actively bleeding, and intra-arterial embolization was performed with several 1 mm Gelfoam beads (Fig. 2). No subsequent transfusion was required. The patient declined operative stabilization of his fractures and was managed successfully with traction and functional bracing.

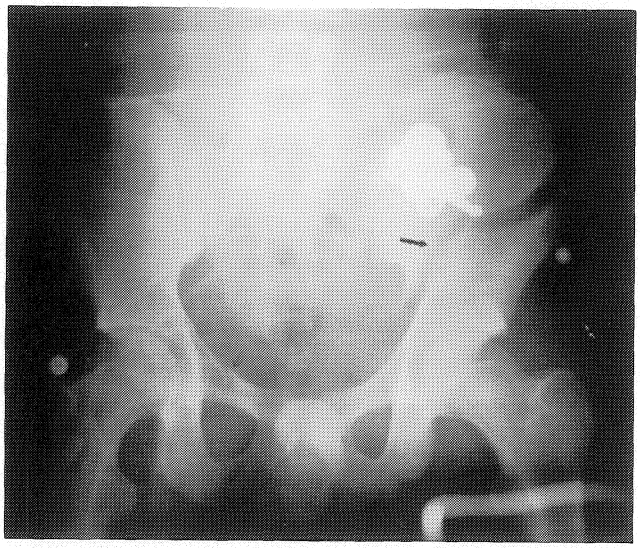

Figure 1
Iliac wing fracture displaced into the greater sciatic notch (large arrow). Note right pubic rami fractures (small arrows).

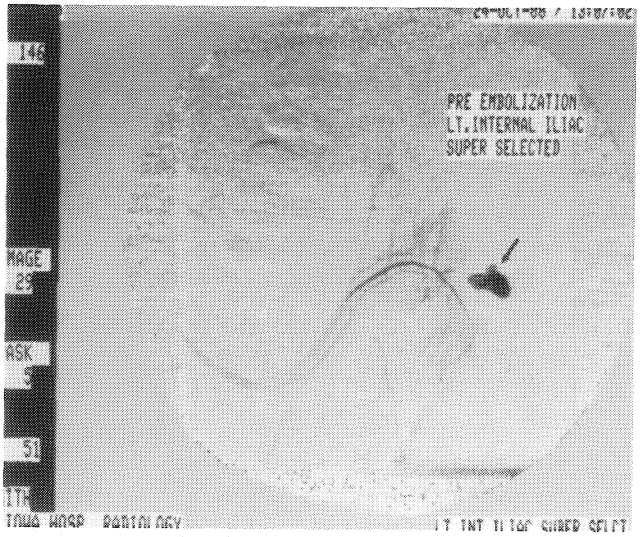

Figure 2
Pre-embolization angiogram demonstrates active bleeding from terminal branch of the superior gluteal artery (arrow).

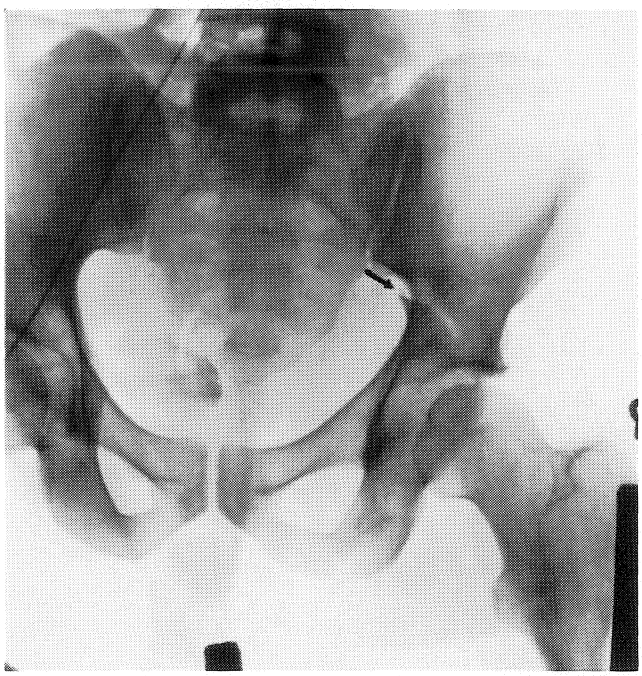

Figure 3
Posterior column acetabulum fracture with extension into the greater sciatic notch.

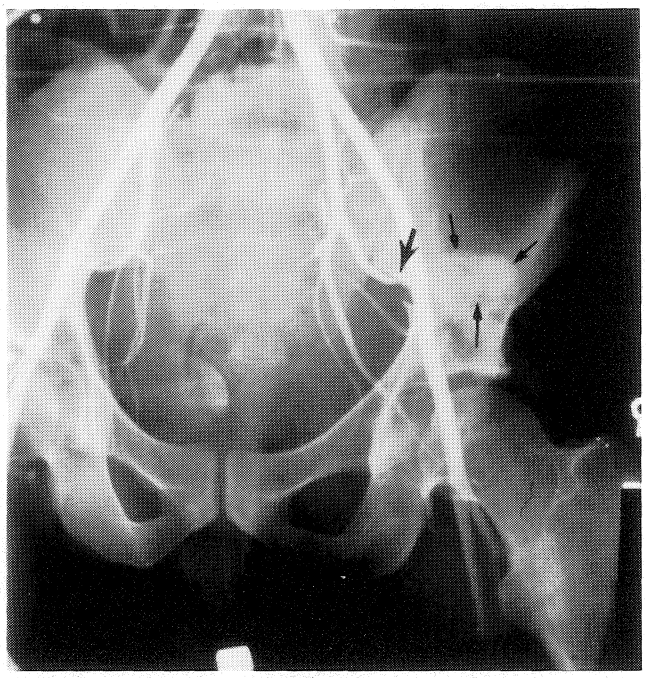

Figure 4
Angiogram shows disrupted SGA (large arrow) and dye extravasation (small arrows).

CASE 2

TS, a 22 year old male involved in a motor vehicle accident, sustained a right posterior column acetabular fracture displaced into the greater sciatic notch (Fig. 3). Other injuries included a left femoral shaft fracture and an open right patellar fracture. Peritoneal lavage was negative. On the night of admission, he underwent intramedullary rodding of the left femur fracture; irrigation, debridement, open reduction and internal fixation of the right patellar fracture and placement of a right distal femoral Steinman pin. Despite receiving 3 units of packed red blood cells intraoperatively, his hemoglobin dropped postoperatively from 12.4 to 8.3 in the first 24 hours. Over the next five days, 8 units of packed red blood cells were required with no obvious source of bleeding identified. Examination on post injury day seven demonstrated a painful right suprapubic mass. A CT scan showed a mass effect in the true pelvis displacing the bladder. Angiography demonstrated active superior gluteal artery bleeding at the greater sciatic notch (Fig. 4) which was embolized with Gelfoam and 2 coils. His hemoglobin stabilized without further transfusions. Four days later, open reduction and internal fixation of the right posterior column acetabular fracture was performed through a Kocher-Langenback incision. Retraction in the greater sciatic notch led to brisk bleeding, and, with difficulty, the SGA was clamped and ligated before the procedure could be resumed. The patient required eight units of packed red blood cells perioperatively. At his six month postoperative visit, the patient was walking without assistance, and radiographs demonstrated significant heterotopic ossification.

DISCUSSION

Arterial bleeding associated with pelvic and acetabular fractures is less common than venous bleeding or bleeding from fractured cancellous surfaces[2,4,14,16,19]. Arterial bleeding may be from large or small vessels. In displaced greater sciatic notch fractures, the superior gluteal artery is at risk due to its proximity to the fracture and its acutely angled course through this region. The mechanism of injury may be direct laceration or avulsion. Bosse found 3 of 8 displaced notch fractures to have SGA bleeding[2]. Letournel and Judet reported 150 patients with posterior column fractures; 5 were noted to have active SGA bleeding at operation[7]. Smith documented 5 cases of SGA bleeding associated with pelvic fracture, only 2 of which were displaced greater sciatic notch fractures. He hypothesized that the SGA may be avulsed in the notch region[19]. Other arteries noted to be at risk in pelvic fractures include the internal pudendal artery with notch fractures and the obturator artery with superior pubic rami fractures[11].

The diagnosis of arterial bleeding associated with pelvic and acetabular fractures is often difficult and may be delayed, as noted in our cases. Arterial bleeding may continue for many days, as in Case 2. A high index of suspicion is necessary in diagnosing SGA disruption with pelvic and acetabular fractures[2,11,18]. Patients with negative or equivocal peritoneal lavage or those who have intra-abdominal

bleeding controlled by laparotomy yet continuing to require transfusions, should have angiography. The patients with life threatening pelvic hemorrhage and expanding retroperitoneal hematomas despite adequate fracture stabilization may also be indicated for angiography[16].

Extensile exposures which ligate the lateral femoral circumflex artery create an abductor flap dependent on an intact superior gluteal artery. Therefore, some patients with posterior column fractures suitable for open reduction and internal fixation should have angiography as part of their preoperative workup to assess both active bleeding and the patency of the superior gluteal artery. Bosse concluded that the extended iliofemoral approach described by Letournel and Judet, the triradiate exposure of Mears and Rubash and the extensile approach by Reinert, et al., are contraindicated in cases of superior gluteal artery disruption[2,7,10,12].

Finally, successful embolization does not insure minimal operative blood loss, as illustrated in Case 2. Gentle retraction of tissues is mandatory to prevent embolic dislodgement. In contrast to the apparent ease with which Letournel and Judet operatively manage superior gluteal artery hemorrhage from a posterior approach, we found the bleeding difficult to control[7].

CONCLUSIONS

The possibility of arterial hemorrhage from the SGA must always be considered in patients with displaced sciatic notch fractures. A high index of suspicion will prevent delays in diagnosis. Bleeding may continue for many days with repeated transfusion requirements. The diagnosis is easily made with angiography and embolization is invaluable if an arterial bleeder is found. Blood product requirements dramatically decrease with successful embolization[1,9,17]. However, clot dislodgement and recurrent bleeding with subsequent fracture surgery may occur. Extensile extrapelvic exposures with an abductor flap must be avoided in cases of a lacerated or occluded SGA; thus, angiography should be part of the preoperative assessment of patients with displaced fractures involving the greater sciatic notch.

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CONGENITAL ABSENCE OF THE PECTORALIS MUSCLES IN TWO COLLEGIATE WRESTLING CHAMPIONS

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INTRODUCTION

Partial or complete absence of the pectoralis major is the most common muscle anomaly[3]. Description of this anomaly is generally restricted to reports of Poland's syndrome (partial or complete absence of the pectoralis major and ipsilateral upper extremity abnormalities, usually synbrachydactyly), but it may also appear as an isolated finding. Although this muscle deficiency is generally not regarded as causing significant disability, we found no objective support of this in the literature. The cases of two amateur wrestlers, with isolated absence of the sternocostal head of the pectoralis major are reported, including the results of functional strength testing.

METHODS

Strength testing was performed using a Cybex II Isokinetic Dynamometer. Rather than testing pure adduction, a more functional movement was tested combining adduction and flexion ("horizontal" adduction). With the subject supine and his arm abducted 90° in neutral flexion/extension, he was then asked to bring his arm across his chest against resistance to a position of neutral abduction/adduction and 90° of flexion (Figure 1). Internal rotation was tested in the seated position with the arm flexed 90° supported by a pad. The range tested was from neutral starting position to 90° of internal rotation (Figure 2). Each test was done at two speeds (90° per second and 180° per second) with three repetitions. The average peak torque (expressed as a percentage of body weight) are reported. In addition to the two subjects, a control was tested.

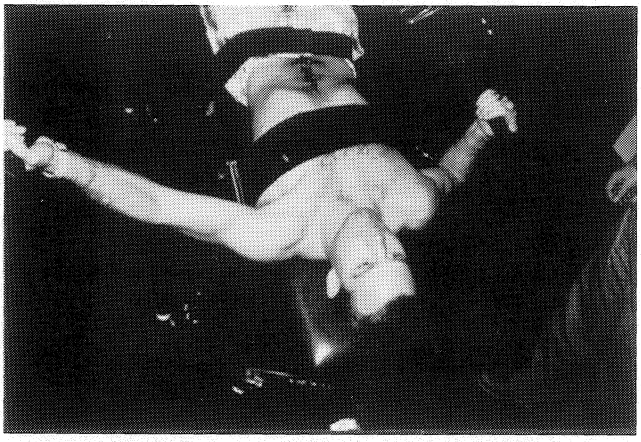

Figure 1
"Horizontal" adduction testing.

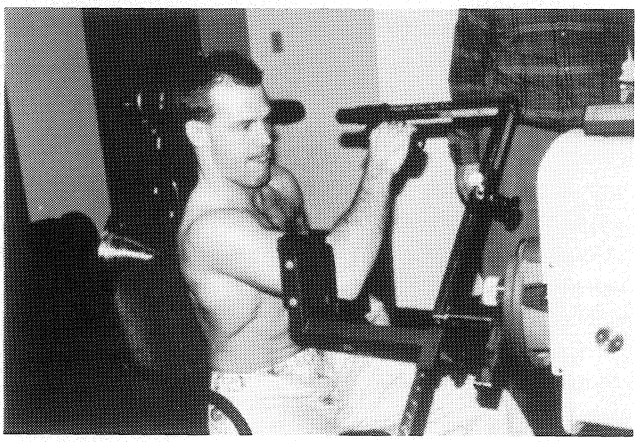

Figure 2
Internal rotation testing.

CASE REPORTS

Case 1

R.L. is a 28 year old, right-handed male. Clinical examination reveals absence of his right pectoralis minor and the sternocostal head of his pectoralis major (Figure 3a). His chest hair is also decreased on the affected side, but the remainder of his upper extremity examination, including his hand, is normal. His wrestling accomplishments include being a four-time all-American, two-time NCAA champion and an Olympic champion.

Case 2

K.D. is a 26 year old, right-handed male whose clinical examination is identical to that of Case 1 except that his nondominant chest wall is affected (Figure 3b). The remainder of the exam is normal. He is a two-time all-American and a NCAA champion.

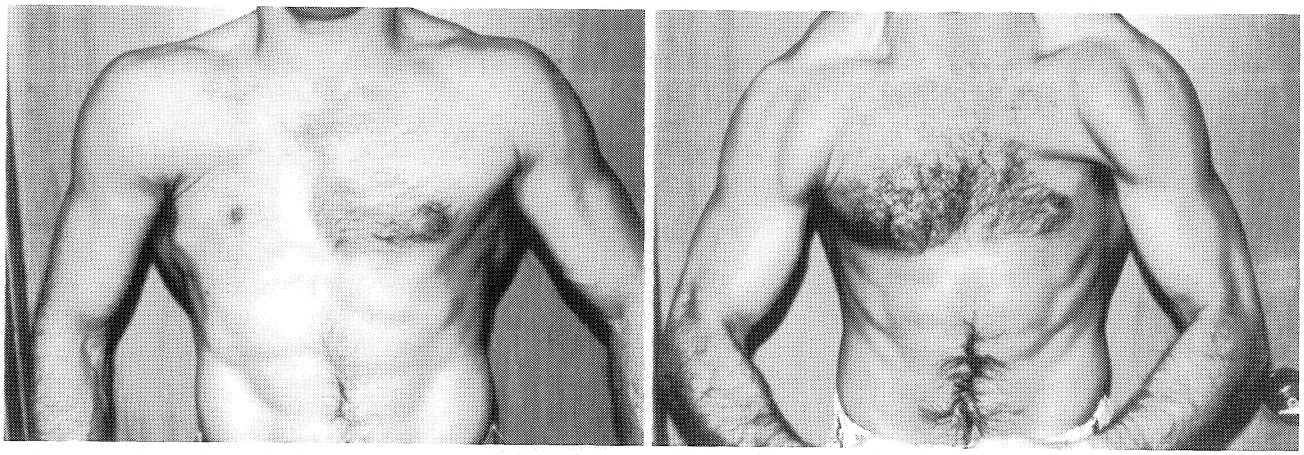

Figure 3A

Figure 3B

Figure 3(A-B): Subjects demonstrating congenital absence of the pectoralis minor and the sternocostal head of the pectoralis major.

RESULTS

Table 1 shows there was no significant difference in the internal rotation torque between the involved and uninvolved sides, but there was a significant difference in the horizontal adduction torque. The subject whose dominant side was involved had an average torque deficit of approximately 20%, and the wrestler whose nondominant side was affected showed an average deficit of 29%.

Table 1 CYBEX RESULTS

Subjects	Test Speed degrees/sec	Shoulder Involv.	Adduction Uninvolv.	Differ.	Shoulder Involv.	Internal Uninvolv.	Rotation Differ.
R.L	90	75	95	−21.1%	46	46	0
	180	70	86	−18.6%	44	49	−10.2%
K.D.	90	52	74	−29.7%	38	39	− 2.6%
	180	60	38	−28.3%	37	38	− 2.6%
Control*	90	91	86	5.8%	55	56	− 1.8%
	180	76	75	1.3%	54	60	−10.0%

Average peak torque values are reported as a percentage of body weight.
*For the control subject, the nondominant extremity is considered the involved side.

DISCUSSION

Absence of the sternocostal head of the pectoralis major is the most commonly recognized congenital muscle anomaly[3]. Unilateral partial absence of the pectoralis major with the clavicular head remaining is the most common finding[2,3,9,10,11,13,15]. Total or bilateral absence is rare; associated absence of the pectoralis minor is variable[2,10,11,13]. Beals felt this defect was most frequently associated with hand abnormalities (i.e., as part of Poland's syndrome), yet several others feel this muscle abnormality is usually an isolated finding[2,3,4,9,11]. There is no consensus whether isolated absence of the pectoralis muscles is a separate entity from Poland's syndrome or if it is a mild variant[6,7,11,12]. Supporting the latter view are reports of patients with an absent pectoralis major muscle having a unilateral absent kidney or acute leukemia (findings similar to those which can be associated with classic Poland's syndrome)[1,11,12]. Whether an isolated pectoralis deficiency is associated with Poland's syndrome or not, the question of functional impairment remains.

The pectoralis major is a primary shoulder adductor and a secondary internal rotator. Depending on the position of the shoulder, it can also aid in flexion or extension[8]. In Poland's original 1841 description, he implied that these patients had an associated inability to internally rotate the shoulder, yet others have not reported this finding[9]. We found no reports of specific strength testing, nor reports of functional impairment in patients with absent pectoralis muscles[5,9,10,13,14]. However, most of these reports include few, if any, subjects who had reached skeletal maturity[9].

Both of our subjects were skeletally mature, and had unilateral congenital absence of the pectoralis minor and the sternocostal head of the pectoralis major. Cybex testing showed no significant difference in internal rotation strength between the affected and unaffected sides, although there was a 20-30% decrease in horizontal adduction strength. The deficit was greater in the wrestler whose

nondominant side was affected; in the single control study, there was no significant difference in strength between dominant and nondominant sides. However, neither subject felt functionally limited and had not consciously changed his training or wrestling techniques to compensate for the anomaly.

CONCLUSION

Two highly successful wrestlers, both with unilateral congenital absence of the pectoralis minor and the sternocostal head of the pectoralis major, are reported. They had no appreciable decrease in shoulder internal rotation strength but did show a 20-30% decrease in shoulder horizontal adduction strength. Therefore, we conclude that patients similarly affected can be advised that their shoulder strength will be slightly decreased, but this is unlikely to limit them functionally.

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CAN BIOMECHANICS CONTRIBUTE TO CLINICAL ORTHOPAEDIC ASSESSMENTS?

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International Society of Biomechanics
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July, 1987

I wish to ask the question, "Can biomechanics contribute to clinical orthopaedic assessments?" This question is important because many articles answer this question affirmatively, even emphatically without compelling evidence or data to support their contention (Strong opinions without data often sway the bulk of medical opinion). It is fair to say that my opinions regarding clinical "gait analysis" are with the minority among locomotion researchers and may even be controversial. However, I believe the bulk of evidence is in my favor.

It is important to state my philosophical notions about medicine, because those notions are inextricably linked with my ideas about medical "tests," regardless of type. Furthermore, the constructive intent might be misinterpreted without grasping its underlying philosophy. My approach might be called an epistemological one, since the process is one of identifying the methods used to gain useful clinical knowledge.

Three philosophical notions affect my interpretation of diagnostic test use. (Aristotle supposedly said, "philosophy begins in medicine, and medicine ends in philosophy.") First, healers and practitioners of the medical arts have for all of recorded history, at least in the Western world, practiced what I would call "interventionism". The perception of "abnormality" demands intervention to restore "normality". Whether such expectation arose more from society or healers is not clear, but both contribute significantly. For practitioners, intervention is a livelihood. Also, the burden of proving efficacy lies with the practitioner who chooses not to intervene, rather than with the practitioner who intervenes; a situation I find logically backward, and ethically despicable. I mention interventionism because I view use of diagnostic and assessment tools as a form of interventionism, and because I believe the researcher who claims clinical applicability of some tool has the responsibility to document that his or her measures provide truly useful and unique, not simply interesting information. Just as a treatment should be proven efficacious, so should a new test.

Second, few would dispute that medical care costs are rising in all industrialized countries. Perhaps the most egregious example is in the United States, where approximately eleven percent of our enormous gross national product is expended on what is inappropriately called "health care". I feel this is more appropriately termed "illness intervention" which is, at best, indirectly related to the health of a society. No end is in sight for this rise in cost, and I see little likelihood that we will direct our resources to health, rather than to illness. A single factor responsible for such a complex and paradoxical phenomenon cannot be identified, but no one could question the role of technological "advances" and our indiscriminate worship of technology. Given the cost of "illness care", any new medical test must be unique, useful and demonstrably cost-effective.

Third, "biomechanics" has emphasized "mechanics" and has largely viewed its biologic subjects as structures with complex geometry and static physical properties, rather than living organisms. Fluxes in geometry, properties and behavior are usually ignored. However, such fluxes ignored by the clinician result in a most unsatisfactory patient-physician encounter. The more useful tests will account for and be conceptually consistent with the dynamic nature of the human organism.

With that background, let me return to biomechanics and clinical tests. I will focus exclusively on the role that biomechanics plays with patients. I am not considering how biomechanics can contribute to our concepts of disease; we all recognize that biomechanics can answer well-formulated questions, sometimes using groups of patients for appropriate observations. I am most familiar with studies of locomotion and will focus upon these. However, I do not intend to exclude other mechanical evaluation methods, and will mention a few examples. Regardless of my focus, my remarks are applicable to other mechanical measures in addition to gait analysis.

A clinician orders a "test" on a patient for one of four reasons: to distinguish between disease entities (i.e., diagnosis); to determine the severity of disease or injury (i.e.,

assessment or evaluation); to select among several treatment options; and to predict prognosis. There are no other patient-related reasons for ordering a clinical test.

Biomechanical measures of locomotor function were introduced over one hundred years ago; however, none have gained widespread clinical use for individual patients. Every day, clinicians routinely order technologically sophisticated tests such as hemoglobin levels, x-rays, serum glucose levels or magnetic resonance imaging, but no "biomechanical" tests are routinely used. Biomechanical measures have largely been technology looking for applications. They remain parochial tools awaiting proof of clinical usefulness, regardless of cleverness or technological sophistication; sophistication is no assurance of usefulness. The establishment of criteria for selecting and developing potential biomechanical measures and the exploration of strategies validating these measures enhance their potential clinical value.

Upon critical reflection, most reported biomechanical measures do not adequately contribute to the four reasons for ordering tests. Many explanations may be cited: most measures can be observed by an astute clinician; many mechanical measures are dependent upon the patient's mood, motivation, performance or pain; some measures exhibit short-term variability rather than a desirable stability; and few have been independently validated.

My former colleague, Roy Crowninshield, and I were thinking about this issue several years ago and came up with several usefulness criteria for biomechanical measures (Brand and Crowninshield, 1981). Since that time, I have added several more criteria to the list. At the present, I think the following constitutes reasonable criteria when developing or selecting a biomechanical measure: it must be accurate and reproducible; the measurement technique must not significantly alter the function it is measuring; it should exhibit reasonable stability; the measure should not be directly observable by the skilled clinician; the measure should be independent of mood, motivation or pain; it must clearly distinguish between normal and abnormal; the measure should be reported in a form analogous to some accepted clinical concept; the measure should be cost-effective; and finally, it must be appropriately validated. I do not assume that my list is all inclusive; others might propose equally or more important criteria.

Let me amplify several of these points. Few would argue with the first two criteria. To me, the most useful tests are those providing measures I can not observe. Being able to quantify what can be determined by simple observation adds very little, although there are a few exceptions. A patient's blood glucose, carbon dioxide content of arterial blood and the presence of a bone cyst represent findings which I could only suspect with my powers of observation. So it is with biomechanical measures; the most useful biomechanical measures will be those providing measures we can not observe, yet the emphasis has been quite the contrary. I hasten to reiterate that such quantitative distinctions assume far more importance in large groups of patients when answering some clearly posed question than in evaluating a single patient. Measures are likely to be more helpful when they are not dependent upon the patient's mood, motivation or level of pain. Tests such as blood glucose meet this criteria; measures such as temporal and distance factors of gait do not.

Measures are also more valuable when there is clarity between normal and abnormal. When there is considerable overlap of normal and abnormal, such as in temporal and distance factors of gait, the measure has far less value and can be interpreted only with corollary information. I will cite two examples of this phenomenon. When we were doing a long-term follow-up study of patients with clubfoot, it seemed logical to "evaluate" their gait (Brand, Laaveg, Crowninshield, & Ponseti, 1981). This we did in several conventional ways, but we additionally determined foot-floor pressures and the location of the center-of-pressure path. There were virtually no surprises. Pressure abnormalities could have been determined far more simply by looking at calluses on the patient's feet. Center-of-pressure paths for these clubfoot patients, while more variable than normals, usually fell within a range of normal. The center of pressure paths also failed to distinguish between dysfunctional feet and feet with only radiographic deformity. Schneider and Chao (1983) reached similar conclusions when looking at patterns of foot-floor reactions in patients with total knee replacements. Apparently, there was little difference between the reactions of normal subjects and patients with total knee replacements. However, when they performed a Fourier analysis they found distinctions between normal and abnormal. This suggests that sometimes the problem is not the measure but the analysis.

A measure is more useful when described in clinically relevant terms and concepts. Any measure requiring new concepts will be difficult to introduce and will likely meet with failure.

Cost-effectiveness does not directly relate to medical efficacy, and can not be the cause for clinical failure of biomechanical measures. However, cost-effectiveness will become increasingly more important in the application of any new measures. The measuring tool should reduce costs by eliminating unnecessary treatment, or by identifying conditions early and avoiding expensive complications.

Validation is critical in establishing medical efficacy. Valid measures may predict a different outcome than would have been previously predicted or suggest a different treatment than would have been recommended. If a measure does not change our predictions about a disease course or it does not change our recommendations for treatment, it is not valid (i.e., useful).

After reviewing the literature, it seems obvious that few measures are actually validated. Some years ago, Roy Crowninshield and I went through our files to classify papers on gait analysis. Our classification scheme was certainly not rigorously scientific, nor was our sample of papers necessarily random; nonetheless, subsequent reading has not changed my opinion about our conclusions: most papers report fascinating technology, but few document clinical usefulness (Table 1). Validation is so critical that any new measure should be designed with this in mind. A measure which a *priori* can not be validated is doomed to failure.

Table 1
GAIT PAPERS
FILES OF RAB AND RDC

Classifications (major purpose)	Number of Papers	Percent of Total
Description of Experimental or Numerical Method	65	44
Descriptive Results	48	32
Proposed Application	12	8
Demonstrated Application	8	6
Validated Application	0	0
	146	100

In the previously mentioned clubfoot study we attempted validation of the biomechanical measure (i.e., location of the center of pressure path) against an independent measure, a functional rating scale conceptually similar to many hip and knee rating systems (Brand et al., 1981). However, our biomechanical measure correlated neither with the functional rating scale nor radiographic changes. Thus, the biochemical measure failed to distinguish normal from abnormal, did not demonstrate anything I could not already observe, and it could not be validated against independent measures.

In a more successful effort, Chao and his colleagues reported what they termed a "performance index", based on the gait patterns of patients with knee disability (Chao, Laughman and Stauffer, 1980). They used stepwise discriminate analysis to select and weigh seven gait variables out of a potential 43 to create their performance index. This procedure usually discriminated between normal and abnormal. Their approach was promising, but contained several unsolved problems. First, the performance index correlated with a Harris Hip Score when all normals and abnormals were considered. However, the performance index did not discriminate knee disability among abnormal patients. Second, in another study of 254 patients considering 54 candidate variables, nine, rather than seven variables, were selected and their relative weighting changed (Laughman, Stauffer, Ilstrup and Chao, 1984). This means that weighting is sensitive to the data base. If such measures are so sensitive, we either need measures less sensitive to the data base or much larger data bases. Third, the weighting coefficients were different for men and women, again demonstrating a different sensitivity of subgroups. Recently, when Chao and colleagues developed a performance index for hip disability, the weighting factors again changed, showing sensitivity to selection of joint (Kaufman, Chao, Cahalan, Askew and Bleimeyer, 1987). This approach holds considerable promise, but it would ideally be less sensitive to such variables. It also requires more extensive validation against independent measures, such as clinical scores.

The minimal elements of appropriate validation include: well-designed cross-sectional and longitudinal clinical trials with statistically significant numbers of normals and abnormal patients, use of appropriate statistical methods to sort out potentially confounding patient variables, correlation of the measure with accepted independent measures, validation studies from several independent institutions.

Cross-sectional and longitudinal trials accomplish two things. First, they tell us whether the measure distinguishes normal from abnormal, or treated from untreated patients. Second, they tell us whether the measure fulfills one of the four criteria of usefulness mentioned earlier. Properly designed, prospective cross-sectional and longitudinal trials are the only way usefulness can be documented. Such trials require adequate normative data controlling for age, height, weight, time since injury, and so forth; hundreds or thousands of normals may be needed. Cross-sectional studies are valuable because of their ease, but are flawed by their poor control. They are most suited for the early phase of validation. Longitudinal studies are more valuable for validation, because each patient serves as his or her own control. In longitudinal studies, changes in a measure reflect variation in a disease process due to treatment or time. Only with longitudinal trials can we tell whether a measure suggest one treatment option over another. However, longitudinal studies are difficult and expensive to perform. These problems with longitudinal studies do not diminish the responsibilities incurred by those who introduce new measures. Good examples of appropriate longitudinal studies are those by Jernberger (1970) and more recently by Kenwright et al. (1986), who used stiffness as a measure of fracture healing.

Biomechanical investigators have recently developed measures with more sophisticated statistical tools. Grouping and weighting a number of measures may prove far more valuable than using single measures. Wong, Simon, and Olshen (1983) and Wooten, Kadaba, and Cochran (1984) used cluster analysis to more clearly distinguish normal from abnormal. Yamamoto and his colleagues (1983) reported principle component analysis of ten candidate gait variables to better distinguish normals from abnormals.

Can biomechanics contribute to clinical orthopaedic assessments? Currently, I must answer with a qualified yes. An example is seen in cerebral palsy where the degree of apparent weakness of the peroneal muscles does not necessarily correlate with the degree of peroneal muscle dysfunction. Nor does the presence of a strong muscle group on manual testing insure that the same muscle group will be normally active during gait. Owing to this lack of correlation, the results of tendon transfer surgery are unpredictable. Performing EMG's during gait and during other activities demonstrates which muscles are active and which are inactive, thus elucidating something we can not observe (Hoffer and Perry, 1983). Current published studies must still be considered preliminary; more definitive longitudinal validation studies are yet to be published.

Another example is a biomechanical test preoperatively predicting the outcome of a surgical procedure, such as high tibial osteotomy, an unpredictable and therefore seldom performed operation. Prodromos, Andriacchi, and Galante (1985) demonstrated that patients with unicompartmental knee arthrosis could be classified into two groups: those with a low adduction moment, and those with a high adduction moment. In a medium-term follow-up study (average 3.2 years), they demonstrated that the group with a low preoperative adduction moment did significantly better than those with a high adduction moment. Unfortunately, longer follow-up is lacking, as is independent verification from another laboratory. Nonetheless, this study is an excellent example of a biomechanical test contributing to clinical orthopedic assessments.

In conclusion, not all biomechanical measures will meet each of the usefulness criteria, nor are all of the criteria equally important. Also, others might formulate equally or more important criteria. Biomechanical measures intended to be clinically applicable must be developed with an appropriate validation strategy. In the future, adequate criteria for selection and validation of biomechanical measures can be met, and these measures can become clinically useful.

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PAGET'S DISEASE: UNCOMMON PRESENTATIONS

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INTRODUCTION

There are numerous accounts describing the classic features of Paget's disease, dating back to 1876 when Sir James Paget presented his meticulous description to the Royal Medical Chirurgical Society of London[9]. The aim of this paper is to bring attention to some of the lesser well known radiographic features of this disorder.

PATIENTS AND METHODS

Clinical histories and radiographs of patients with unusual aspects of Paget's disease were retrospectively reviewed. The cases were chosen from the Middlemore Hospital Bone Tumor Registry and Film Museum in Auckland, New Zealand and from the University of Iowa Hospitals and Clinics. Representative cases were chosen to demonstrate features of unusual sites, bone to bone progression, post-immobilization lysis and metastases to Pagetic bone.

RESULTS

Unusual Sites

Hand films of six patients with Paget's disease of the phalanges were reviewed. Although the historical information for some of these patients is scanty, hand lesions were incidental findings. Paget's disease in the hand has only occasionally been reported. The majority of cases have been in the proximal phalanges and occasionally the middle phalanges[1,3,4]. Five of our cases involved proximal phalanges; one occurred in a middle phalanx. Five showed typical appearances of moderately advanced blastic Paget's disease with thickening of the cortex, coarsening of trabeculae, and a generalized increase in bone density (Fig. 1a). Another was in the proximal phalanx of the thumb and demonstrated less advanced changes of mixed lytic and blastic Paget's disease of the proximal and ventral aspects of the bone (Fig. 1b). The Pagetic changes in phalanges are virtually identical to those seen in larger long bones. Yet, because of the infrequency of Paget's disease in the hands, the correct diagnosis may not always be considered.

Another unusual site is the patella[5,8]. The patient whose radiograph is illustrated in Figure 2 experienced sudden knee pain when walking down stairs and was found to have a pathologic fracture of the lower pole of the patella through moderately advanced Paget's disease.

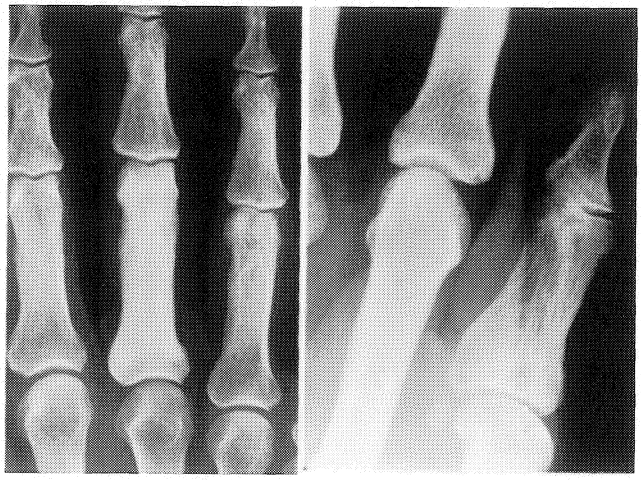

Figure 1A Figure 1B

Figure 1
Paget's disease in phalanges. (1a) Typical thickened cortices, coarsened trabeculae, increased density, and generalized enlargement of the proximal phalanx in established blastic Paget's disease in an asymptomatic 58 year old woman. (1b) Less advanced changes in the proximal phalanx of the thumb with the lytic front progressing diagonally across the bone and not yet affecting the distal dorsolateral aspect. The blastic phase has already started at the base. This was an incidental finding in a 62 year old man with symptomatic Paget's disease in his femur.

In addition to unusual bones affected by Paget's disease, there are unusual sites within bones. When long bones are involved with Paget's disease, the process typically starts at one end and progresses through the shaft until the

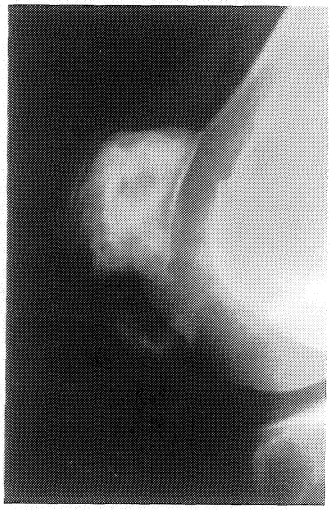

Figure 2
Pathologic fracture through the lower pole of the patella secondary to Paget's disease.

entire bone is involved[8]. There have been occasional reports of Paget's disease starting in the mid shaft and extending towards the ends[8,10]. Such a case is illustrated in Figure 3. Although polyostotic Paget's disease is very common, involvement of two separate parts within the same bone is uncommon[8]. Figure 4 shows Paget's disease in the proximal and distal tibia with a section of the intervening shaft still unaffected. Typical V-shaped fronts are seen advancing towards one another.

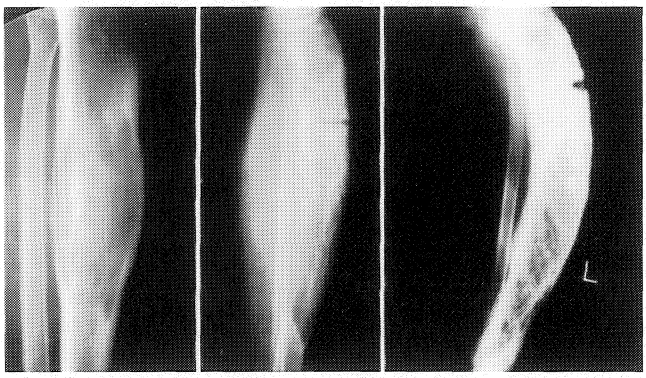

Figure 3A Figure 3B Figure 3C

Figure 3
Diaphyseal Paget's disease. (3a) 1941. Mainly lytic phase of Paget's disease but with some coarsened trabeculae and enlargement of the affected segment. (3b) 1952. A lytic front of Paget's disease is progressing distally through the lower tibial shaft. The proximal lytic front has encompassed the whole proximal tibia. (3c) 1967. The entire tibia has been replaced with blastic Pagetic bone.

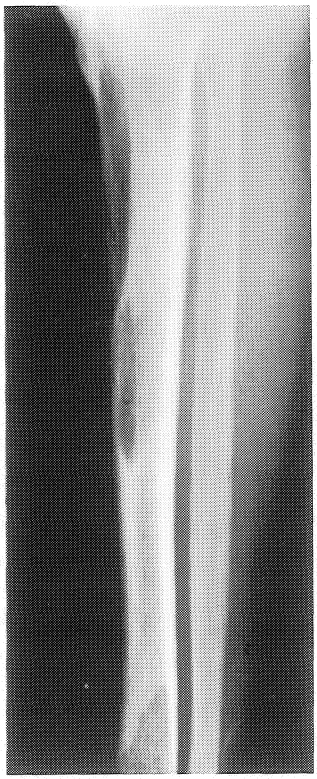

Figure 4
Two foci of Paget's disease in one bone. V-shaped lytic Pagetic fronts are seen approaching one another from either end of the tibia.

Progression from Femur to Tibia

Despite the frequent polyostotic nature of Paget's disease, the process should not cross joints. This rule does not, however, apply after the joint has been surgically fused. The patient whose films are illustrated in Figure 5 developed degenerative disease of his knee secondary to advanced Paget's disease of the femur. There was no Paget's disease in the tibia until after arthrodesis of the knee joint, when the Pagetic process could be seen extending across the fusion and into the previously unaffected tibia.

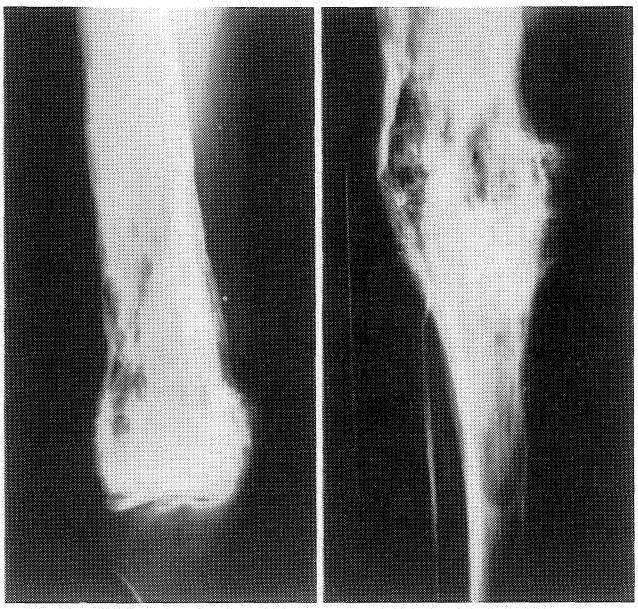

Figure 5A Figure 5B

Figure 5
Bone to bone progression. (5a) Advanced Paget's disease is seen in the femur. The tibia is unaffected. (5b) Some years later the Pagetic process has crossed the arthrodesis and is advancing down the tibia. (Courtesy of Prof. R. Gibson, Christchurch, New Zealand)

Post-Immobilization Lysis

Despite the markedly increased turnover of bone in active Paget's disease, serum calcium and phosphate are usually normal. However, after fractures or prolonged immobilization, urinary calcium excretion may be increased and rarely hypercalcemia may be encountered[7]. This is due to an increase in bone resorption in excess of bone formation. Radiographs of two patients with proximal tibial Paget's disease and tibial shaft fractures were reviewed. In both patients, advanced osteopenia developed proximal to the fracture and involved only the Pagetic bone. In the case illustrated in Figure 6, a biopsy was taken from the tibia in the center of the lytic region eight weeks after the fracture occurred. The histologic appearances confirmed the generalized osteoclastic resorption of Pagetic bone with no evidence of infection or neoplasm. This patient's

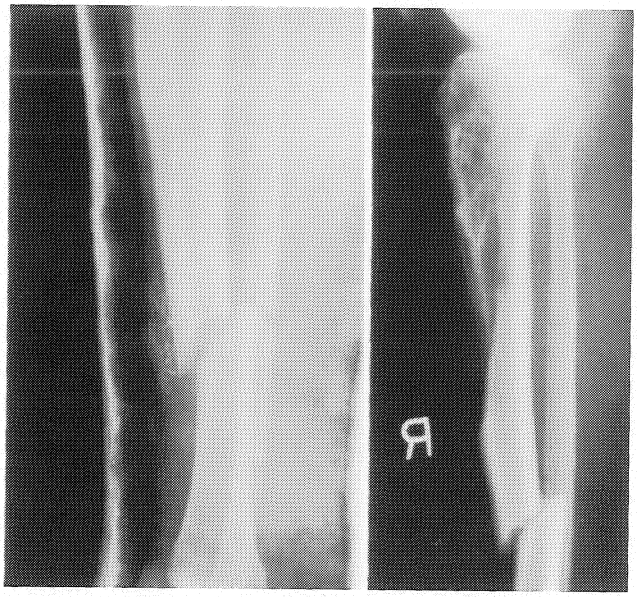

Figure 6A Figure 6B

Figure 6: Post-immobilization lysis. (6a) Acute fractures of the tibial and fibular shafts. Note the thickened anterior cortex of the tibia. (6b) Eight weeks after the fracture, there appears to be a destructive lesion of the proximal tibia with a well demarcated oblique junction between abnormal and normal bone.

serum calcium levels remained within normal limits. His alkaline phosphatase level was double the normal value. In both cases, the differentiation from reflex sympathetic dystrophy or disuse osteoporosis was easy because the Pagetic bone occurred proximal to the fracture sites. The preservation of essentially normal bone density distal to the fractures would exclude these forms of acute osteoporosis.

Metastases to Pagetic Bone

It has been suggested that the increased vascularity of bone affected by Paget's disease makes it more susceptible to hematogenous metastases[6]. Blastic metastases, particularly prostatic carcinoma metastatic to the pelvis and lumbar spine, can sometimes be confused with blastic Paget's disease[2]. The tendency for Paget's disease to affect specific bones and to produce coarse trabeculae, thickened cortices, and expanded bone, allows differentiation between blastic metastases and Pagetic bone. However, metastatic disease superimposed upon Paget's disease can cause a greater diagnostic dilemma. An example is a 78 year old man with pain in his mid back who was found to have radiographic changes of Paget's disease in the first lumbar vertebra in addition to loss of the left pedicles of T12 and L1 (Fig. 7a,b). On computerized tomography, L1 showed the typical thickened cortex, coarse trabeculation, and well corticated small rounded lytic regions of Paget's disease. In addition to destruction of the left pedicle associated with a soft-tissue mass, there were also other lytic

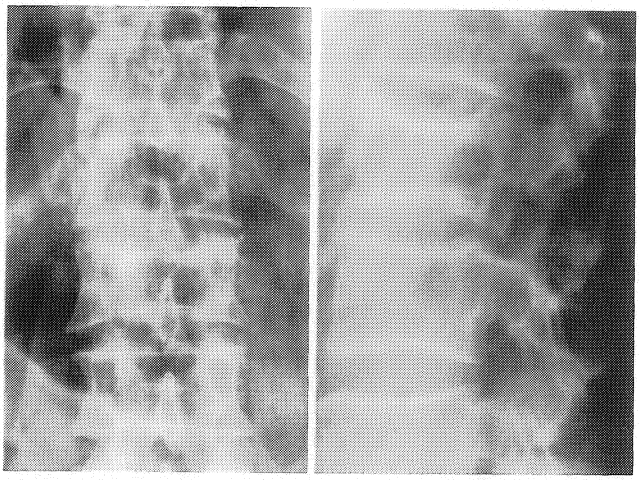

Figure 7A Figure 7B

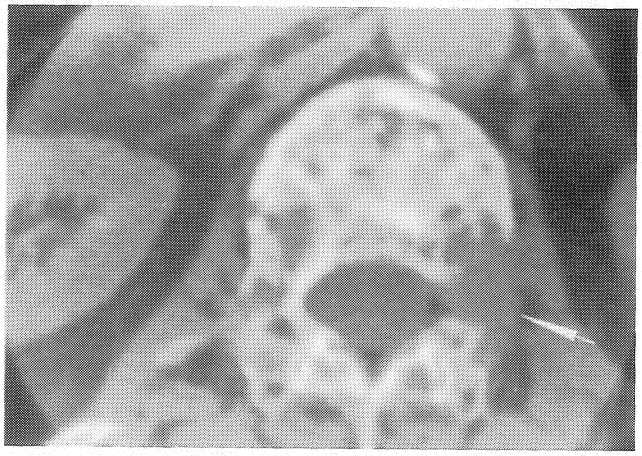

Figure 7C

Figure 7: Metastases to Pagetic bone. (7a,b) AP and lateral lumbar spine radiographs demonstrate the typical "picture frame" appearance of Paget's disease of L1 and the not well defined right L1 pedicle. (7c) CT through L1 shows thickened cortices and coarsened blastic appearances typical of Paget's disease. In addition, there is a metastatic lesion partially destroying the right pedicle and completely destroying the left pedicle. There is also a soft tissue mass adjacent to the left pedicle (arrow).

lesions showing ill-defined margins suggestive of a more aggressive process (Fig. 7c). A biopsy confirmed the coexistence of Paget's disease of bone and a poorly differentiated metastatic prostate carcinoma. In this case, the radiologic diagnosis was made easier by the purely lytic metastasis.

The cases presented demonstrate uncommon presentations of Paget's disease. Because we are unaccustomed to seeing Paget's disease in unusual sites such as the hands and patellae, the correct diagnosis may not be considered. The diagnosis may also be overlooked if it presents in the diaphysis or two separate sites within a frequently affected bone [10]. The phenomenon of post-immobilization lysis in Paget's disease is well known, yet it does not usually

produce the dramatic changes seen in Figure 6. This case was initially thought to be an aggressive neoplastic process, but review of the initial films revealed considerable thickening of the anterior cortex of the tibia typical of blastic Paget's disease. Because both Paget's disease and bone metastases are found more in the older age group, these conditions can be expected to occasionally co-exist, irrespective of the susceptibility of Pagetic bone for metastases. Blastic metastatic deposits are usually round or oval lesions which do not resemble Paget's disease. However, widespread blastic metastases can be more confusing. They are generally more symmetrically distributed than Paget's disease and should not coarsen the trabeculae or increase the size of affected bones[8]. Lytic metastases do not usually cause confusion with Paget's disease although occasionally a large metastasis can mimic the lytic phase of Paget's disease[8] (Fig. 8).

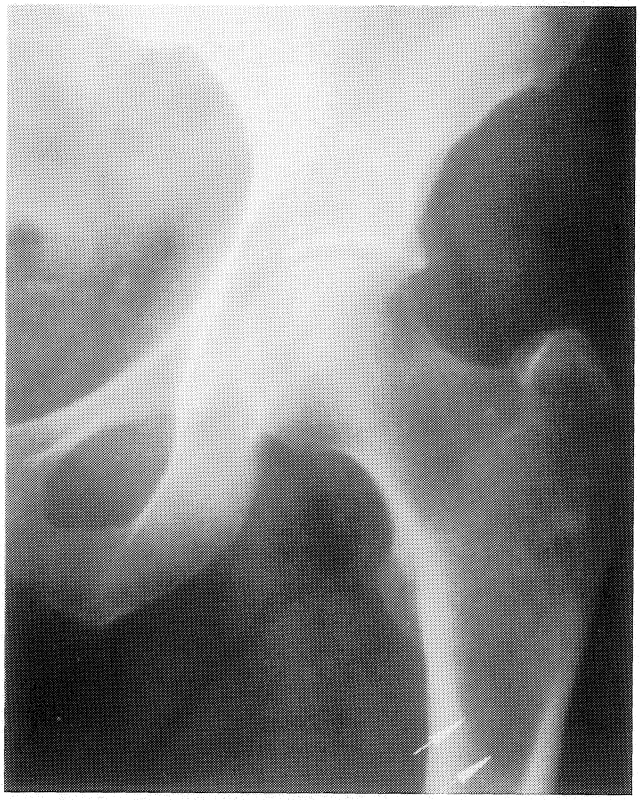

Figure 8
Large solitary lytic metastasis to proximal femur from breast carcinoma. The V-shaped inferior margin (arrow) gave the mistaken impression of lytic Paget's disease.

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BONE RESORPTION IN THE SPINAL CANAL FOLLOWING A THORACOLUMBAR FRACTURE WITH A DISPLACED FRAGMENT

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INTRODUCTION

The management of thoracolumbar burst fractures is considered one of the more controversial topics involving musculoskeletal trauma[2,12]. Posterior stabilization of the spine removing any fragments from the neural canal through a subsequent anterior approach is a common method of treating thoracolumbar spine fractures[6,8,13]. Significant risks exist with anterior decompression of thoracolumbar fractures while controlled prospective studies are lacking[8,13]. Also, the natural history of spinal stenosis caused by retention of displaced fracture fragments has not been well established[2]. This case demonstrates nearly complete remodeling of the neural canal with evidence of bone resorption.

CASE REPORT

A fifteen year old male high school student was riding a four-wheeler up a steep hill. The four-wheeler flipped over on him as he landed on his buttocks. His primary complaint was pain in the mid-back. He denied sensory or motor disturbance in his lower extremities and denied sphincter dysfunction.

Physical examination on presentation to the emergency room revealed an alert, cooperative and well-developed Caucasian male with tenderness at the thoracolumbar junction. Abdominal exam elicited hypoactive bowel sounds. Neurologic evaluation was completely normal.

AP and lateral roentgenograms and computerized tomography showed a burst-type fracture of L1 with a retropulsed fragment one centimeter into the spinal canal (Fig. 1).

Six days after injury, the patient's fracture was stabilized posteriorly using Harrington rods which included two levels above and below the fracture. Bone grafting was not performed. Postoperative roentgenograms and computerized tomography showed a decrease in the kyphotic

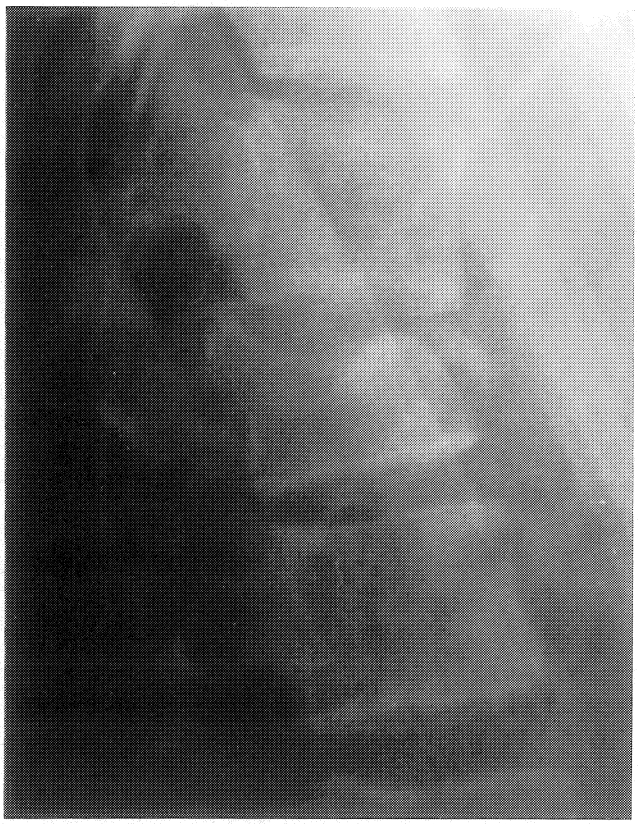

Figure 1A

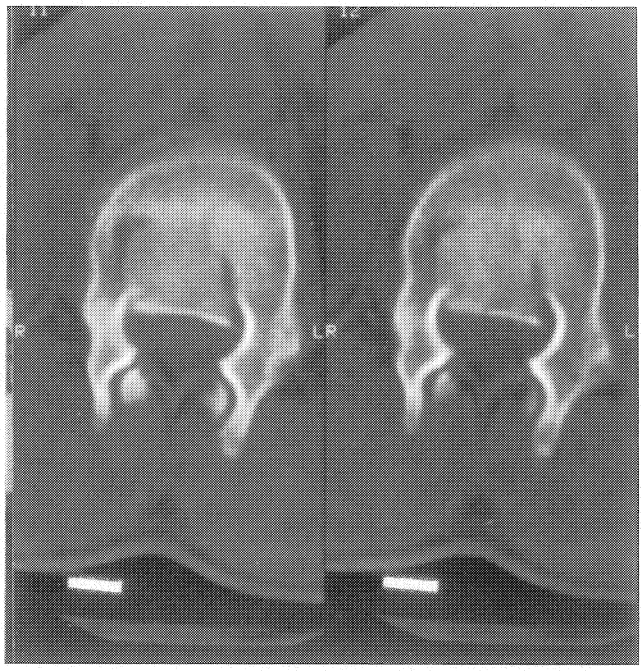

Figure 1B

Figure 1A-B
Radiograph and CT scan shows a burst fracture of L1 and a 12 degree kyphotic deformity. The retropulsed fragment is displaced approximately 1 centimeter into the spinal canal.

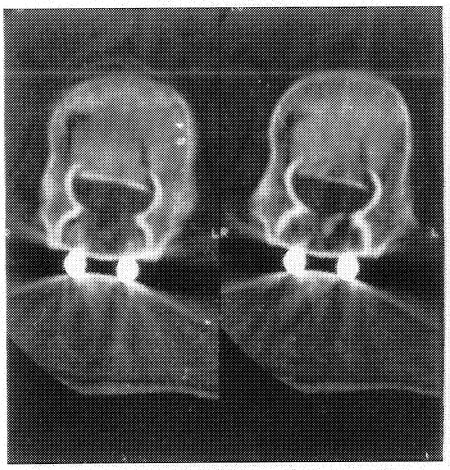

Figure 2
Computerized tomography of the L1 vertebral body after Harrington rod insertion, without significant change in the position of the retropulsed vertebral body fragments.

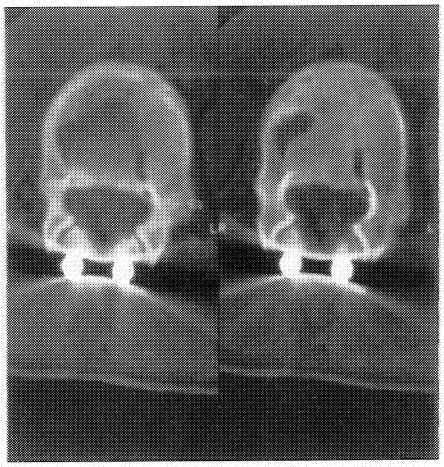

Figure 3
Three months after injury. Note the resorption occurring in the retropulsed bone.

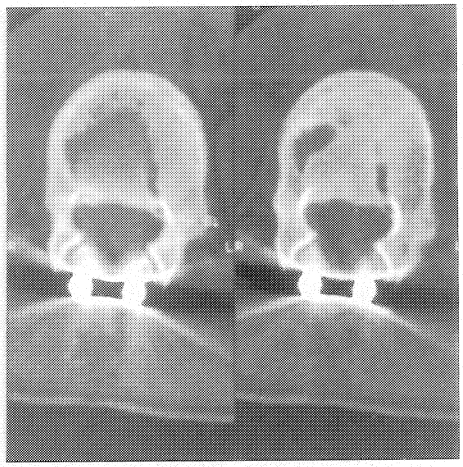

Figure 4
Nine months after injury. The protruding portion of the retropulsed vertebral body fragment has resorbed from the spinal canal.

deformity but little change in the position of the retropulsed fragment (Fig. 2). The patient was placed in a Jewett brace and returned to school three weeks after injury.

Three months after injury computerized tomography was repeated to evaluate healing before discontinuing the brace (Fig. 3). This scan showed stabilization of the fracture fragment with significant resorption. Further resorption was seen at 9 months follow-up (Fig. 4). The instrumentation was removed 21 months after surgery without complication (Fig. 5). The patient has been asymptomatic and has returned to competitive athletics.

DISCUSSION

Burst fractures of the thoracolumbar spine frequently have posterior displacement into the neural canal[8]. Posterior Harrington instrumentation is often sufficient to restore the bony dimensions of the spinal canal. However, in some cases posterior instrumentation alone does not obtain adequate reduction of free bony fragments or the displaced posterior cortex of the vertebral body[1,4,7,8,13]. Reduction of a retropulsed fragment may be difficult because entrapment occurs between the rigid pedicles[13]. This phenomenon is demonstrated in this and many other cases[1,2,3,7,9,11].

In animal experiments, both early stabilization and early decompression decrease the amount of residual paralysis after spinal cord trauma[2]. This has never been shown in human clinical studies. Recent long-term follow-up studies of thoracolumbar spine fractures from the University of Iowa suggest large non-reduced intraspinal bone fragments do not lead to residual sequelae[15]. The anterior or anterolateral approach to the thoracolumbar spine is technically difficult requiring prolonged bedrest, increasing the risk of pulmonary embolism[10]. Also, the anterior approach can disturb the vascular supply of the spinal cord and, in one series, averaged an additional blood loss of eight units[10,13]. If bone resorption were predictable, the care of these complex cases would be simplified without compromising the goals of treatment as stated by Dr. Stauffer: obtain a stable, pain-free spine without progressive deformity; achieve maximum recovery from neurologic deficit; prevent future functional or neurologic deterioration; and minimize complications[2].

I have also observed significant bone resorption of a large pelvic fracture fragment originally protruding into the birth canal of a fourteen year old girl (unpublished data). It is apparent that fracture callus in any location is unlikely to progressively encroach upon nerves or blood vessels. Does this mean that nerves and vessels secrete a substance that causes bone remodeling? Or, do nerves and vessels mechanically or electrically cause bone to remodel? The electrical theory is proposed by Cruess based on work by Bassett and Becker[14]. The resorption of bone in this case supports the postulate of Wolff's Law: "All bone, normal or abnormal, develops the structure most suited to resist the force acting upon it"[5]. A better understanding of this process would be helpful in predicting the long-term outcome of post-traumatic spinal stenosis.

SUMMARY

Serial radiographs and computerized tomography of an L1 vertebral body burst fracture in a fifteen year old boy show significant bone resorption of a large retropulsed fragment over 21 months. If predictable amounts of resorption were known to occur in these injuries, surgical decompression in some patients could be avoided.

ACKNOWLEDGEMENTS

I wish to thank Mrs. Margaret Bennet and Carol Roblee for their patience and expert editorial assistance.

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HIP DISLOCATION AS A COMPLICATION OF VALGUS REDUCTION AND FIXATION FOR AN INTERTROCHANTERIC FRACTURE—A CASE REPORT

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INTRODUCTION

Hip dislocation following open reduction and internal fixation for intertrochanteric hip fractures is an uncommon but serious complication usually associated with hip joint infection. Malcolm and Schatzker have reported 4 cases, 3 of which presented late as a result of sepsis, and the other presented early without signs of infection. No other reports of early non-infected dislocation complicating operative fixation of intertrochanteric fractures could be found. We report a case of hip dislocation secondary to valgus reduction and internal fixation of an intertrochanteric hip fracture.

CASE REPORT

A 66 year old diabetic female presented to the University of Iowa with hip pain 3 months after internal fixation of an unstable intertrochanteric hip fracture. Admission radiographs demonstrated generalized osteopenia with bone loss in the superior femoral head and neck, proximal migration of the screw and varus collapse of the fracture (Fig. 1).

The patient was taken to the operating room where the old screw and side plate were removed through a lateral approach. To augment fixation, the head was reamed and partially curetted to allow packing with methylmethacrylate. A short threaded compression screw was placed and a valgus reduction was maintained with application of a 145 degree side plate. The fixation was secure and position confirmed by intraoperative radiographs. Radiographs on the first postoperative day demonstrated the reconstruction of the proximal femur; however, a superolateral dislocation of the hip had occurred (Fig. 2). CT scan showed no evidence of cement or other loose body within the joint (Fig. 3). Subsequent closed reductions of the hip dislocation were unsuccessful.

The patient was returned to the operating room where an anterior approach was performed. Even though the inferior medial spike of the femoral neck and the lesser trochanter were resected, a reduction could still not be achieved. The 145 degree side plate was replaced with a 135 degree side plate without removal of the firmly anchored compression screw. The reduction was now stable with 30 degrees of abduction and was held in this position with a spica cast for 6 weeks; radiographs at this juncture demonstrated maintenance of reduction (Fig. 4). Subsequently, at 7 weeks the patient presented with leg pain during ambulation. Radiographs revealed failure of the screw fixation with the femoral head remaining reduced (Fig. 5). Due to recurrent urinary tract sepsis, the patient was treated with a resection arthroplasty instead of prosthetic reconstruction.

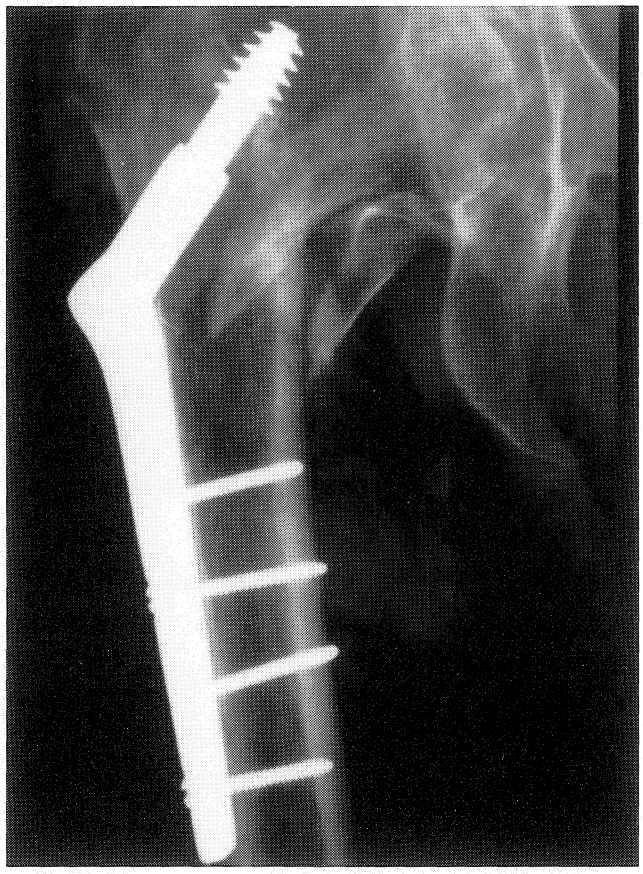

Figure 1
Presenting radiograph with proximal migration of the screw and varus collapse of the fracture.

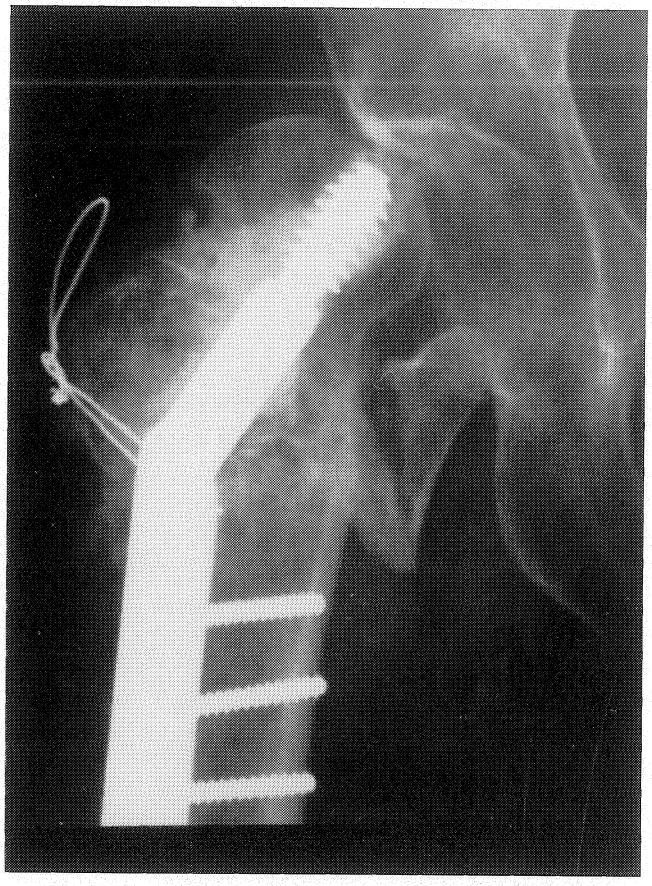

Figure 2
Routine film on postoperative day one, the femoral head is dislocated superolaterally.

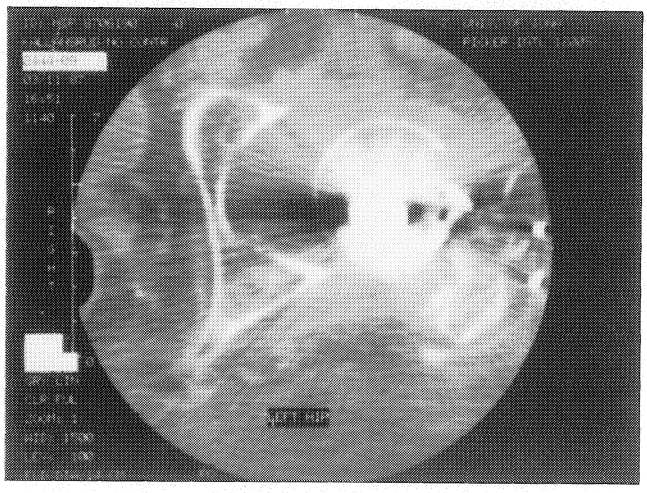

Figure 3
CT scan shows no evidence of a loose body within the joint.

DISCUSSION

Valgus reduction and internal fixation with or without trochanteric osteotomy is a recommended method of treating unstable intertrochanteric fractures. While many authors favor a more anatomic reduction of these fractures, valgus osteotomy is still useful in difficult fractures or revisions[1,3].

Several factors, in addition to the valgus positioning, may have contributed to the dislocation. Prior to open reduction, we felt the lesser trochanter and medial neck spike may have contributed by levering the femoral head

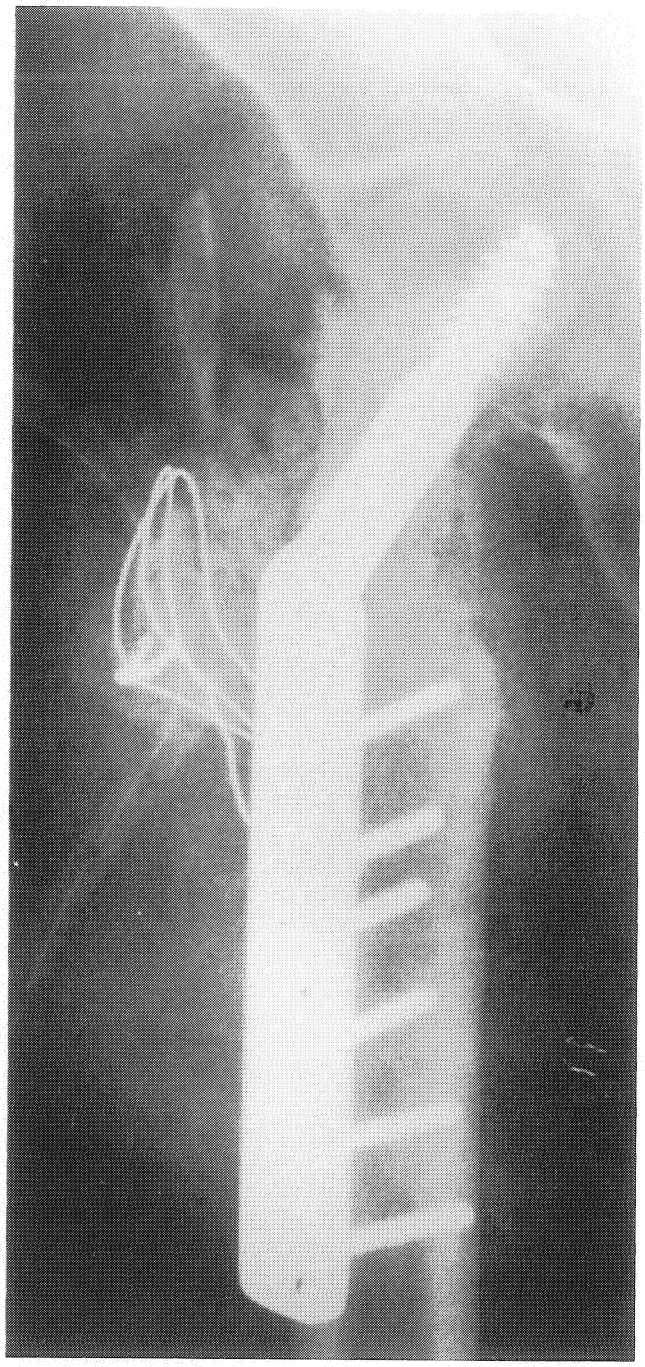

Figure 4
Radiograph at six weeks with maintenance of reduction.

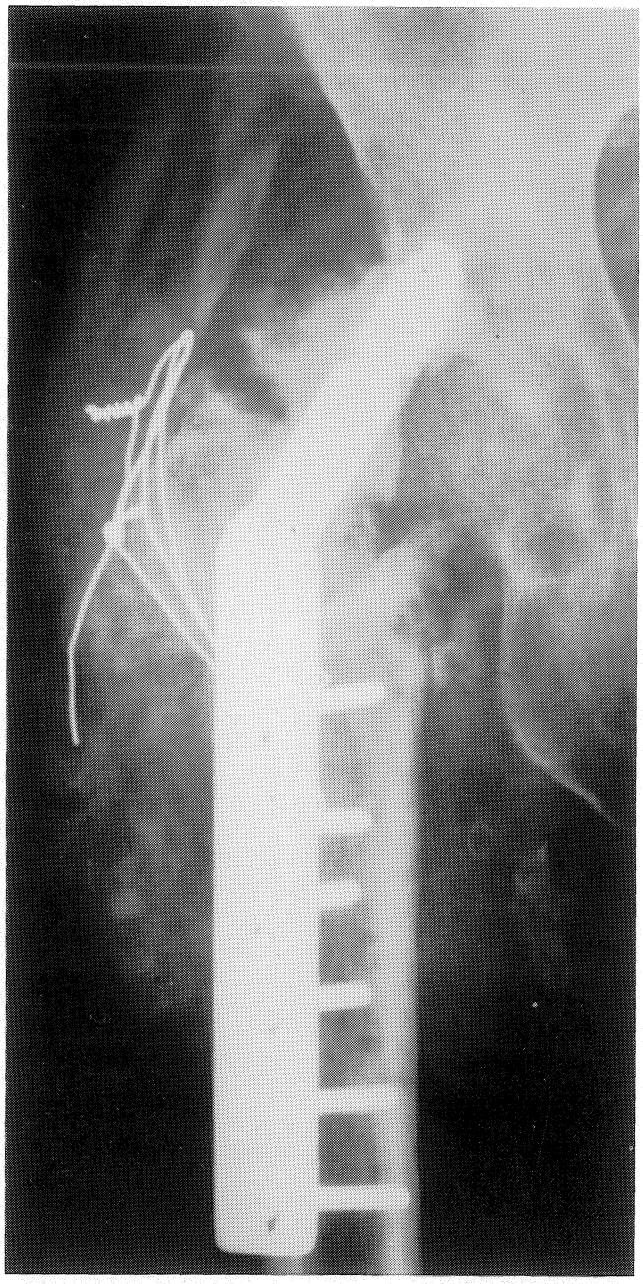

Figure 5
Radiograph at seven weeks. The head remains reduced but fixation has failed.

out of the acetabulum; however, a stable reduction could not be obtained after resecting these fragments. Only after decreasing the neck-shaft angle from 145 degrees to 135 degrees were we able to maintain reduction of the hip. The preoperative varus positioning (Fig. 1) allowed soft tissue (abductor) contracture. Repositioning the fracture from varus to 145 degrees increased muscle tension across the hip joint until dislocation occurred. In retrospect, intraoperative femoral shortening might have avoided this complication. We do not feel the same valgus positioning would have caused this dislocation if this were an acute fracture. Sarmiento, in reporting the results of 250 valgus osteotomies, did not include dislocation as a possible complication[4,5]. Most authors who have noted dislocation after internal fixation of intertrochanteric fractures have found it to be a delayed complication of hip joint sepsis[2]. Malcolm and Schatzker felt that soft tissue contracture combined with excessive valgus positioning and abductor spasm were responsible for the complication. We recommend, in revision cases where fixation failure has caused preoperative varus, that the hip be examined out of traction in adduction to check for stability. Identification of instability would suggest the need for a smaller angled side plate, femoral shortening or abduction immobilization during early healing.

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SCIATICA: AN UNCOMMON ETIOLOGY

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INTRODUCTION

One of the most commonly encountered benign bone tumors is the osteochondroma[1,2,3,4]. Multiple osteochondromatosis or multiple hereditary exostoses is one of the more common bone dysplasias. Osteochondromas are typically found on long tubular bones and less commonly involve the axial skeleton[2]. This is a report of a 22 year old white male with a history of multiple osteochondromatosis with a large pedunculated osteochondroma on the posterior aspect of the femur causing sciatica.

CASE REPORT

This 22 year old white male had a history of multiple osteochondromatosis and developed radiating pain and intermittent numbness down the left anterior medial thigh into the leg and foot. This was aggravated by activity and relieved by rest. He had leg pain with cough, sneeze and valsalva. He was unable to recall antecedent injury. A year and a half prior to this presentation, he had had an exostosis excision from the distal right femur.

The physical exam showed a healthy, well developed 22 year old white male with normal painless range of motion of the spine except for painful left lateral bending. Straight leg raise was positive and accentuated by dorsiflexion of the left foot. He did have sciatic notch tenderness as well as tenderness to palpation along the posterior thigh. Reflexes were normal and symmetric in both the knees and ankles. He had no motor deficit. The patient was treated conservatively with anti-inflammatory medication and continued to have radicular symptoms. CT scan of the L4-5 disc demonstrated a prominent annulus. There was narrowing of the spinal canal to 12 mm. at the L4-5 level. The L5-S1 disc level demonstrated no herniation or stenosis. A CT scan of the thigh demonstrated a large osteochondroma of the posterior medial aspect of the proximal left femur (Figure 1).

Due to the patient's continuing symptoms, surgical removal of the osteochondroma was indicated. A Ludloff approach was used to remove the large pedunculated exostosis which measured 8 cm. X 5 cm. X 5 cm. (Figure 2). The patient had no residual neurologic symptoms.

DISCUSSION

Osteochondromas are cartilage capped bony growths produced by progressive enchondral ossification from aberrant growth plate cartilage rests. The cortex of an osteochondroma is continuous with that of the bone from which it arises[1]. Osteochondromas can occur as solitary lesions with no known genetic component whereas multiple lesions occur in hereditary multiple exostoses also known as multiple osteochondromatoses and diaphyseal aclasis[2]. The incidence of malignant transformation of solitary osteochondroma is 1%. The incidence of malignant transformation in multiple osteochondromatoses is approximately 10%[1,3].

Osteochondromatoses are commonly known to cause mechanical type pain and occasionally nerve impingement[2].

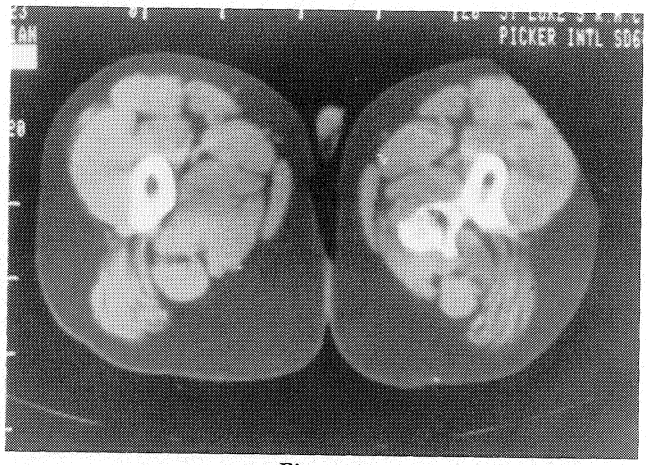

Figure 1
CT scan showing large osteochondroma of the posterior medial aspect of the proximal left femur.

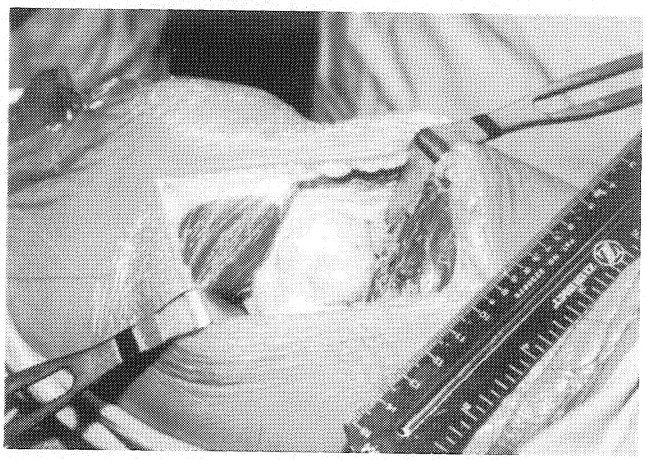

Figure 2
Intraoperative appearance of left femoral exostosis.

This case represents an uncommon cause of sciatica from a posterior pedunculated femoral osteochondroma.

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A BRIEF HISTORY OF FINGER ARTHROPLASTY

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INTRODUCTION

Almost every orthopaedic surgeon has had the opportunity to evaluate and treat patients with osteoarthritic and rheumatoid hand deformities. Distal to the wrist, these deformities are found primarily in the metacarpophalangeal (MCP) and proximal-interphalangeal (PIP) joints. Arthroplastic intervention has now become commonplace for severe deformities of these joints; however, this has not always been true. As recently as 1954, Smith-Peterson was advocating nonoperative management for arthritic hand deformities, claiming that surgical correction of more proximal joints, such as the elbow and shoulder, would prevent the "gravity" induced deformities commonly found in the hand [35].

In the rare cases of arthritic distal-interphalangeal (DIP) joints of the hand symptomatic enough to warrant operative intervention, arthrodesis has been recommended as the procedure of choice. Also, reports of silicone rubber arthroplasty have been published[2]. Arthrodesis of the more commonly affected PIP or MCP joints carries significant functional limitations and has not been recommended under most clinical circumstances. Over the past 70 years, a significant effort has been made to develop surgical procedures that will provide patients with painless and functional MCP and PIP joints. The purpose of this manuscript is to outline the history of the development of these procedures.

RESECTION/INTERPOSITION ARTHROPLASTY

Prior to 1914, arthrodesis was the most commonly employed treatment for severe arthritic conditions of the PIP joint. Bunnell felt that this was the treatment of choice in an ankylosed and malpositioned joint (6). However, the period around World War I witnessed a new demand for improving functional range of motion in patients with severe post-traumatic degenerative changes in the PIP joint. Numerous anecdotal reports surfaced from 1914 through 1925 describing limited experience with resection arthroplasty with various soft tissue interposition techniques.

In 1950, Liebolt reported his results from procedures to improve motion in ankylosed PIP and MCP joints in soldiers injured in World War II[28]. He refined the procedure into two stages. The first stage was a capsulectomy, in which the collateral ligaments were also divided. When there was obvious destruction of the articular cartilage radiographically, resection of either the proximal or distal surface of the joint with interposition of ulnar fascia was indicated. A postoperative range of motion from 30 degrees extension to 70 degrees flexion could be anticipated. Carroll (1954) described his resection arthroplasty technique without soft tissue interposition combined with long-term postoperative digital traction in 30 patients with ankylosed PIP joints[11]. His prerequisites for surgery included severe joint deformity, an intact extrinsic tendon apparatus and strong patient motivation. He reported fair to good results in 80% of his patients, with instability not being a major postoperative complaint.

Resection arthroplasty of the MCP joint was first reported by Fowler in 1962 and followed by Tupper and Vainio, using a variety of resection and interposition techniques (Fig. 1)[18,41]. These techniques offer a satisfactory method of reducing pain in a severely degenerated MCP joint, yet they are considered to result in a high degree of joint instability and have been reserved largely for salvage situations.

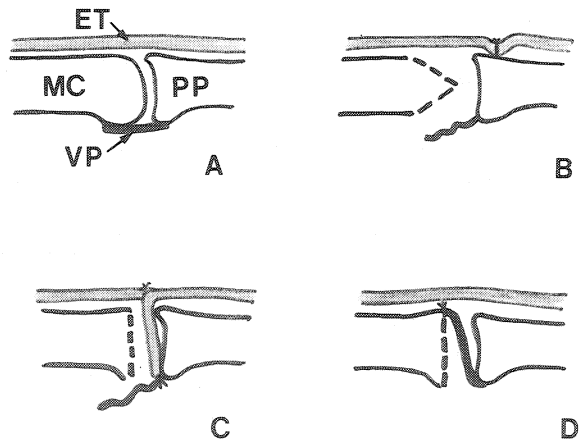

Figure 1 A-D
(A): Normal joint schematic showing the metacarpal (MC), proximal phalanx (PP), extensor tendon (ET) and volar plate (VP). (B-D): Schematic of metacarpophangeal joint resection arthroplasties. The dotted line represents the metacarpal osteotomy. (B) Fowler. (C) Vainio. (D) Tupper. (after Flatt, A.E. "The Pathomechanics of Ulnar Drift" Final Report, Social and Rehabilitation Services Grant No. R02226M, 1971, p. 103.)

TRANSPLANT ARTHROPLASTY

The concept of transplanting articular surfaces in the finger joints was introduced by Wolff in 1910. He reported excellent results in a female with tuberculosis involving one of her proximal phalanges in whom he resected the entire proximal phalanx and autotransplanted the proximal phalanx of the 2nd toe. One year later, Goebel reported removing an entire proximal phalanx for treatment of an enchondroma, replacing it with an autologous transplant of the 2nd toe proximal phalanx. Postoperatively, the patient was noted to have active flexion of all finger joints, but was noted to be quite limited functionally. Oeleker performed an entire joint cadaver transplant in a patient with an ankylosed PIP joint caused by a gunshot wound, obtaining excellent results. In 1948, Riordan and Graham reported several cases of autologus partial transplants, in which the metacarpal heads were resected and replaced by 4th metatarsal heads. They reported satisfactory ranges of motion and no radiographic evidence of degenerative changes at one year follow-up. In 1954, Graham performed an entire thumb MCP joint autotransplant in a three year old boy injured in a washing machine wringer accident. The joint was replaced with a 4th MTP joint, and at 22 month follow-up examination revealed satisfactory range of motion and continued growth at the physeal plates.

"CAP" ARTHROPLASTY

Encouraged by Smith-Peterson's results of Vitallium cup arthroplasty of the hip, Burman performed a Vitallium "cap" arthroplasty for a severely degenerated metacarpal head in 1940[9]. In 1943, a methylmethacrylate "cap" for degenerated metacarpal heads was used in two patients by Burman and Abrahmson[10]. They reported good results, but implied that lateral instability of the MCP joints remained a significant problem.

METALLIC HINGED PROSTHESES

Largely in response to intraoperative technical problems, questionable long-term results, and residual lateral instability in existing finger arthroplasties, Brannon and Klein developed a metallic hinged implant for the MCP joint (Fig. 2)[7]. The device was manufactured from titanium, and consisted of noncemented proximal and distal stems articulating through a hinge stabilized by a screw. Problems with longitudinal rotation *in vivo* prompted revision of the stems into a more triangular cross-sectional shape. Additionally, significant bony resorption occurred around the stems, which prompted further revision allowing fixation to the adjacent bone with staples. They reported their experience in 14 patients at a maximum of three year follow-up. All patients were felt to have functional ranges of motion and no lateral instability. All patients showed radiographic evidence of bone resorption and "settling" of the prosthesis. Two patients experienced asymptomatic screw loosening, and one patient was noted to have asymptomatic penetration of one of the stems into an adjacent joint space. One patient complained that small metallic objects were attracted to his finger post-operatively. No evidence of tissue reaction to titanium was noted.

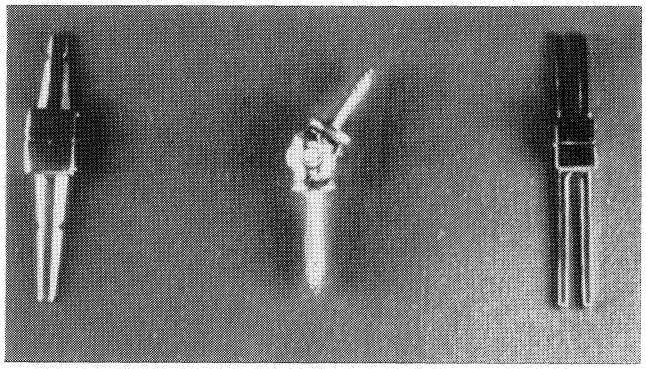

Figure 2
Metallic hinged prostheses. Left to right: Brannon, Richards and Flatt designs. There is no clinical data available on the Richards prosthesis. (Courtesy of Department of Orthopaedic Surgery Archives, University of Iowa College of Medicine.)

Flatt felt that the rotational instability of the Brannon prosthesis remained unacceptable, in spite of the revision of stem shape. In 1959, he began implanting a double-pronged, hinged metal prosthesis for MCP and PIP joints (Fig. 2). This design theoretically reduced the tendency of longitudinal rotation and allowed bony ingrowth between the prongs. At 14 years follow-up of 167 MCP and 75 PIP implants, 10% of the prostheses were removed because of infection, failure of the screws, fracture of prongs, skin breakdown, periarticular fibrosis, or severe settling and bone resorption[17]. Independent reviews by Zachariae as well as by Girzadas and Clayton confirmed major problems due to significant bone resorption with subsequent shift of the Flatt prostheses[21,43]. Blair et al. reviewed patients with Flatt MCP and PIP prostheses and reported high patient satisfaction in spite of major complications[5,14].

POLYETHYLENE-METAL PROSTHESES

Almost simultaneously, several "2nd generation" finger joint prostheses appeared, each made of a proximal stem composed of polyethylene which articulated with a metallic distal stem (Fig. 3). Each stem was designed to be cemented in the medullary canal of the respective bone. The St. Georg-Buchholz design incorporated a fixed center of rotation with two models differing only in the range of allowed radial-ulnar deviation (Fig. 3). Gillespie et al., in their study of the characteristics of several "2nd generation" prostheses in cadaver fingers, found that the passive range of motion of the St. Georg-Buchholz prosthesis

averaged 45 degrees to 160 degrees and allowed 5 to 15 degrees of radio-ulnar deviation[20]. Also noted was significant cold flow of the polyethylene hinge resulting in permanent deformation. The Schultz prosthesis had a changing center of rotation by incorporating a slot in the articulation of the polyethylene component, which allowed the distal metal articulation to glide as the joint was rotated (Fig. 3). Gillespie et al. measured an average range of motion from 0 to 90 degrees and 8 degrees radio-ulnar deviation in implanted Schultz prostheses in cadaver fingers[20]. The Strickland prosthesis offered a fixed center of rotation and an ulnar "shoulder" in the polyethylene articulation which theoretically resisted ulnar deviation of the distal metallic stem (Fig. 3). Gillespie et al. reported a range of motion averaging 5 to 90 degrees, and found an average range of radial-ulnar deviation of 15 deg. in extension and 0 degrees in flexion of the prosthesis[20].

In 1964, Steffee et al. designed a prosthesis with a Teflon proximal stem which was later revised to high-density polyethylene[29]. Following trials in cadavers and chimpanzees, this prosthesis, Model I, was implanted in humans in 1968. Production was delayed until 1974, in part because of the initial success of the silastic joint replacements. Model II (Fig. 3), developed in the interim, included a volar offset center of rotation to increase the extensor moment arm (to prevent recurrence of boutonniere deformities) and a longer distal stem to counter the tendency to tilt volarward. Clinical trial reports by Steffee et al. included 160 Model I implants and 272 Model II implants, with 55% and 86% satisfactory results respectively. Overall, pain relief was found in 94% of the trials and correction of ulnar deviation in 87%[38]. At two year follow-up, there were 2 infections, 2 dislocations and 2 fractured polyethylene components at the level of the articulation. Radiographic loosening of the distal component was noted in 18% of the fingers.

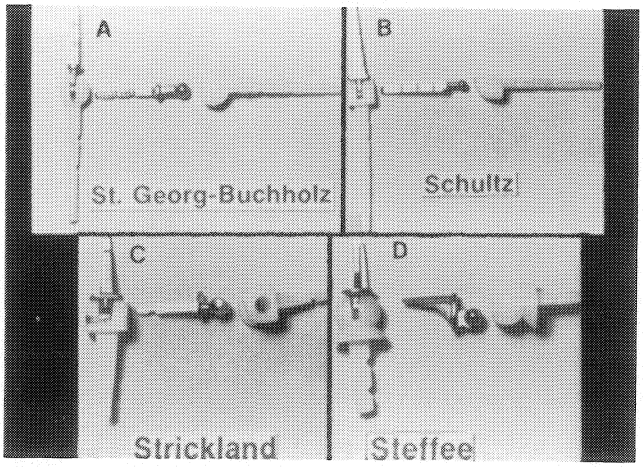

Figure 3A-D
Polyethylene-metal prostheses. (Reprinted from Gillespie et al., J. Hand Surg., 6:512, 1979.)

Overall, Gillespie et al. found that the polyethylene-metal prostheses were unforgiving in terms of placement error, that the polyethylene had a strong tendency to deform under physiologic loading conditions, and measured fingertip forces generally fell below baseline levels[20].

SILICONE RUBBER PROSTHESES

Silicone rubber (silastic) was introduced as a material for use in finger arthroplasty almost simultaneously by Swanson and Niebauer in the early 1960's. Swanson cited several theoretical advantages for using Silastic over other materials including heat stability, durability, excellent flexion characteristics and force dampening properties, biologic inertness, low cost of production, and ease of handling[36]. It was noted, however, that Silastic tears easily when its surface is lacerated. Swanson began development of his MCP and PIP implants in 1962, with over 10,000 implants placed less than a decade later. The basic design of the Swanson MCP and PIP implants is a single Silastic unit with tapered proximal and distal stems and a dorsally offset flexion region (Fig. 4). Swanson described two processes that occur with implantation, theoretically enhancing performance of the implant. The first process is "encapsulation" or development of a fibrous joint capsule surrounding the implant which enhances joint stability. The process of encapsulation occurs in part because of the "intrinsic dynamic spacer" function of the implant. The second process is the "piston effect", or the gliding motion of the stems within the medullary canals during flexion and extension of the joint. Theoretically, the piston effect increases the lifespan of the implant because forces are dispersed over a broad area of the implant. Gliding also allows a greater range of motion. In his first report of nearly 4000 MCP implants, Swanson listed 96 complications, including 1% fractures, 0.7% infections and 0.8% dislocation rates[36]. Similar complication rates were noted for the PIP arthroplasties[37]. Despite the low complication rate reported for both implants, 10% were revised in a series of patients followed for at least one year. In other studies, Swanson-design finger joint implants were shown to have fracture rates ranging between 9% and 44%, recurrence of ulnar drift in up to 43%, low infection rates, up to 24% incidence of peri-implant bone resorption and evidence of silicone synovitis. Common to all reports was a sense of high patient satisfaction with the procedure[1,3,4,15,39,40]. In their study of Swanson-design PIP joint implants, Dryer et al. found post-operative swan-neck or boutonniere deformities in 37% of patients evaluated[14]. Gillespie et al., in their biomechanical study of finger joint implants found the range of motion to average -30 to 90 degrees with an unpredictable range of radio-ulnar deviation[20] and an erratic center of rotation. The majority of motion occurred at the bone-implant interface,

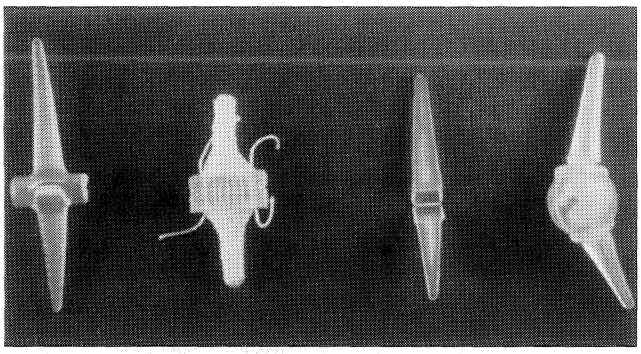

Figure 4
Silicone rubber prostheses. Left to right: Swanson, Niebauer, Reis-Calnan and Nicolle-Calnan designs. (Courtesy of Department of Orthopaedic Surgery Archives, University of Iowa College of Medicine.)

rather than at the hinge region of the implant. Interestingly, they also found that the implant behaved independent of its orientation. The measured parameters were statistically unchanged with the implant right side up, upside down or rotated axially 90 degrees. Recently, the Silastic has been upgraded in an effort to reduce the incidence of fractures propagated through surface lacerations. Additionally, metallic grommets have been introduced to reduce shearing at the bone-implant interface.

Niebauer et al. began work on a Dacron covered Silastic finger joint implant in 1966, with the idea for this construction stemming from the clinical success noted in Dacron covered Silastic used in prosthetic heart valves[31]. The first design consisted of wide stems covered with Dacron leading to a bare hinge region (Fig. 4). Of 165 MCP and 13 PIP implants utilizing the first design, complications included infections, buckling of the prosthesis and resorption at the bone-implant interface. Clinically, MCP total range of motion averaged 40 degrees. The basic design was revised narrowing the stems near the hinge region, increasing the size of the hinge region and providing Dacron tethers for bony fixation of the prosthesis. Of 89 MCPs implanted with the revised model, Niebauer et al. reported recurrent ulnar drift of less than 20 degrees in 64% (greater than 35 degrees in 20%), pain relief in 84%, subluxation in 58%, severe bone destruction in 43%, and a 12% major complication rate including infection and unacceptable subluxation[12]. Patient satisfaction was high with 64% noting significant improvement after implantation. In a follow-up of 441 MCP implants, Goldner found similar results, including a 68% patient satisfaction rate; however, he noted significant lateral instability[22]. In their biomechanical study, Gillespie et al. found an erratic center of rotation and consistent buckling of the implants at the bone-implant interface (exaggerated with the revised model)[20]. Additionally, after physiologic loading a 20% stretch of the Dacron suture used to fix the implant to the bone occurred, which they felt would effectively negate any bone fixation attained at the time of implantation.

The Reis-Calnan finger joint implant incorporated polyprophylene stems to increase their stiffness and strength reducing the tendency for the prosthesis to buckle at the bone-implant interface[33]. Nicolle and Calnan modified this design by adding a Silastic capsule at the hinge region to reduce soft tissue entrapment and to increase the moment arm for the extensor tendons crossing the joint (Fig. 4)[30]. In their report of 68 implants at less than one year follow-up, there were no infections, fractures or cases of loosening; however, three joints remained painful. Other designs incorporating Silastic materials are appearing, such as the Sutter Biomedical Inc. MCP implant. It claims to have an enhanced range of motion, increased stability, and improved mechanical advantages. At the time of this writing, however, no clinical trials are available for review.

NEW DESIGNS

Dissatisfaction with the performance of currently available finger joint implants has motivated numerous investigative groups to design new implants, utilizing more sophisticated design techniques and materials. For example, Weightman et al. have published a new MCP prosthesis design, utilizing sophisticated biomechanical input parameters and testing systems[42]. The design incorporates paired prostheses joined by common hinge pins which theoretically reduce the tendency for recurrent ulnar drift to occur, reduce the torque applied to the individual prostheses, and retard loosening of the implants. No clinical trials have been reported to date. As in the hip, knee, shoulder and elbow, the concept of biologic fixation of prostheses through bony ingrowth has been incorporated into the design of new finger joint prostheses. Doi. et al. have designed a hinged prosthesis composed of alumina ceramics and high density polyethylene[13]. Preliminary clinical results on 13 patients at one to three year follow-up showed no fractures, dislocations, or infections. All patients were noted to have improved ranges of motion and complete relief of pain. Nine patients had returned to heavy labor jobs. Beckenbaugh et al. have been developing a semiconstrained cementless pyrolytic carbon MCP prosthesis, designed with a deep spherical cup in the distal component which provides stability in the anteroposterior direction. Because of the lack of intrinsic lateral stability, they advise limiting its proposed use to patients with minimal joint deformities[2]. Currently, no clinical data is available on this implant.

CONCLUSION

The number of surgical options available for treatment of arthritic metacarpophalangeal and proximal interphalangeal joint deformities is impressive. However, it seems that much work remains to improve further function and

biocompatibility of joint implants. The Silastic joint implants are currently widely used with proven clinical success, low tissue reactivity (in most circumstances), relative low cost and high patient satisfaction. It will be very interesting to follow the developmental progress of the newest generation of implants, which seem to have in common cementless fixation and relatively constrained kinematics.

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GANGLION CYST OF BONE ASSOCIATED WITH BONE NECROSIS

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INTRODUCTION

The etiology of ganglion cysts of bone has been debated. One of the proposed etiologic factors has been focal bone necrosis; however, no case has reported pathologic evidence of bone necrosis. This paper discusses a case of a ganglion cyst in the lateral condyle of the distal femoral epiphysis associated with local bone necrosis and osteochondritis dissecans.

CASE REPORT

B.H., a fifteen year old boy, gave a one and one-half year history of right knee pain with activity and mild knee stiffness. No prior history of injury or previous symptoms existed. Physical examination revealed an antalgic gait on the right with one-half inch quadriceps atrophy. The anterior aspect of the lateral femoral condyle was tender. Roentgenograms revealed a multi-loculated radiolucent lesion in the posterolateral portion of the right distal femoral epiphysis (Figs. 1A-B). A zone of radiodensity surrounded the lesion and flattening of the posterior portion of the subchondral cortex of the lateral femoral condyle could be seen on the intercondylar notch view (Fig. 1C). All laboratory studies were normal.

A biopsy performed through a window in the lateral cortex of the epiphysis showed grossly dense bone surrounding the lesion. The cyst proper had a brownish-grey wall containing viscous clear fluid. The cyst wall separated easily from the surrounding bone. No obvious communication existed with the knee joint. An arthrotomy of the knee joint was not performed.

Microscopic examination of the cyst wall showed loose fibrous tissue with areas of focal myxomatous degeneration (Figs. 2A-B). The bone immediately surrounding the cyst contained myxomatous tissue. Fragments of necrotic bone surrounded by active osteolytic fibrous tissue were also seen. (Fig. 2C) Microscopic examination of the radiodense bone revealed trabeculae of dead bone surrounded by repair bone (Fig. 2D). The marrow in this zone showed an increase in fibrous tissue with a decrease in normal appearing hematopoietic cells.

Radiographically, the lesion was healing at six weeks (Figs. 3A-C). However, he returned six months postoperatively with symptoms of recurrent effusion and giving way. A loose body was palpable in the suprapatellar pouch. An arthrotomy was then performed and a three by

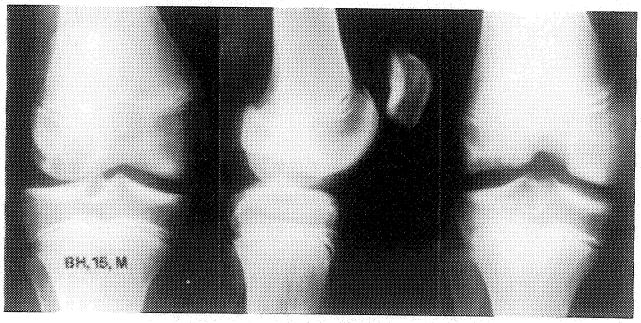

Figure 1a Figure 1b Figure 1c

Figure 1A
Anterior-posterior view of the right knee shows multicystic radiolucent lesion with surrounding zone of radiodensity in distal femoral epiphysis.

Figure 1B-C
A lateral view of the right knee shows condylar flattening. Intercondylar view of the right knee shows flattening of the distal and posterior portion of the subchondral cortex.

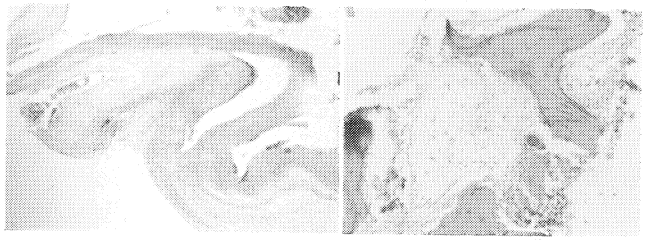

Figure 2A
Microscopic section of cyst wall from distal femoral epiphysis (10x).

Figure 2B
Microscopic section of cyst wall showing area of myxomatous degeneration (40x).

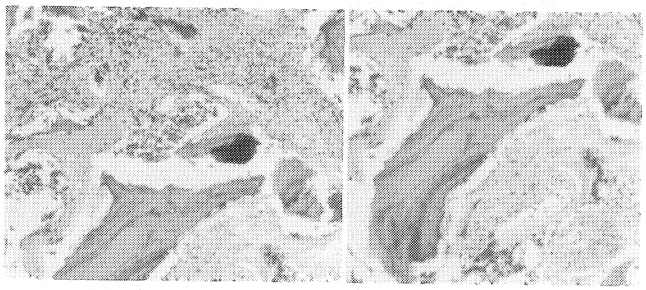

Figure 2C
Microscopic section of necrotic bone surrounded by fibrovascular stroma from the cyst wall (40x).

Figure 2D
Fragments of necrotic bone are undergoing repair (100x).

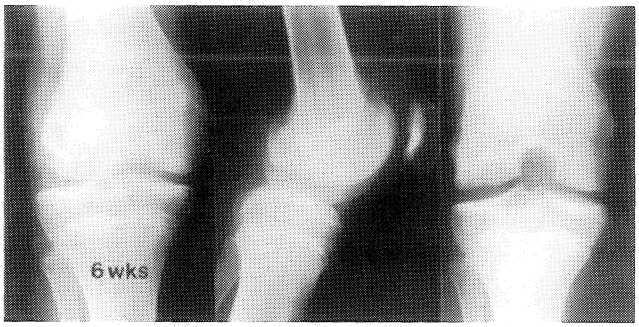

Figure 3a Figure 3b Figure 3c

Figure 3A-C
AP, lateral and intercondylar views of right knee six weeks following curettage shows early healing of the ganglion cyst.

four centimeter cartilaginous loose body removed. The articular surface of the lateral femoral condyle had a large defect in its posterior central portion which was covered by fibrocartilaginous repair tissue.

Following removal of the loose body, his right knee improved symptomatically and radiographically. The cyst continued to show evidence of healing at follow-up three years later (Figs. 4A-C). During the postoperative period, he complained of mild left knee pain, however physical exam was normal. Radiographs of the left knee showed a typical area of osteochondritis dissecans involving the left medial femoral condyle and slight narrowing of the medial joint cartilage shadow (Fig. 5). He has had no further left knee symptoms and has not required further surgical therapy. A skeletal survey of other joints revealed no other sites of bone necrosis.

DISCUSSION

Hicks in 1956 first described the occurrence of cystic lesions in bone which he felt arose from synovial metaplasia of bone or herniation of synovial tissue into subchondral bone[1]. Since that time, subsequent reports have suggested other etiologic factors[2]. Woods suggested that a localized vascular disturbance might cause cystic lesions in bone similar to those found in Bugnion's study of wrist bones; however, he found no microscopic evidence of necrosis in the seven cases he reported[3,4]. Rhaney and Lamb studied osteoarthritic cysts and felt that focal bone necrosis preceded bone repair which ended in cyst formation[5]. They studied thirty-six femoral heads and found that direct synovial intrusion, as proposed by Landells, was not necessary for cyst formation. However, when synovial intrusion did occur it served to expand the cyst and prevent osseous healing[6].

A large portion of the lateral femoral condyle in this case had probably undergone necrosis, although portions of the condyle had successfully undergone repair. The posterior central portion of the epiphysis contained a large multilocular cyst. Microscopic examination of the cyst wall was typical of a ganglion cyst of bone as described by Schajowicz and other authors[7]. Immediately surrounding the cyst was necrotic bone which showed various stages of repair.

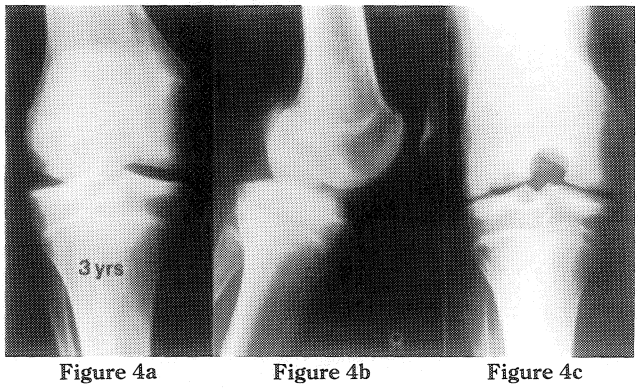

Figure 4a Figure 4b Figure 4c

Figure 4A-C
AP, lateral and intercondylar views of right knee three years following curettage shows persistent flattening of the subchondral cortex and narrowing of the medial joint cartilage shadow.

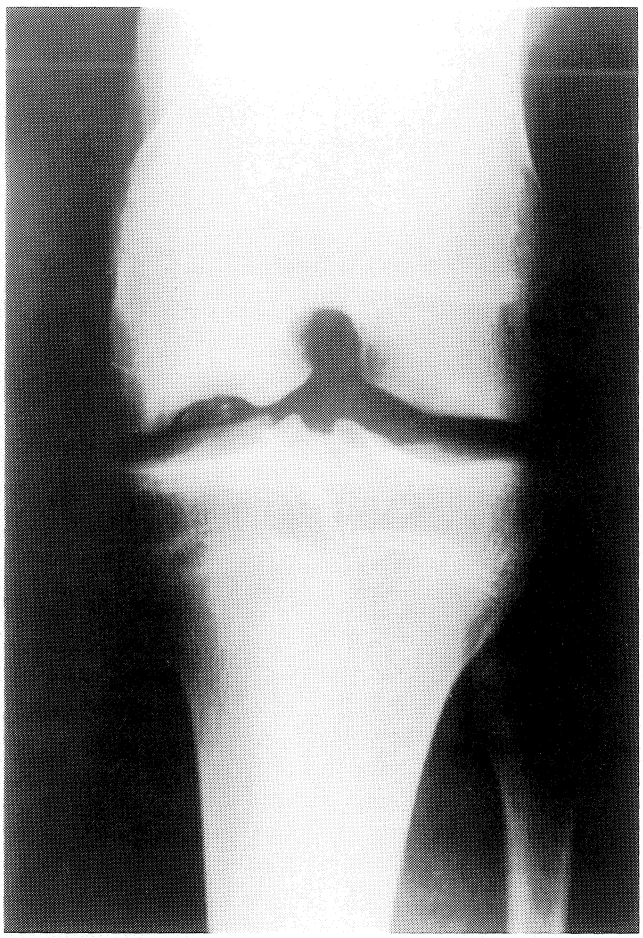

Figure 5
Intercondylar view of the left knee showing osteochondritis dissecans of the medial femoral condyle. Note cystic changes in the repair zone along the necrotic fragment.

The etiologic factors for the formation of this ganglion cyst of bone are related to bone necrosis. The cyst may have developed secondary to a failure in the repair process. Development of this cyst may be similar to degenerative cysts although there were no degenerative changes in this knee at presentation.

Following biopsy of the cyst, a large loose body formed from a portion of the articular cartilage and subchondral bone. The history strongly suggests osteochondritis dissecans of the right lateral femoral condyle. The typical radiographic lesions of osteochondritis dissecans were also present in the opposite knee.

Only two previous cases of ganglion cysts have been reported in the epiphysis prior to epiphyseal closure. The distal femur has been reported as the site of ganglion cysts four times from approximately one hundred reported cases in the English literature.

CONCLUSION

A skeletally immature patient with a ganglion cyst of the distal femur is reported. Microscopic examination showed definite evidence of necrosis of bone about the periphery of this cystic lesion and probable osteochondritis dissecans of the femoral condyle. Osteochondritis dissecans of the opposite knee was observed coincidentally. This case represents the first direct evidence that necrosis of bone followed by repair may precede the formation of ganglion cyst of bone.

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A COMMON FOREFOOT PROBLEM ASSOCIATED WITH HALLUX VALGUS

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INTRODUCTION

Hallux valgus is a common deformity. Relatively little has been written on less common second metatarsophalangeal joint problems that can arise secondarily to hallux valgus. This area can be a distinct source of debilitating pain. The diagnosis requires an awareness of the problem and an appreciation for its variable symptoms.

ETIOLOGY

As hallux valgus progresses, the great toe may underlap the second toe. This sustained upward pressure indirectly applies stress to the second MTP joint. This continual abnormal pressure transmitted through the joint can eventually cause stretching or insufficiency of the second MTP capsular structures. Surrounding ligaments and other soft tissue structures are involved resulting in subluxation or frank dislocation. This subluxation is often very painful and may represent the patient's chief complaint, in spite of the presence of more obvious deformities.

As the deformity increases, shoe impingement on the dorsal aspect of the toe causes a painful callus. The toe box exerts pressure through the toe and second metatarsal, producing increased pressure on the plantar aspect of the second metatarsal head. Further, as the second toe no longer bears weight, more load is placed proximally onto the second metatarsal head. This may result in a very painful plantar callus or corn.

Aside from the callosities, the joint itself develops inflammation manifested as capsulitis or synovitis. This is the situation where a steroid injection is often inappropriately administered. The medication may temporarily relieve the symptoms, but in our experience causes accelerated weakening of the soft tissue constraints and allows further, more rapid deterioration and subluxation of the joint.

DIAGNOSIS

The diagnosis is not difficult. The patient complains of pain in the region of the second MTP joint. Observation and palpation generally reveal the cause of the problem. If the problem is long-standing, callosities will be present. By palpating the second MTP joint, two different facts can be ascertained. The first is the presence or absence of pain between the metatarsal heads. This would be more suggestive of an interdigital neuroma, which might present with a similar array of symptoms. The second important step is to gauge the integrity of the second MTP joint. Fixing the metatarsal head with one hand, the base of the proximal phalanx can be manipulated to gauge the amount of joint laxity that is present. This has been previously described as the "Lachman Test of the metatarsophalangeal joint"[1]. It is this test which confirms the diagnosis. Plain roentgenograms, either with or without this stress can also demonstrate the instability of the joint.

TREATMENT

The treatment of any problem in orthopaedics should be directed at the causative agent, thereby removing or correcting the underlying pathophysiology. This is especially true in this instance. The problem is with the second MTP joint, and one might be tempted to solely address this anatomic area. The second toe problems are caused by the pre-existent hallux valgus and this is the deformity that must be addressed if a successful outcome is to be reached in treatment of the second toe deformity.

The first step is to perform a bunionectomy, usually with the Keller technique. This very effectively realigns the great toe, removes the upward force to the second toe and relieves the pressure on the second MTP joint. The second MTP joint itself then should be reduced into anatomic alignment. Often, because of the long-standing dislocation, this is physically impossible. In this case, the joint must be decompressed in order to allow proper realignment, best done by removal of a portion of the proximal phalanx sufficient to allow safe correction. The second toe is then secured in position with a K-wire. Though this procedure does not produce a toe with normal motion, the abnormal forces have been neutralized, and pain relief is satisfactory. The K-wire is removed at three weeks postoperatively.

CASE REPORT

A sixty-one year old white female was having pain beneath her second metatarsal head with a callus on the plantar surface. She was seen by a podiatrist who told her that she had a "pinched nerve in her foot." A cortisone shot

was administered with the patient having a systemic reaction, possibly to the cortisone, resulting in foot, truncal, and facial swelling. This swelling took several weeks to resolve while the foot pain increased. She began noticing the pain on the dorsum of her foot as well. She noticed that her second toe was drifting dorsally more rapidly than previously. She had no pain in the first MTP joint.

Physical examination revealed forty-five degree hallux valgus bilaterally with fifty percent underlapping as well. She had splay foot deformities along with short first metatarsals bilaterally. When her right second metatarsal was stabilized, the base of the proximal phalanx was easily subluxed dorsally at the MTP joint. She stated that this duplicated a portion of the discomfort she was having. X-rays were obtained which demonstrated dorsal subluxation of the second MTP joint (Figs. 1, 2, and 3). The patient had a Keller bunionectomy along with excision of the base of the proximal phalanx and straightening of the second toe. She is now pain free and has an excellent functional result.

SUMMARY

Subluxation of the second metatarsophalangeal joint is a common and underdiagnosed cause of forefoot pain. The diagnosis and treatment is illustrated with a typical case presentation.

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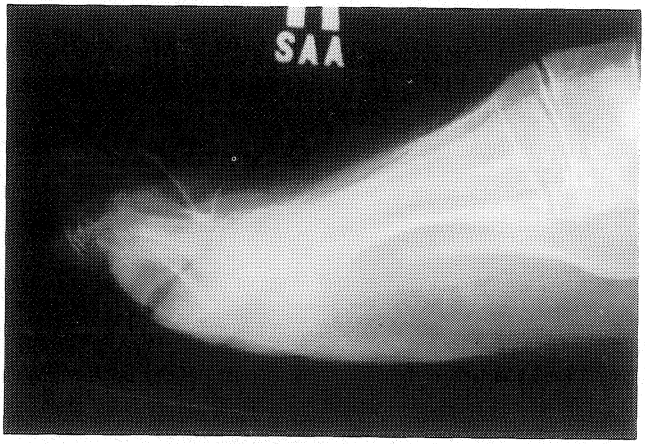

Figure 1
Lateral view of the right foot clearly showing dorsal subluxation of base of proximal phalanx of second toe.

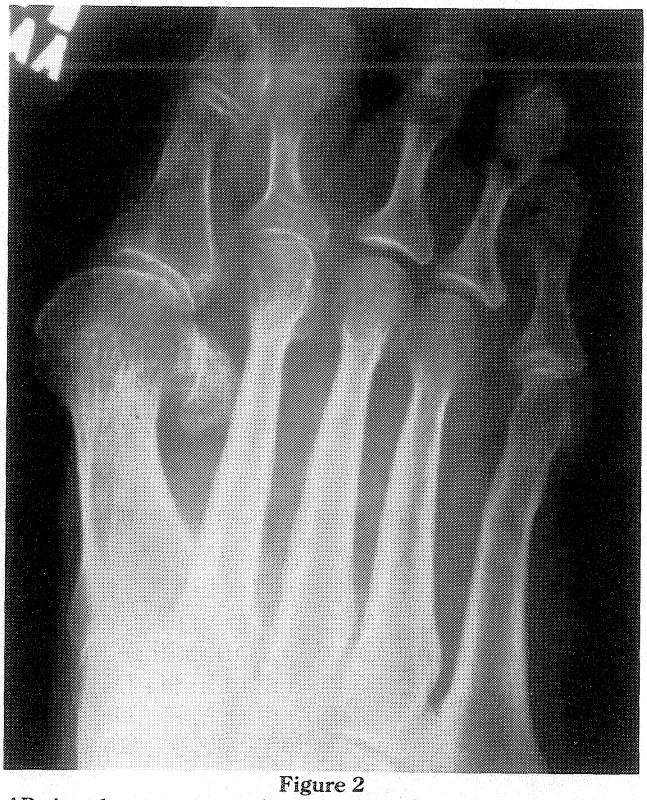

Figure 2
AP view demonstrates subluxation of base of proximal phalanx of second toe.

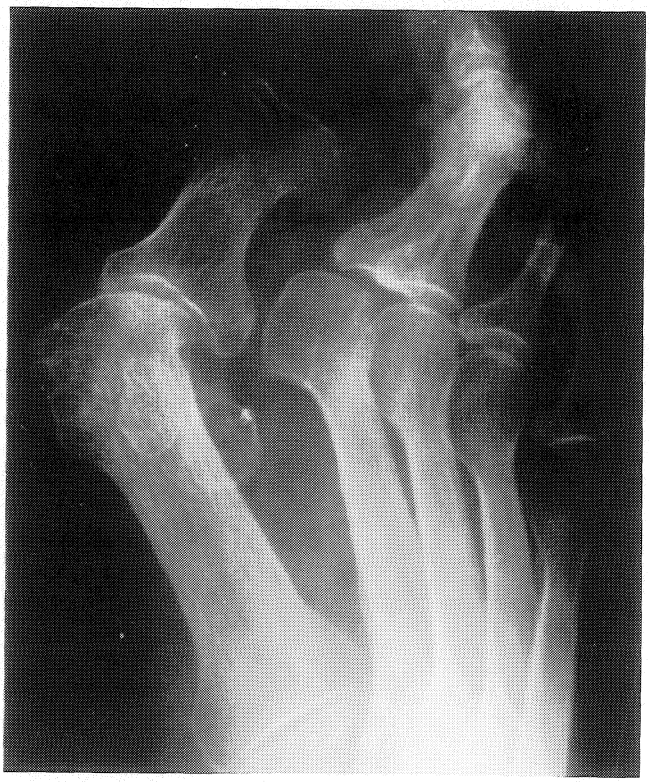

Figure 3
Oblique view also demonstrates the subluxation.

IDIOPATHIC POSTMENOPAUSAL OSTEOPOROSIS—AN OVERVIEW

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Osteoporosis, initially described in the 1920's, is a generalized disorder of bone characterized by a decreased bone density with the remaining bone having normal architectural and chemical content. As mankind has controlled infectious and other acute diseases, life expectancy has increased allowing diseases such as osteoporosis to become clinically evident. Our understanding of osteoporosis as well as its treatment and prevention continue to evolve.

The exact cause of postmenopausal osteoporosis is not determined but a few facts are known. Estimates place the prevalence of the disease at 15 percent of the white female population over the age of 65[11]. Maximum bone density is reached sometime early in life around the age of 25. From that point to the time of menopause, women lose bone density at a rate of .1%—.3% per year. After menopause, there is an acceleration of bone loss to .7% - 1% per year. Postmenopausal osteoporosis is a problem of bone remodeling and not a problem of calcium homeostasis. Although all osteoporotic patients exhibit net bone loss, iliac crest biopsies reveal nonhomogenous histomorphology. Ten percent of the biopsies show increased remodeling while thirty percent show decreased osteoblastic activity. Sixty percent of the biopsies have a pattern indistinguishable from nonosteoporotic subjects[11].

Epidemiologic studies have yielded some consistent phenotypic features of osteoporosis. Females are more commonly involved than males. People of Northwest European ethnic background possess a predisposition for the disease, while it is uncommon in blacks. Obese people seem at less risk because they have higher levels of circulating estrogen which has an indirect effect on bone density. Other common features include a fair complexion, freckles, blond hair, ligamentous laxity, scoliosis and inactivity.

The osteoporotic patient is at greater risk for a fracture due to accelerated loss of bone. There exists a critical bone density, the fracture threshold, below which most osteoporotic patients eventually fall[24]. The likelihood of reaching the fracture threshold in an individual depends on several factors: the peak bone density, the rate of bone loss and the duration of bone loss. Lane et al. has shown that by the eighth decade 50% of the white female population will suffer a vertebral compression fracture, and by age 90, 33% of white females will suffer a hip fracture[11]. Hip fractures in this elderly group carry a mortality rate of 40% to 70% within one to two years following fracture. Also, the acute care costs of treating osteoporotic hip fractures are greater than three billion dollars per year.

There are many noninvasive means of quantitating bone density. Radiographic evaluation is imprecise and requires 30% to 40% of the bone mass to be lost before osteopenia can be detected. Photodensitometry estimates bone mass from a measure of optical density; however, this method does not compensate well for the soft tissue thickness in the region. Single photon absorptometry uses a nuclear counter to measure the attenuation by the bone of a monoenergenic beam. Shortcomings of this technique include inability to control for soft tissue attenuation and position variability. Additionally, it can not be used on the axial skeleton. Double photon absorptometry is a newer technique with the advantage of controlling for soft tissue attenuation and can be applied both to the axial and appendicular skeleton[15]. However, it is expensive and not currently widespread. Quantitative computerized tomography allows the same precision and accuracy as dual photon absorptometry. Likewise, expense and availability are shortcomings of this technique.

Osteoporosis treatment goals are fracture management and prophylaxis. Vertebral compression fractures are managed with bedrest and analgesics for the first five to ten days. Then, progressive mobilization begins over two to three months utilizing a lumbosacral corset during fracture healing. Since each osteoporotic fracture contributes to the kyphotic posture of the spine, walking in an upright position will require a compensatory increase in lumbar lordosis. This can result in postural low back pain. The treatment of Colles' fractures and hip fractures in the osteoporotic patient is the same as for a nonosteoporotic patient. Both have similar healing potentials but osteoporotics have a higher rate of complications such as failure of fixation and malunion.

Preventative treatment obviously requires the identification of susceptible patients. Those patients that have suffered their first osteoporotic fracture require preventative treatment to help prevent future fractures. The question then becomes does the patient with osteoporosis without a history of fracture need prophylaxis? Is it possible to identify a subset of osteoporotic patients more prone to fracture? Recent studies have suggested that single photon absorptometry, dual photon absorptometry and quantitated computerized tomography can determine a critical bone density or fracture threshold below which

the risk of fracture significantly increases. It is unknown which method is most appropriate. Still, other problems exist. The cost of massive screening may not be justified. Also, precise bone density information represents only one variable in a multifactorial fracture problem.

Prevention of osteoporosis includes avoidance of certain substances and exercise. Excessive use of alcohol and tobacco should be avoided due to their deleterious effect on osteoblastic function[3]. In addition, cortical steroid usage inhibits bone formation.

Recent studies have confirmed clinical impressions that mechanical stimuli provide osteoregulatory signals to the skeletal system. The magnitude of the strain induced by a mechanical stimulus helps to regulate the regional amount of bone formation. To have a beneficial effect on bone formation, these stimuli should occur briefly but at recurrent intervals. The optimum exercise program for osteoporotic patients is yet to be determined[1,2,13]. Current pharmacologic intervention for osteoporotic patients include the use of oral calcium, vitamin D, estrogen and sodium fluoride[19,20]. The recommended daily dose of elemental calcium for the postmenopausal patient is 1.5 grams. Side effects of calcium are constipation and renal calculi. Estrogen .625 mg daily should be used for the first 5—10 years after menopause. The current recommendation is 3 weeks of estrogen therapy followed by 1 week of Provera 10 mg daily. This decreased the possible side effects of estrogen which include an increased risk of uterine cancer, stroke and thrombosis. Vitamin D has been recommended in the dose of 400—800 i.u. per day to help treat any unrecognized, associated osteomalacia which coexists in a significant number of postmenopausal osteoporotic patients. These three drugs, estrogen, vitamin D. and calcium, have in many studies been shown to decrease the rate of bone loss. However, sodium fluoride is the only drug shown to increase bone accretion[11]. Sodium fluoride should be used only after a significant period of calcium and vitamin D administration. If this is not done, the unmineralized portion of osteoid will be worsened by the sodium fluoride treatment. A suggested dose of sodium fluoride is 1 mg per kg. per day in divided doses. Known self-limited side effects are nausea, vomiting and arthralgias. The suggested duration of sodium fluoride therapy is 2 years after the last vertebral compression fracture, allowing maximum bone accretion. Physiologic doses of calcium and vitamin D should be continued indefinitely. Calcitonin and anabolic steroids might hold promise for future treatment programs.

Idiopathic, postmenopausal osteoporosis is a multifactorial disease of unclear etiology. The diagnosis is aided by histomorphology and bone density measurements but the efficacy of screening programs is unclear. Prevention includes avoidance of certain substances and exercise. Calcium, vitamin D, estrogen and sodium fluoride are useful therapeutically.

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BENCHTOP MECHANICAL PERFORMANCE OF PROPHYLACTIC KNEE BRACES UNDER DYNAMIC VALGUS LOADING: A CADAVER STUDY

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INTRODUCTION

The large number of serious knee ligament injuries occurring in competitive and recreational athletics remains a foremost concern of the sports medicine community. Despite aggressive pursuit of improved treatment methods, disproportionately little attention has been focused on the area of injury prevention. The introduction, *circa* 1980, of lightweight prophylactic knee braces was therefore enthusiastically welcomed in many quarters. Several major collegiate football programs quickly moved to institute on-field trials, and preliminary experience suggested that these devices were indeed useful in reducing the rates of serious ligament injury[7,13].

However, as interest in prophylactic braces widened and data began to accumulate from larger-scale studies, the favorable experience reported in the initial trials was unfortunately not supported. Recently, Tietz et al. have called into question the entire concept of prophylactic knee bracing, a position supported by the work of Hewson et al. and of Rovere et al.[8,14,16]. But, as pointed out by Albright[1], there are still a number of difficulties in drawing unambiguous conclusions from even the best designed on-field brace studies, owing to variations of injury grading, usage patterns and historical controls.

Given the relative novelty of these brace designs, it may be premature to extrapolate directly from current on-field results to an across-the-board condemnation of the entire concept of prophylactic knee bracing. Because of the enormity of the knee injury problem (upwards of 100,000 serious ligament injuries and 30,000 surgeries per year in the U.S. alone) and the intuitive appeal of protective bracing, we believe that there is a potential for some brace design improvements that will help avoid an appreciable number of these injuries[10]. As noted by the AAOS, the currently-produced braces were not subjected to rigorous performance testing prior to production[2]. Hence, despite aggressive marketing efforts mounted by the brace manufacturers, there is no substantiated reason to think that any of the current designs offer optimal protection.

If knee brace design is to be approached from a sound biomechanical perspective, it seems reasonable to devise laboratory tests to directly measure the effectiveness of load uptake by braces under realistically simulated injury conditions. When the factors governing load uptake (or lack thereof) in prophylactic knee braces are better understood, it seems reasonable that objectively grounded design innovations will be forthcoming. At the very least, standardized laboratory tests can serve both as a method of evaluating the relative performance of current products, and as one means of assessing future design innovations.

An important first step in this direction was taken by Paulos et al., who performed both quasi-static and moderately dynamic lateral loading experiments in a series of fourteen braced versus unbraced cadaver knees[11]. Using foil strain gages to bone near ligament insertion sites, they inferred subfailure and failure ligament loads when the distal femur and proximal tibia were transfixed by thick rods passing in a sagittal plane about 20 cm. from the joint midline. Their data showed that the two braces tested (the Anderson Knee Stabler and McDavid Knee Guard) were "ineffective and possibly detrimental" in resisting valgus forces. While the valgus load uptake in this initial cadaver model may have been unphysiologic due to the absence of axial joint loading and due to the use of rod restraints against sagittal plane motion, such data raise serious questions about the ability of these two braces to directly protect against MCL failure[3,9].

The objective of this study was to investigate the mechanical performance of representative knee braces under more realistic dynamic valgus loading and support conditions. Although every on-field injury involves a unique and complex combination of multiple loading components, the purely valgus load is of particular interest since it represents the major source of medial collateral ligament injuries against which current braces ostensibly protect. We describe design considerations for a laboratory cadaver

model developed to measure MCL response to dynamic valgus loading, and report mechanical performance data for two representative commercial braces.

METHODS

Prophylactic knee brace performance was studied in-vitro using a series of twenty fresh-frozen cadaver legs (eight male, twelve female, average age = 77.1, s.d. = 9.2) taken from decedents with no evidence of knee injury or knee surgery and having no history of musculoskeletal disease relevant to knee joint mechanics. The shank and foot were kept intact, but the femur was transected at the subtrochanteric level. The thigh segment was then denuded of soft tissue over a distance of about 15 cm. below the transection level to facilitate PMMA gripping of the femur within a cylindrical metal sleeve.

A testing fixture (Figure 1) was constructed to provide physiologically realistic specimen support both proximally and distally. Proximally, a two axis bearing positioned at the estimated hip center allowed for free internal/external rotation (axis A-A) and free abduction/adduction (axis B-B). A female cylindrical housing attached to bearing axis B-B accepted the male cylindrical sleeve from the PMMA femoral grip. The hip bearings were in turn coupled to a pneumatic actuator. Upon pressurization, this actuator applied a static longitudinal compressive force (305 N) to the femur, thus inducing axial compression at the knee, simulating weight bearing. Distally, the specimen foot was held within a tightly laced conventional athletic shoe. The sole of the shoe was in turn bolted rigidly to the same massive support fixture as that used for the pneumatic actuator mount. This support fixture, was configured to position the leg specimen horizontally above the ram of an MTS servohydraulic materials testing machine.

To measure MCL protection due to bracing, we monitored ligament distension using liquid metal strain gages (LMSG's)[5]. We exposed the MCL through a minimal longitudinal incision. Owing to marked strain deviation on various sites of the MCL, we affixed two LMSG's at similar sites on each MCL specimen, one on the longitudinal fiber bundle and one on the posterior fiber bundle. To ensure firm attachment of the LMSG to the ligament body at each mounting site, we bonded small rectangular plastic wings to both ends of the LMSG, using cyanoacrylate adhesive. Direct attachment to the ligament body was achieved with 3-0 silk suture passed in an interrupted fashion through tiny holes punched in the gage wings and then firmly into the substance of the ligament. We brought the LMSG lead wires out through the incision, and re-approximated the skin edges with running 4-0 suture. We pre-calibrated each LMSG prior to attachment.

Prior to applying a valgus loading pulse to the horizontally oriented leg specimens, gravity plus the absence of

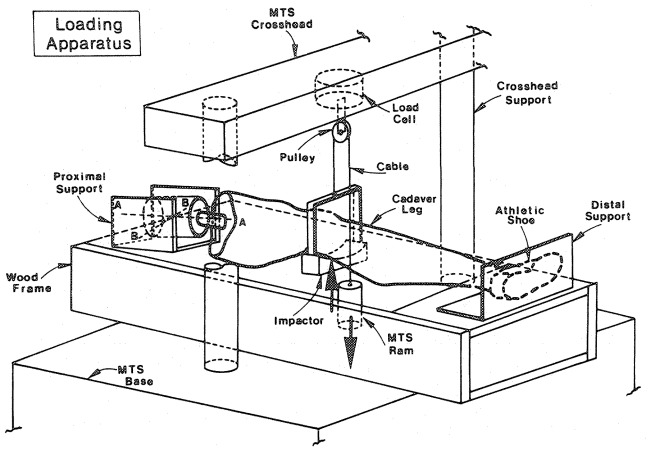

Figure 1
Schematic diagram of the specimen loading and support fixture.

active muscle support resulted in variable degrees of varus angulation. To facilitate series wide comparison of MCL strain changes due to bracing, we subjected each specimen to a static valgus preload, P_o (Figure 2), the force necessary to overcome the initial slack and to pass through the toe region of the MCL's load/distension curve and reach the threshold of quasilinear load-vs-strain behavior. While each individual specimen required a different valgus preload level to reach this threshold, that preload level was kept constant for all trials of a given specimen. In many instances this preload turned out to be fairly substantial, corresponding on an average to 27.3% (s.d. = 8.4%) of the eventual failure load.

Delivery of a dynamic valgus blow was provided by a massive steel impactor, driven by the actuator of the MTS machine. To insure that no undetected force components would be developed in directions other than that of intended loading, a pulley and cable system converted downward

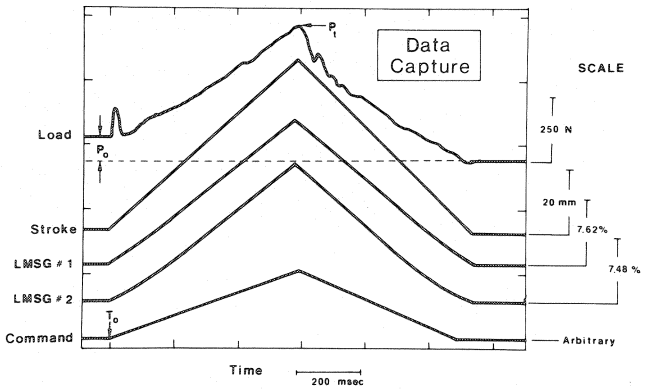

Figure 2
Typical capture of time-variant load uptake data. The small sharp transient in load cell output just after the beginning of dynamic loading and just after target cutoff load attainment corresponds to the force needed to overcome impactor inertia for discontinuous commanded velocities.

actuator displacement into upward impactor displacement. The valgus loadings were imposed at a constant MTS actuator speed (100 mm/sec). The signal from the MTS load cell reversed the actuator motion at the instant when a preselected contact force level was achieved.

The twenty cadaver leg specimens, thus instrumented, mounted and preloaded, were subjected to a series of valgus blows. At each of several target load cutoff levels, we tested the specimen first with the AmPro dual-upright brace (Figure 3a), next with the Anderson Stabler single-upright brace (Figure 3b), and finally in the unbraced state (i.e., three trials at each load). Since the failure load for each individual specimen could not be predicted in advance, our strategy was to test each of the three configurations beginning at a low target load cutoff level, and then to repeat the three trial sequence at incrementally higher target cutoff loads. To assure test repeatability, trials for a given target cutoff load and configuration were repeated frequently at random intervals for each specimen.

Three major parameters were used to compare the two braces' performance to each other and to the unbraced configuration. The first, which we termed relative strain attenuation (RSA), reflects the ability of each brace to reduce the load induced distension of the MCL. Because of large interspecimen variation in absolute failure or in pre-failure maximum strain (e.g., ranging from 0.012 to 0.257 in the case of the unbraced configuration), simply comparing the average absolute strains would not be an appropriate method for assessing brace performance.

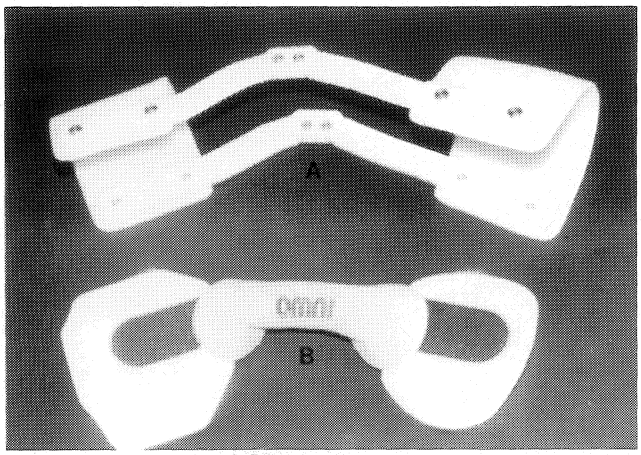

Figure 3A-B
The two representative prophylactic braces tested: (a) the AmPro, which has dual plastic uprights, and (b) the Anderson Knee Stabler, which has a single steel upright.

Instead, the ability of bracing to lower the strain experienced by the MCL was expressed in terms of the series-averaged relative strain attenuation, RSA. The RSA was computed for individual specimens by subtracting from unity the ratio of peak pre-failure strain occurring for the braced versus unbraced configurations.

The second performance parameter was the apparent gross stiffness exhibited by specimens in the braced versus unbraced configuration. This was again tabulated for the highest sub-failure load trial, and included the entire dynamic valgus load uptake event (i.e., spanning the interval from dynamic load initiation until target cutoff load attainment). The apparent gross stiffness was defined simply as the ratio of valgus contact force change to valgus knee displacement occurring over that interval. The increase in apparent gross stiffness after application of a particular brace reflects the overall degree of structural buttressing afforded to the knee by that brace, but does not address the question of MCL protection.

The third performance parameter was the total failure load. We defined this as the sum of the static preload plus the applied dynamic valgus load present at the instant of first detectable specimen failure. Obviously, failure could occur for each specimen in only one of the three testing configurations. While intraspecimen (i.e., in each of the three configurations) analysis of this parameter is therefore not possible, the series averaged total failure load levels do provide rough estimates of absolute MCL tolerance to valgus blows under the three respective test configurations. It is of interest that several modes of MCL disruption occurred in our series, including partial tears, complete rupture ("mop-end" tears), and avulsion of the ligamentous attachment to bone. Also, there were a few instances where some ACL damage was apparent. To provide a consistent criterion for failure that would be applicable under these varied circumstances, failure was defined to have occurred at the instant of first decrease of valgus load carrying capacity of the knee or knee/brace construct. Each specimen was tested at each given load first with the dual upright brace, then with the single upright brace and finally unbraced.

The statistical significance of parametric differences was determined using paired Student's T-testing of unbraced versus dual-upright, unbraced versus single-upright, and dual-upright versus single-upright configurations.

RESULTS

Seven of the specimens exhibited behavior which necessitated their exclusion from final data tabulation. Two of these were specimens which failed to exhibit reproducible load/deformation behavior upon retesting at sub-failure load levels. In two other specimens ligament failure occurred at excessively low load levels, prior to completion of even the first round of testing. The remaining three excluded specimens did not successfully uptake the imposed valgus loads, due to unstable rotation or flexion during the dynamic loading event.

Of the remaining thirteen specimens, four ultimately failed for some reason other than MCL rupture. In one of

these specimens, there was frank rupture of the midsubstance of the anterior cruciate ligament, without any grossly visible evidence of compromise of the MCL. In the other three specimens, failure occurred due to a fracture of the femur occurring at or near the site of proximal fixation. Although it was inappropriate to include these four specimens in the MCL failure load tabulations, their pre-failure behavior met our criteria for inclusion in the relative strain relief and apparent gross stiffness tabulations.

The peak sub-failure MCL strain averaged 0.126 (s.d. = 0.092) for the unbraced knee, versus 0.109 (s.d. = 0.087) and 0.099 (s.d. = 0.077) for the single-upright and dual-upright braces, respectively (Table 1). Taken as a whole, these absolute peak sub-failure strains are consistent with strain-to-failure levels reported by other investigators for excised ligaments subjected to simple axial loading[6]. The relative strain attenuation averaged 18.3% when the specimens were braced with the single-upright device and 25.1% when they were braced with the dual-upright device (Table 2). Both braces afforded statistically significant ($p < 0.005$) reductions in peak sub-failure strains when compared to the unbraced configuration. While there was a distinct trend toward preferentially better strain reduction (i.e., a 7.4% greater RSA) for the dual-upright brace, the data was not statistically significant.

The unbraced specimens exhibited an average gross stiffness of 4.26 N/mm, whereas the corresponding values for the dual-upright and single-upright braces were 4.20 N/mm and 3.79 N/mm, respectively (Table 3). These differences were not statistically significant.

Despite care in implementing relatively modest load increments, the new higher loads seen by the dual-upright configuration caused failure in four of the specimens. Two of the specimens failed when braced with the dual-upright brace. Failure of an unbraced specimen occurred in three instances under a load for which sufficient protection had been afforded by both braces.

Table 1
Peak (Absolute) Medial Collateral Ligament Strain

Specimen #	Unbraced	Dual-Upright	Single-Upright
3	0.257	0.214	N/A
6	0.089	0.047	0.047
7	0.200	0.107	0.214
8	0.207	0.164	0.200
10	N/A	N/A	N/A
12	0.200	0.194	0.176
14	0.086	0.064	0.064
15	0.050	0.057	0.043
16	0.157	0.118	0.118
17	0.225	0.195	0.208
18	0.014	0.011	0.013
19	0.013	0.012	0.011
20	0.012	0.005	0.006
Mean:	0.126	0.099	0.109
S.D.:	0.092	0.077	0.087

Table 2
Relative Peak Sub-Failure Strains
(= Braced/Unbraced Strain at Highest Sub-Failure Target Cutoff Load)

Specimen #	Unbraced	Dual-Upright	Single-Upright
3	100%	83.3%	N/A
6	100%	53.1%	53.1%
7	100%	53.5%	107.0%
8	100%	79.3%	96.8%
10	100%	42.2%	79.9%
12	100%	97.2%	90.0%
14	100%	74.4%	74.4%
15	100%	124.2%	90.2%
16	100%	75.2%	75.2%
17	100%	85.0%	93.3%
18	100%	76.9%	89.5%
19	100%	91.7%	80.4%
20	100%	37.5%	50.0%
	Mean = 100%	Mean = 74.9%	Mean = 81.7%
	S.D. = 0	S.D. = 23.8	S.D. = 16.9
	N = 13	N = 13	N = 12

Relative Strain Attenuation (RSA) = 100% — Relative Strain
For dual-upright: RSA = 25.1%
For single-upright: RSA = 18.3%

DISCUSSION

The primary intent of our investigation was to ascertain if either of the two brace designs could afford substantial mechanical protection to the knee. Under purely valgus loading in full knee extension, it seemed intuitive that any such protection would tend to be maximal. Our data showed that both braces offered modest but statistically significant degrees of MCL protection under these test conditions. Of the two specific designs tested, the plastic dual-upright brace appeared to be more protective than the metal single-upright brace, although this difference was much less than the difference between either brace and the unbraced condition. For the three knees which failed in the unbraced configuration, the average valgus failure load of 471 N

Table 3
Apparent Gross Stiffness
(Stiffness = Valgus Load/Valgus Displacement (N/mm))

Specimen #	Unbraced	Dual-Upright	Single-Upright
3	7.8	6.3	N/A
6	4.8	4.8	4.8
7	5.5	6.3	4.2
8	5.0	4.5	4.2
10	6.3	4.7	4.7
12	3.0	3.5	3.5
14	3.0	3.0	2.9
15	3.7	3.7	3.3
16	4.5	4.8	4.8
17	2.6	3.0	2.6
18	2.4	2.6	3.1
19	3.3	3.9	3.5
20	3.5	3.5	3.9
Mean:	4.26	4.20	3.79
S.D.:	1.59	1.18	0.76

corresponds to a valgus bending moment of approximately 95 N-m. This value is substantially higher than the 48 N-m "near-failure" valgus moment reported by Seering et al.[15]. One possible explanation for this difference is that because our specimens were axially loaded so as to approximate playing field conditions, a portion of our applied valgus moment was required simply to overcome the varus moment (about the lateral compartment center of rotation (associated with the medial-compartment contact force. Furthermore, minimal dissection of secondary valgus support structures was undertaken in our study. Also, our definition of failure load included the static preload necessary to overcome the gravitational varus moment. Finally, other things being equal, the distinction between frank failure and "near-failure" imparts a different definition to the terminal load.

The laboratory test conditions in this investigation vary considerably from those conditions occurring during athletic activity. Our specimens were obtained from elderly decedents who proportion of fat to muscle was much greater than that which would be encountered in a young, well-conditioned athlete. Further, to facilitate proximal fixation in the loading apparatus, it was necessary to incise the origins of the proximal femoral musculature. These factors dictated that the braces be affixed to a highly compliant and deformable substrate. Consequently, the coupling between brace and knee in our study was probably inferior to that which would occur in a living subject, a factor which would tend to lessen the apparent degree of protection afforded by the brace.

However, cadaver knees are inherently less able to resist valgus loading than those of live athletes, owing to the absence of muscle contraction in the cadavers. Pope et al.[12] have shown that for quasistatic valgus loadings below the pain threshold in normal volunteers, active sartorius contraction causes a 282% increase in apparent valgus stiffness of the knee, and active quadriceps contraction causes a 247% increase in valgus stiffness of the knee[12]. Also, it would be expected that the ligament stiffnesses and strengths in our elderly specimen population would be greatly inferior to those of young athletes. Therefore, one would expect the fraction of the overall valgus load taken by the brace to increase as the passive stiffness of the anatomic knee decreases. This factor would act to exaggerate the apparent degree of protection afforded by the brace, and would tend to counteract the protection-decreasing influence of overly compliant brace/knee coupling noted above.

Another factor which warrants consideration is the occurrence of knee rotation and flexion sometimes observed during load delivery. Our specimen support fixture, offered no constraint against internal/external rotation, and no control against flexion/extension once rotation began. The freedom of rotational movement designed into our test apparatus necessitated properly balanced alignment of the impactor onto the specimen in order to avoid rotation, thereby delivering a purely valgus blow. Nevertheless, there were instances when so much rotation occurred that the specimen was unable to take enough load to reach the target cutoff force level. In these instances, the data were discarded and the specimen was repositioned for another trial. It was evident that the substantial lateral offset (approximately 2.5 cm. beyond the skin surface) of the single-upright brace contributed markedly to this rotational tendency. If the line of action of the impact force did not pass precisely through the center of the specimen as well as through the lateral stabilizing bar, the brace/knee unit began to "spin off" from the bar/impactor contact point. Such rotation, once underway, rapidly cascaded due to the substantially increasing moment (level arm effect) of the contact force about the specimen axis. In our cadaver test, these loading sequences were always terminated automatically well below the damage threshold, and the data were simply discarded. On the playing field, however, it is not difficult to imagine that a similar but unchecked "spin off" mechanism could lead to serious rotational injury.

Owing to the limited number of specimens in our study, to the substantial degree of data dispersion and to the one failure per specimen nature of the testing, we do not believe that the average failure loads documented in this series (Table 4) necessarily represent the full valgus load uptake capacity of the respective bracing configurations. However, we do believe that these data suggest that there are circumstances under which one or both of these braces could avert a ligament failure. These failure results, as well as the statistically significant differences in pre-failure

Table 4
Failure Measurements
(Total Failure Load = Dynamic Failure Load + Static Preload)

Specimen #	Dynamic Failure Load	Static Preload	Total Failure Load	Configuration at Failure
3	475	125	600	Unbraced
6	875	250	1125	Dual-Upright
7	250	75	325	Dual-Upright
8	200	75	275	Dual-Upright
10	N/A	N/A	N/A	Femur Ex.
12	250	238	488	Unbraced
14	250	113	363	Single-Upright
15	250	75	325	Dual-Upright
16	287	100	387	Single-Upright
17	225	100	325	Unbraced
18	N/A	N/A	N/A	ACL Rupture
19	N/A	N/A	N/A	Femur Fx.
20	N/A	N/A	N/A	Femur Fx.
Average Failure Loads:				
Unbraced = 471, Single-Upright = 375, Dual-Upright = 512				
S.D. = 138				

Note: Test sequence at each target cutoff load cutoff was: Dual-Upright, then Single-Upright, then unbraced. Thus, if knee failed prior to attaining the target cutoff load during a single-upright, it had already achieved that target load in the dual-upright test, etc.

MCL strain, suggest that both braces do offer at least a moderate degree of protection against purely valgus blows sustained at full knee extension. However, such idealized loading conditions obviously represent only a small subset of the potential injury situations arising on the playing field, and it is our impression that the degree of protection afforded by these two devices diminishes markedly if the knee rotates or flexes. While it remains to document this impression with objective data, it may be part of the explanation for some epidemiologic reports concluding that current braces are ineffective in reducing the incidence of serious knee injuries.

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KNEE LIGAMENT INJURIES IPSILATERAL TO FEMORAL SHAFT FRACTURES

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INTRODUCTION

The combination of femoral shaft fracture and ipsilateral knee ligament injury has received attention in the orthopaedic literature. Ritchey et al. in 1958 reported a 17 percent incidence of "unstable knee injuries" ipsilateral to femoral fractures in a series of thirty automobile dashboard impact injuries. In 1968, Omer et al. reported one knee ligament injury in a series of 25 patients with either tibial or femoral fractures[5]. In 1968, Pederson and Serra reported 10 patients with the combination of knee ligament injury and femoral fracture[6]. Of the 6 cases that gave accident details, 5 were pedestrian accidents. The total number of femoral fractures was not reported in this series so an incidence is unavailable. Fraser et al., in 1978, reported retrospectively a 5 percent incidence of ipsilateral knee ligament laxity in 222 patients seen acutely and a 39 percent incidence of ligamentous "laxity" among 69 patients seen in follow-up[3]. In 1978, Dunbar and Coleman reported that 14 of 20 patients studied retrospectively showed at least "minimal" knee laxity ipsilateral to femoral fracture and 5 of 20 showed "significant" laxity[2]. Walker and Kennedy in 1980 reported retrospectively a 49 percent incidence of ligamentous injury in 54 fractures[10]. They reported that femur fractures occurred in motor vehicle accidents (48), athletic injuries (4), or falls (2). They, however, did not give the incidence of ligamentous damage with each fracture etiology. Additionally, they imply that dashboard type accidents cause most of the knee ligament injuries. With a mean follow-up of 5 years, Rowntree and Getty reported in 85 patients a 45 percent incidence of ligamentous laxity ipsilateral to femoral shaft fractures[8]. Walling et al. prospectively reported a 33 percent incidence of this injury combination, and they reported the injury occurrence in 6 of 10 motorcycle accidents and 2 of 7 pedestrian accidents. More recently in 1988, Moore et al. reported a 5 percent incidence of this injury combination in a retrospective study of 320 femoral shaft fractures[4].

There is, therefore, a large difference in the reported incidences (5 to 70 percent). Moore et al. called for further studies to consider the nature of the trauma in order to "have a better understanding of the frequency of the injury and whether patterns exist"[4]. The purpose of this study is to identify accident patterns that more likely result in the combined femur fracture and knee ligament injury. Also, we will discuss possible reasons for this disparity in incidence reports.

METHODS AND MATERIALS

The medical records of patients with femoral shaft fractures treated at the University of New Mexico Medical Center from the years 1981-1987 were reviewed. Patients with pathologic fractures or fractures secondary to missile injuries were excluded. Pediatric patients, patients with a history of previous femur fractures and patients with previous knee injuries were also excluded. A total of 157 patients with 163 femoral shaft fractures composed the study group. The accident type as well as the grade of ipsilateral knee ligament instability were recorded.

Clinical examination under general anesthesia after femoral stabilization and observation at arthrotomy were used to determine ipsilateral knee ligament injury. Knee ligament instabilities were graded using the *Standard Nomenclature of Athletic Injuries* formulated by the Committee on the Medical Aspects of Sports within the American Medical Association[1].

RESULTS

Of the 163 femoral shaft fractures, 19 had one or more ipsilateral knee ligament instability patterns for an overall incidence of 11.6 percent. Two of the 83 fractures sustained in automobile accidents showed this injury combination, an incidence of 2 percent. Six of the 26 patients sustaining femoral shaft fractures sustained in pedestrian versus motor vehicle accidents displayed some form of knee ligament instability. This produced an incidence of 23 percent. Twelve of the 25 patients sustaining femoral shaft fractures from motorcycle accidents had ipsilateral knee ligament instability, an incidence of 34 percent. None of the 18 femoral shaft fractures sustained in lower energy sporting events had ipsilateral knee ligament instability. The average patient age was 27 with a range from 18-42 years.

The medial collateral ligament (MCL) was the most commonly injured (13). The anterior cruciate ligament

(ACL), the posterior cruciate ligament (PCL), and the lateral-collateral ligament (LCL) were injured in 6, 5 and 3 patients respectively. Six patients sustained injuries to 2 knee ligaments. Three of these patients had combined MCL and ACL instability. Fifteen of the ligament injuries were Grade II, 10 were Grade III and no Grade I injuries were identified. Eleven of the 19 patients had 2 or more major additional fractures (tibia, pelvis, ankle or humerus).

DISCUSSION

The data collected in this study suggest that motorcycle accidents or motor vehicle versus pedestrian accidents causing femoral shaft fractures have a relatively high incidence of ipsilateral knee ligament instability. In this series, motor vehicle passengers sustaining femoral shaft fractures had a low rate of ipsilateral knee ligament injury. Although a common cause in other series, the motor vehicle dashboard injury was an uncommon cause of ipsilateral knee ligament injuries in our follow-up[10].

Discussions of the trauma types causing this combination injury have been published but no large series describes the incidence of the various trauma types[6,11]. Multiple trauma has been implicated in patients sustaining this injury combination[4]. Our results showed multiple trauma to be very common in both femoral fractures without ipsilateral knee ligament injury (64 percent) and with ipsilateral knee ligament injury (68 percent). Low energy trauma, such as sporting injuries, made this injury combination very unlikely (0 percent in our series).

We feel the disparity in previously reported incidences of this injury combination is related to three factors. First, selection of patients is important. If patients with pathologic fractures or missile induced fractures are included then a lower incidence will be reported. Second, prospective studies tend to report higher incidences, especially if the authors report minimal ligamentous injury. Retrospective studies, in contrast, report lower incidences due to inadequate documentation, exclusion of minor ligament injuries and missed ligament injuries. Third, studies basing their reports on the initial evaluation report lower incidences because of inadequate documentation and missed diagnoses later reported in follow-up. Studies which report a longer follow-up have higher incidences. However, these reports may select out problem patients willing to return for long-term follow-up (often a low percentage of the initial number) and artificially raise the incidence.

SUMMARY

Patients incurring femoral shaft fractures in motorcycle or pedestrian versus motor vehicle accidents are at high risk for ipsilateral knee ligament injuries. The most common ligament lesion in our series was either Grade II or Grade III MCL injury. In our follow-up, motor vehicle accidents were an uncommon cause of this injury combination, and it did not occur as a result of sporting activity injuries. We found an 11.6 percent incidence of ipsilateral knee ligament injuries in patients with femoral shaft fractures. We recommend future studies to better describe the mechanism causing this injury combination.

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